fortune Journals

Original Article

FORTUNE JOURNAL OF HEALTH SCIENCES

ISSN: 2644-2906

Long-Chain Per- and Polyfluoroalkyl Substance Exposure Through Breast feeding and Its Association with Infant Growth: The Hidden Influence of Maternal Age and Obesity

Nalae Moon[1], SungChul Seo[2], Ju Hee Kim*,[1]

Abstract

This study investigated the concentrations of per- and polyfluoroalkyl substances (PFASs) in the breast milk of 201 Korean mothers and their potential impact on infant growth. Despite regulatory efforts, certain PFAS have shown increasing trends with levels higher than those reported in many other countries. The geometric mean concentrations of perfluorooctanoic acid (PFOA) and perfluorooctane sulfonate (PFOS) in breast milk were 0.098 and 0.085 ng/mL, respectively. Notably, PFOS (0.085 ng/mL) and perfluorononanoic acid (PFNA) (0.023 ng/mL) concentrations were significantly higher in obese mothers (BMI ≥ 25 kg/m²), correlating with reduced weight-for-length values in male infants (β = –0.53, p = 0.024 for PFOS; β = –0.60, p = 0.006 for PFNA). The estimated daily intake of PFASs in breastfed infants was 17.53 ng/kg bw/day for PFOA, 13.37 ng/kg bw/day for PFOS, and 4.36 ng/kg bw/day for PFNA, exceeding previous domestic reports. Sensitivity analysis showed that the PFAS concentration in breast milk was the most influential factor affecting oral exposure. Considering the endocrine-disrupting properties and sex-specific effects of PFASs, targeted regulatory measures are crucial. Future research should employ longitudinal designs with larger cohorts to better assess the long-term health risks and develop effective mitigation strategies for high-risk populations.

Keywords: perfluorooctanoic acid (PFOA, perfluorooctane sulfonate (PFOS), perfluorononanoic acid (PFNA), polyfluoroalkyl substances (PFASs)

Introduction

Per- and polyfluoroalkyl substances (PFASs) are synthetic chemicals that are widely used in industrial and consumer products, such as non-stick cookware, waterproof textiles, food packaging, and firefighting foams (Fenton et al., 2021; van Beijsterveldt et al., 2022). Owing to their strong carbon-fluorine bonds, PFASs are highly resistant to degradation, leading to widespread environmental contamination and bioaccumulation in humans (Fenton et al., 2021; Bernasconi et al., 2022; Panieri et al., 2022). Among them, long-chain PFASs, including perfluorooctanoic acid (PFOA) and perfluorooctane sulfonate (PFOS), exhibit a greater persistence and bioaccumulation potential than short-chain PFASs. Long-chain PFASs accumulate in the human blood, liver, and breast milk, leading to prolonged exposure, particularly in vulnerable populations such as pregnant women and infants (Ng and Hungerbühler, 2013; Gomis et al., 2018).

Breast milk serves as a critical biological matrix for mothers and newborns, providing essential nutrients, immune factors, and bioactive compounds that promote infant development (Victora et al., 2016). However, they also act as a medium for the transfer of PFASs from maternal excretion to infant intake (Mondal

Affiliation:

[1]Department of Nursing, College of Nursing Science, Kyung Hee University, Seoul 02447, Korea

[2]Department of Nano, Chemical and Biological Engineering, College of Natural Science and Engineering, Seokyeong University, Seoul, 02713, Republic of Korea

***Corresponding author:**
Ju Hee Kim, Department of Nursing, College of Nursing Science, Kyung Hee University, Seoul 02447, Korea.

Citation: Nalae Moon, SungChul Seo, Ju Hee Kim. Long-Chain Per- and Polyfluoroalkyl Substance Exposure Through Breastfeeding and Its Association with Infant Growth: The Hidden Influence of Maternal Age and Obesity. Fortune Journal of Health Sciences. 9 (2026): 132-148.

Received: February 20, 2026
Accepted: February 26, 2026
Published: March 18, 2026

et al., 2014; Blomberg et al., 2023). PFAS enter breast milk primarily through maternal blood circulation, facilitated by their amphiphilic properties and affinity for proteins (LaKind et al., 2022). Previous epidemiological studies in humans have reported that PFOS, the most frequently detected PFAS in breast milk, was found in 31–100% of samples, with mean concentrations ranging from 0.015 to 79.3 ng/L (Cariou et al., 2015; Rawn et al., 2022; Fiedler et al., 2022; Rovira et al., 2022; Mahfouz et al., 2023; Hartmann et al., 2024; Li et al., 2024). These PFAS compounds in breast milk are transmitted to newborns through breastfeeding by binding to lipids and proteins (Pham et al., 2024). Due to their incomplete metabolism, newborns cannot completely excrete PFASs, leading to bioaccumulation and potential adverse health effects (Yao et al., 2023). Previous studies have reported that exposure to PFASs in breast milk negatively affects infant growth and neurological development and causes liver toxicity, endocrine dysfunction, and inflammatory diseases (Mogensen et al., 2015; Jin et al., 2020; Costello et al., 2022; Zhou et al., 2023; Hoadley et al., 2023; Hyötyläinen et al., 2024; Tao et al., 2025). Several epidemiological studies have examined the effect of PFAS exposure through breastfeeding on fetal and infant growth (Gao et al., 2022; Zhang et al., 2022; Mahfouz et al., 2023; Li et al., 2024; Tao et al., 2025). For example, Verner et al. (2015) conducted a cohort study and found that higher PFAS exposure in breastfed infants was associated with reduced weight gain during early infancy. Høyer et al. (2018) reported that increased prenatal and postnatal exposure to PFOA and PFOS was linked to lower birth weight and decreased infant length during the first year of life. Additionally, Papadopoulou et al. (2022) found that PFAS exposure through breastfeeding is associated with alterations in infant body mass index (BMI) trajectories, with some infants showing slower weight gain over time. Mahfouz et al. (2023) reported that higher long-chain PFAS concentrations in breast milk were associated with lower weight-for-length (WFL) z-scores in newborns. In a recent study, Tao et al. (2025) found that exposure to PFAS mixtures through breastfeeding was negatively associated with z-scores for height-for-age and head circumference-for-age, whereas a positive association was found between BMI-for-age z-scores, but only in girls. Despite these findings, research on the health effects of PFAS exposure has yielded mixed results, particularly in studies examining their impact on infant growth. Findings in this area have been inconsistent, particularly regarding sex-specific differences. While some studies have suggested that male infants may be more sensitive to prenatal PFAS exposure (Gao et al., 2022; Li et al., 2024), others have reported reduced growth in female infants or no significant associations (Starling et al., 2019; Cai et al., 2023).

Emerging evidence suggests that PFAS concentrations in breast milk vary according to maternal factors such as age, BMI, and parity (Lee et al., 2018; Luzardo et al., 2019; Serrano et al., 2021; Rovira et al., 2022; Rawn et al., 2022). Older mothers tend to have higher PFAS body burdens owing to cumulative lifetime exposure and slower excretion rates (Luzardo et al., 2019; LaKind et al., 2022). Likewise, maternal obesity may influence PFAS distribution and metabolism, as PFAS have an affinity for adipose tissue, and BMI-related metabolic changes could alter their bioavailability and transfer to the infant (Starling et al., 2014). Similarly, a previous study found that mothers who experienced weight gain since preconception had a 2.2-fold higher risk of increased PFAS concentrations in breast milk (Serrano et al., 2021). Jensen et al. (2020) highlighted that maternal BMI was associated with differences in PFAS partitioning between plasma and breast milk, potentially affecting infant exposure levels. Furthermore, primiparous mothers (first-time mothers) have been found to exhibit higher PFAS levels in breast milk compared to multiparous mothers, as repeated pregnancies and lactation may contribute to PFAS excretion (Brantsæter et al., 2013; Rawn et al., 2022). Conversely, another study found that mothers who breastfed for more than one month but less than ten months had a 1.8-fold higher risk of increased long-chain PFAS concentrations in breast milk (Serrano et al., 2021). In a previous Korean study (Lee et al., 2018), the concentrations of the sum of four PFASs in breast milk varied according to maternal demographics, increasing from 130 ng/L to 232 ng/L as maternal age increased from the twenties to the forties. Similarly, as the BMI increased from 18.5 kg/m² to 25 kg/m², PFAS concentrations increased from 131 ng/L to 151 ng/L. Additionally, primiparous mothers exhibited a concentration of 156 ng/L, whereas multiparous mothers exhibited a slightly lower concentration of 148 ng/L (Lee et al., 2018). These findings highlight the importance of considering maternal age, BMI, and parity when assessing PFAS exposure in breast milk. Thus, this study aimed to analyze the PFAS concentrations in breast milk from a cohort of healthy mother-infant pairs in South Korea, evaluate their estimated daily intake (EDI), and compare the findings with international data. We also investigated the association between PFAS exposure in breast milk and infant growth. Furthermore, we sought to uncover the potential hidden roles of maternal factors, such as age, BMI, and parity, which may influence PFAS concentrations in breast milk.

Materials and Methods

Study design and population

The study population consisted of mother-infant pairs recruited from the LIFE-POPs Cohort (Longitudinal Impact of Fetal and Early-Life Persistent Organic Pollutants), which includes healthy Korean mother-infant dyads in South Korea

Moon N, et al., Fortune J Health Sci 2026
DOI:10.26502/fjhs.403

between 2023 and 2025. The eligible participants were mothers who had initiated breastfeeding and their singleton infants. The inclusion criteria for this study were as follows: (1) Healthy mothers currently breastfeeding infants younger than six months. (2) Mothers without congenital defects. (3) Mothers without breast-related medical conditions (e.g., mastitis, inflammation, or immune diseases). Mothers with chronic illnesses or pregnancy complications that could affect lactation or infant growth were excluded. Of the 204 participants, three were excluded because of an insufficient volume of breast milk or missing information on the mother or child. A total of 201 mother-infant pairs were included in the final analysis. The study protocol was approved by the Institutional Review Board of Kyung Hee University [KHSIRB-24-218(NA) and KHSIRB-21-598(NA)]. Written informed consent was obtained from all participating mothers before enrollment.

Study variables

The questionnaire employed in this study included sociodemographic information on mothers (maternal age, height, weight, education level, monthly household income, employment status, residence area, parity, and underlying diseases) and infants (birth age, sex, birth weight and height, current weight and height, and congenital diseases). Breast milk intake (feeding frequency, amount, and daily feeding patterns) was also assessed. Infant growth was assessed using regular anthropometric measurements. Infant weight and length were measured at birth and during follow-up visits at 6 and 12 months by trained registered nurses using standardized techniques. For example, the nurses ensured that the infants were laid straight on a recumbent length board, with the measurement line parallel to their bodies. The infants' heads and feet were kept immobile, and their knees were straightened while the measuring board was firmly positioned against both heels. Measurements were recorded to one decimal place in centimeters. Infant weight was measured using an infant scale, with infants wearing only a new diaper and light undergarments, and was recorded to one decimal place in kilograms. In this study, we used WFL to assess infant growth more accurately. WFL is an index that evaluates an infant's weight relative to its length, as recommended by the American Academy of Pediatrics and is known to better reflect early growth patterns than the BMI (Daniels et al., 2015). To calculate the z-scores, we utilized the World Health Organization (WHO) standard data for children under two years of age to generate WFL measurements (WHO Multicenter Growth Reference Study Group, 2006). In this study, we categorized mothers based on maternal age, parity, and BMI, as previous studies have reported these factors to significantly influence differences in PFAS concentrations (Lee et al., 2018; Luzardo et al., 2019; Serrano et al., 2021; Rovira et al., 2022; LaKind et al., 2022; Rawn et al., 2022). These factors were considered as potential confounding variables in the analysis because of their known associations with infant growth and/or PFAS metabolism.

PFAS analysis in breast milk

To ensure the safe collection of breast milk samples with minimal contamination, registered nurses collected 20 mL of breast milk into pre-cleaned polypropylene tubes using the hand-expression method. Breast milk was collected once from each mother under conditions in which their usual hydration, diet, and lifestyle were maintained. All breast milk samples were immediately stored at –70°C until laboratory analysis.

The breast milk samples were analyzed for five PFASs using liquid chromatography-tandem mass spectrometry (LC-MS/MS). The analytical methods followed those described by Lee et al. (2018) and Kim et al. (2023). PFAS standards were obtained from Wellington Laboratories (Guelph, ON, Canada), and reagents (ammonium acetate, acetonitrile, and sodium carbonate) were sourced from Sigma-Aldrich. Following the addition of internal standards at 5 ng, an ion-pairing buffer comprising 0.25 M sodium carbonate and 0.5 M tetrabutylammonium solutions was introduced into approximately 3 mL of breast milk samples. The samples were extracted with methyl tertiary butyl ether by mechanical shaking for 30 min. Subsequently, the organic layer was separated by centrifugation; this extraction step was repeated twice. The collected extract (15 mL) was evaporated under a nitrogen atmosphere. The residues were reconstituted in 5 mL methanol and further diluted with 45 mL Milli-Q water. An Oasis WAX cartridge (cartridge No. 186002493) was used for purification and washed with 0.1% ammonium hydroxide in methanol, followed by washing with methanol and Milli-Q water. The five PFASs were quantified using a Vanquish UPLC system connected to a TSQ Quantis mass spectrometer employing a Thermo Scientific Accucore RP-MS column.

Blank samples were consistently used to monitor background contamination throughout all the experimental stages, and trace PFAS levels in the blanks were deduced from the concentrations measured in the test samples. Calibration standards were routinely analyzed to ensure instrumentation stability. The relative standard deviations (RSD) of the calibration standards were maintained within a 30% threshold. Native standards were spiked into ten selected samples to assess the analytical efficiency, with recoveries of these native standards ranging between 70–130%. The recoveries of the mass-labelled PFAS standards ranged from 85–117%. The limits of detection (LOD) for the PFAS were 0.069 ng/mL for perfluorohexane sulfonate (PFHxS), 0.070 ng/mL for PFOS, 0.047 ng/mL for PFOA, 0.013 ng/mL for perfluorononanoic acid (PFNA), and 0.017 ng/mL for perfluorodecanoic acid (PFDeA).

Moon N, et al., Fortune J Health Sci 2026
DOI:10.26502/fjhs.403

Estimated daily intake (EDI) of PFAS in breastfed infants

The EDI of PFAS for each infant was determined using the following formula:

$$EDI = (C * DI)/BW \text{ (ng/kg bw day)}$$

where C represents the PFAS concentration of the analyte in the breast milk samples (ng/mL), DI is the daily intake volume (calculated as the frequency of breastfeeding multiplied by the volume per feeding, mL/d), and BW is the infant's body weight (kg).

In this study, we calculated the EDI of PFAS by assessing the actual body weight and breast milk intake of 81 exclusively breastfed infants under six months of age. Sensitivity analysis was performed to assess how body weight, breast milk intake, and PFAS concentration influenced the EDI. All variables were standardized to z-scores. A linear regression model was built with the EDI as the dependent variable and the three variables as predictors. Standardized regression coefficients were used to evaluate the relative sensitivity of each variable.

Statistical analyses

Participant characteristics were summarized using descriptive statistics, including frequencies, percentages, means, and standard deviations (SD). The concentrations of the analyzed chemicals were reported as geometric means (GM) and percentile values. For chemicals with 70% or more of the population, values below the LOD were imputed by dividing the LOD by the square root of 2 (Hornung and Reed, 1990). PFHxS was detected in only 4% of the samples, which was too low for statistical analysis; therefore, it was excluded from the association analysis. Owing to the skewed distribution of PFAS concentrations in breast milk, a natural logarithmic (ln) transformation was applied to the chemical concentration data. Spearman's correlation tests and multiple linear regression analyses were conducted to explore the associations between the concentrations of chemicals in breast milk and the indices of infant growth. Stratified analyses were conducted to assess the association between PFAS exposure and infant growth across subgroups defined by maternal age (≥ 35 years and < 35 years), parity status (multiparous and primiparous), and maternal BMI (≥ 25 kg/m^2 and < 25 kg/m^2) categories. To evaluate whether the associations differed by infant sex, the interaction term between sex and PFAS exposure was included in the multiple linear regression models. To allow for a direct comparison of effect sizes, the PFAS concentrations were natural log-transformed and standardized prior to analysis. All the variables were included after verifying the absence of multicollinearity. SPSS version 24.0 (IBM SPSS, Armonk, NY, USA) and R software (The R Foundation, Vienna, Austria) were used for all statistical analyses and graphics.

Table 1: Socioeconomic characteristics of participants (n=201)

Variables	Categories	n/ M	%/SD	Median	Min	Max
Maternal age (years)		34.09	3.47	34	25	43
Maternal BMI (kg/m2)		23.17	2.86	22.81	16.8	32.66
Education	< college	44	21.9			
	≥ college	157	78.1			
Household income ($/month)	≤5,000	94	46.8			
	> 5,000	107	53.2			
Employment status	Yes	117	58.2			
	No	84	41.8			
Residence area	Metropolitan	108	53.7			
	Non-metropolitan	93	46.3			
Parity	Primipara	129	64.2			
	Multipara	72	35.8			
Infant sex	Boys	95	47.3			
	Girls	106	52.7			
Age (months)		2.4	1.5	2.1	0.1	5.9
Weight (kg)		5.6	1.6	5.5	3	9.4
Height (cm)		58.5	6.1	58	42	84
Birth weight (kg)		3.2	0.4	3.2	2.1	4.3
Birth height (cm)		49.7	2.56	50	36.5	55
Breast milk intake (mL/day)		881.5	221.5	900	70	1400

M, mean; SD, standard deviation; Min, minimum; Max, maximum; BMI, body mass index.

Moon N, et al., Fortune J Health Sci 2026
DOI:10.26502/fjhs.403

Results

Study participants

Table 1 presents the general characteristics of the mother-infant dyads who participated in this study. The average age of the 201 mothers was 34.1 years (range: 25–43 years). The mean BMI of the mothers was 23.2 kg/m², and approximately 78% reported having a college education or higher. More than half of the mothers reported a monthly household income exceeding $5,000, were employed, and lived in metropolitan areas. Additionally, approximately 64% of the mothers were primiparous. Of the 201 infants, 95 were male and 106 were female, with an average age of 2.4 months. The average weight and height of the infants were 5.6 kg and 58.5 cm, respectively. The mean birth weight and height were 3.2 kg and 49.7 cm, respectively.

Concentrations of PFAS in breast milk

Table 2 presents the concentrations of the five PFASs detected in the breast milk samples collected from the participating mothers. The five long-chain PFASs included PFDeA, PFHxS, PFNA, PFOA, and PFOS, with LOD of 0.017, 0.069, 0.013, 0.047, and 0.070 ng/mL, respectively. The detection rates in breast milk were the highest for PFOA (92%), followed by PFNA (77%), PFOS (46%), PFDeA (43%), and PFHxS (4%). The GM concentrations were highest for PFOA (0.098 ng/mL), followed by PFOS (0.085 ng/mL), PFHxS (0.051 ng/mL), PFNA (0.023 ng/mL), and PFDeA (0.021 ng/mL). We also compared the mean (SD) values of PFAS in breast milk according to maternal age, parity, and BMI (Supplementary Table 1). Although the PFOA concentrations in the primiparous (0.127 ng/mL) and BMI ≥ 25 kg/m² group (0.139 ng/mL) were higher than those in the multiparous (0.103 ng/mL) and BMI < 25 kg/m² groups, respectively, the differences were not statistically significant (p = 0.080 and 0.086).

Daily intake of PFAS in breastfed infants and sensitivity analysis

We calculated the EDI of PFAS in breastfed infants based on their individual breast milk consumption and body weight (Figure 1). The range of infants' body weight and daily breast milk intake were 3.5–9.4 kg, and 70–1,400 mL/day, respectively. The mean (SD) EDI values of each PFAS were 4.59 (6.79) ng/kg bw/day for PFDeA, 4.36 (4.15) ng/kg bw/day for PFNA, 17.53 (19.49) ng/kg bw/day for PFOA, and 13.37 (14.61) ng/kg bw/day for PFOS. The median (min–max) EDI values of each PFAS were 2.35 (0.19–44.73) ng/kg bw/day for PFDeA, 3.44(0.15–29.98) ng/kg bw/day for PFNA, 13.20 (0.78–151.21) ng/kg bw/day for PFOA, and 9.04 (0.05–120.82) ng/kg bw/day for PFOS. In addition, we conducted a sensitivity analysis to compare the relative importance of each variable. The PFAS concentration in breast milk had the strongest positive effect on EDI, followed by breast milk intake, whereas infant body weight exerted a negative effect on EDI.

Associations between PFAS concentrations in breast milk and infant growth

We evaluated the correlations between the natural log-transformed PFAS concentrations in breast milk and the WFL values of the infants (Supplementary Figure 1). All four PFASs in breast milk showed weak negative correlations with the infant WFL values; however, these correlations were not statistically significant. In contrast, positive correlations among the PFASs were statistically significant. Table 3 and Figure 2 present the associations between PFAS concentrations in breast milk and the WFL of 201 infants according to subgroup and sex. In the sub-group of maternal age ≥ 35 years, the one unit increase in PFOA (β = –0.30, 95% CI = –0.51, –0.08) and PFOS (β = –0.28, 95% CI = –0.49, –0.06) concentrations in breast milk were significantly associated with lower WFL values in both male and female infants combined. In the same age group, although only male infants showed a similar association between PFOA (β = –0.42, 95% CI = –0.72, –0.12) concentrations and WFL, the interaction effect of infant sex was not statistically significant (p = 0.226). In the sub-group of maternal BMI ≥25 kg/m², the one unit increase in PFOS (β = –0.55, 95% CI = –0.09, –0.17) and PFNA (β = –0.90, 95% CI = –1.53,

Table 2: Concentrations of Per- and Polyfluoroalkyl Substances (PFAS) in breast milk (ng/mL, n=201)

Analytes	LOD	DF (%)	GM	Min	5th	25th	50th	75th	95th	Max
PFDeA	0.017	43	0.021	< LOD	< LOD	< LOD	< LOD	0.028	0.125	1.572
PFHxS	0.069	4	0.051	< LOD	< LOD	< LOD	< LOD	< LOD	< LOD	0.849
PFNA	0.013	77	0.023	< LOD	< LOD	0.014	0.023	0.036	0.073	0.26
PFOA	0.047	92	0.098	< LOD	< LOD	0.068	0.097	0.144	0.261	0.945
PFOS	0.07	46	0.085	< LOD	< LOD	< LOD	< LOD	0.104	0.164	0.755

PFDeA, Perfluorodecanoic acid; PFHxS, Perfluorohexane sulfonic acid; PFNA, Perfluorononanoic acid; PFOA, Perfluorooctanoic acid; PFOS, Perfluorooctane sulfonic acid; LOD, Limit of detection; DF, detection frequency; GM, Geometric mean; Min, Minimum; Max, Maximum.

Moon N, et al., Fortune J Health Sci 2026
DOI:10.26502/fjhs.403

–0.28) concentrations were significantly associated with lower WFL in both male and female infants combined. In the same BMI group, only male infants showed a similar trend of significant association between PFOS (β = –0.53, 95% CI = –0.91, –0.16) and PFNA (β = –0.60, 95% CI = –0.99, –0.20) concentrations and WFL. The interaction effect of infant sex in this group was statistically significant ($p = 0.024$ for PFOS and $p = 0.006$ for PFNA).

Table 3: Associations between concentration of Polyfluoroalkyl Substances (PFAS) in breast milk and infant growth (weight for length) according to maternal age, parity, and BMI

Groups	Sub-groups	PFAS (per ln)	Both sexes combined	*p*	Males only	*p*	Females only	*p*	*p*-value for interaction
			β (95% CI)		β (95% CI)		β (95% CI)		
Maternal age	≥35 years old	PFOA	**–0.30 (–0.51, –0.08)**	**0.007**	**–0.42 (–0.72, –0.12)**	**0.007**	–0.22 (–0.54, 0.10)	0.164	0.226
		PFOS	**–0.28 (–0.49, –0.06)**	**0.014**	–0.25 (–0.57, 0.07)	0.128	–0.31 (–0.63, <–0.001)	0.05	0.885
		PFNA	–0.08 (–0.37, 0.22)	0.618	–0.28 (–0.64, 0.08)	0.119	0.1 (–0.25, 0.45)	0.578	0.347
		PFDeA	–0.19 (–0.42, 0.03)	0.09	–0.22 (–0.57, 0.14)	0.217	–0.10 (–0.42, 0.22)	0.544	0.579
	< 35 years old	PFOA	–0.05 (–0.27, 0.17)	0.662	–0.16 (–0.43, 0.12)	0.256	0.14 (–0.17, 0.45)	0.364	0.33
		PFOS	–0.07 (–0.29, 0.16)	0.556	–0.10 (–0.37, 0.18)	0.49	–0.01 (–0.32, 0.29)	0.93	0.26
		PFNA	–0.18 (–0.39, 0.03)	0.099	**–0.27 (–0.53, –0.003)**	**0.047**	0.04 (–0.25, 0.32)	0.8	0.396
		PFDeA	0.16 (–0.05, 0.37)	0.125	0.18 (–0.09, 0.45)	0.185	0.15 (–0.14, 0.43)	0.306	0.247
BMI	≥25kg/m^2	PFOA	–0.38 (–0.85, 0.09)	0.113	–0.41 (–0.89, 0.08)	0.094	–0.11 (–0.60, 0.37)	0.627	0.171
		PFOS	**–0.55 (–0.09, –0.17)**	**0.006**	**–0.53 (–0.91, –0.16)**	**0.008**	–0.19 (–0.69, 0.30)	0.419	**0.024**
		PFNA	**–0.90 (–1.53, –0.28)**	**0.006**	**–0.60 (–0.99, –0.20)**	**0.005**	–0.05 (–0.55, 0.45)	0.835	**0.006**
		PFDeA	–0.24 (–0.76, 0.27)	0.342	–0.07 (–0.67, 0.54)	0.819	–0.19 (–0.66, 0.27)	0.391	0.624
	< 25kg/m^2	PFOA	–0.14 (–0.30, 0.03)	0.096	–0.17 (–0.40, 0.06)	0.143	–0.12 (–0.36, 0.12)	0.324	0.827
		PFOS	–0.11 (–0.28, 0.05)	0.17	–0.11 (–0.34, 0.12)	0.342	–0.13 (–0.37, 0.11)	0.279	0.804
		PFNA	–0.14 (–0.31, 0.03)	0.105	–0.03 (–0.27, 0.20)	0.782	**–0.24 (–0.48, –0.01)**	**0.049**	0.534
		PFDeA	0.04 (–0.13, 0.21)	0.665	0.11 (–0.12, 0.34)	0.334	–0.02 (–0.27, 0.23)	0.849	0.602
Parity	Primipara	PFOA	–0.14 (–0.34, 0.06)	0.179	–0.13 (–0.38, 0.13)	0.322	–0.18 (–0.46, 0.09)	0.189	0.958
		PFOS	–0.20 (–0.39, 0.001)	0.051	–0.21 (–0.46, 0.04)	0.1	–0.17 (–0.44, 0.10)	0.209	0.427

Citation: Dr. Pratyush Kumar, Dr. Amitrajit Pal, Dr. Dattatray Pawar, Dr. Akhilesh Sharma. A Retrospective Real-World Evidence Study Evaluating the Clinical Effectiveness and Safety of Cefixime in Managing Typhoid Fever in Indian Population. Fortune Journal of Health Sciences. 9 (2026): 132-148.

Moon N, et al., Fortune J Health Sci 2026
DOI:10.26502/fjhs.403

		PFNA	–0.12 (–0.35, 0.11)	0.3	–0.03 (–0.30, 0.23)	0.8	–0.18 (–0.45, 0.09)	0.195	0.972
		PFDeA	–0.03 (–0.21, 0.16)	0.794	–0.06 (–0.33, 0.21)	0.664	0.08 (–0.18, 0.35)	0.537	0.324
	Multipara	PFOA	–0.02 (–0.28, 0.24)	0.877	–0.12 (–0.48, 0.24)	0.497	0.09 (–0.30, 0.49)	0.637	0.304
		PFOS	0.01 (–0.27, 0.29)	0.953	–0.11 (–0.47, 0.25)	0.554	0.03 (–0.40, 0.47)	0.875	0.434
		PFNA	–0.06 (–0.31, 0.18)	0.605	–0.09 (–0.44, 0.26)	0.603	0.08 (–0.31, 0.48)	0.675	0.414
		PFDeA	–0.46 (–1.15, 0.24)	0.192	0.18 (–0.15, 0.52)	0.278	–0.23 (–0.63, 0.17)	0.24	0.101

PFOA, Perfluorooctanoic acid; PFOS, Perfluorooctane sulfonic acid; PFNA, Perfluorononanoic acid; PFDeA, Perfluorodecanoic acid; ln, natural log; β, standardized coefficient; CI, confidence interval, *p, p*-value; bold values mean statistically significant results at p < 0.05 level; Multiple linear regression after adjusting covariates including infant's age, current infant supplement intake, maternal education level, and residential area.

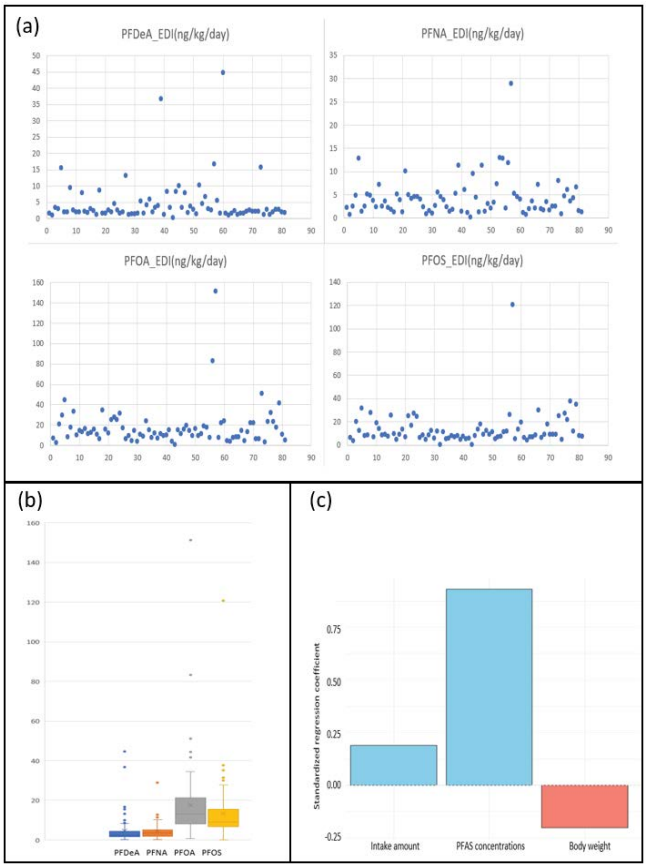


Figure 1: Estimated daily intake (EDI) of PFAS in breastfed infants aged under 6 months and sensitivity analysis results PFDeA, Perfluorodecanoic acid; PFHxS, Perfluorohexane sulfonic acid; PFNA, Perfluorononanoic acid; PFOA, Perfluorooctanoic acid; PFOS, Perfluorooctane sulfonic acid; (a) scatter plots presenting EDI values of each PFAS (b) box plots presenting EDI values of each PFAS (c) sensitivity analysis results of variables influencing EDI.

Discussion

Temporal Trends in PFAS Concentrations and Estimated Daily Intake (EDI) in Breast Milk

Following the adoption of the Stockholm Convention by the United Nations Environment Program (UNEP) in 2001, the Korean government introduced the "POPs Control Act" in 2008 to regulate the production and emission of key PFAS compounds, a framework that remains in place today (Kim, 2014; Fiedler et al., 2022). However, the findings of this study indicate that PFAS concentrations in breast milk have either remained similar or increased compared to previous domestic research. For PFOA, prior studies analyzing breast milk samples between 2007 and 2018 reported a consistent increase in the arithmetic mean or GM concentrations, ranging from 41 pg/mL to 0.114 ng/mL; however, a slight decrease was observed in the present study (Kim et al., 2011; Kang et al., 2016; Lee et al., 2018; Kim et al., 2023). Although the LOD in this study was relatively higher than those in previous reports, the arithmetic means or GM concentrations showed an upward trend from 61 pg/mL and 0.058 ng/mL in 2007 and 2018, respectively, to 0.085 ng/mL in this study (Kim et al., 2011; Kim et al., 2023). For PFNA, the median concentration declined from 17.1 ng/L in 2012 to 0.007 ng/mL in 2018 but subsequently increased to 0.023 ng/mL in this study (Lee et al., 2018; Kim et al., 2023). Similarly, PFDeA was previously reported to have GM concentrations of 0.0009 and 0.007 ng/mL in 2012 and 2018, respectively, yet increased significantly to 0.021 ng/mL in the present study (Lee et al., 2018; Kim et al., 2023). These findings highlight that despite the prolonged enforcement of PFAS regulations in Korea, maternal exposure levels have continued to increase, likely reflecting the persistent and bioaccumulative characteristics of these substances.

Moon N, et al., Fortune J Health Sci 2026
DOI:10.26502/fjhs.403

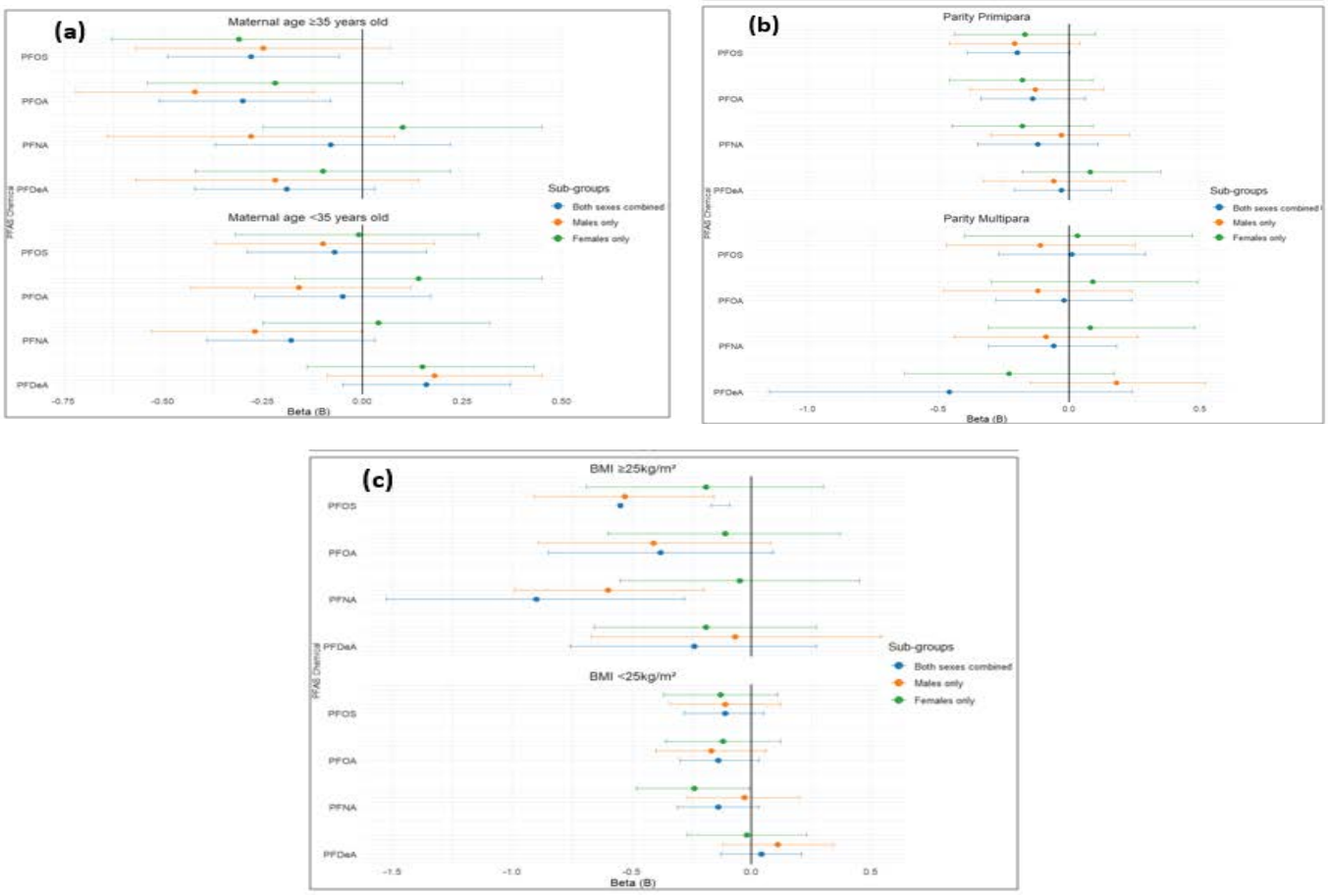


Figure 2: Forest plots of changes in infants' weight for length associated with a one unit increase in natural log of PFAS concentrations in breast milk, stratified by maternal age, parity, and body mass index.

PFDeA, Perfluorodecanoic acid; PFHxS, Perfluorohexane sulfonic acid; PFNA, Perfluorononanoic acid; PFOA, Perfluorooctanoic acid; PFOS, Perfluorooctane sulfonic acid; (a) comparison of infant growth (weight for length, WFL) by maternal age groups (35 years old) (b) comparison of infant WFL by maternal parity (c) comparison of infant WFL by maternal body mass index 25kg/m^2

PFAS concentrations in the breast milk of Korean mothers were comparable to or higher than those reported in other countries (Supplementary Table 2). The GM concentration of PFOA in breast milk observed in this study was higher than that reported in Australia (0.03 ng/L; Hartmann et al., 2024), Lebanon (34.29 ng/L; Mahfouz et al., 2023), Spain (8.2 pg/mL; Rovira et al., 2022), Brazil (22.1 pg/g; Fiedler et al., 2022), Canada (41.4 ng/L; Rawn et al., 2022), and France (0.041 ng/mL; Cariou et al., 2015). However, it was lower than the concentration reported in China (0.35 ng/mL; Li et al., 2024). Similarly, the PFOS GM concentration in this study exceeded those recorded in China (0.05 ng/mL; Li et al., 2024), Australia (0.015 ng/L; Hartmann et al., 2024), Lebanon (79.3 ng/L; Mahfouz et al., 2023), Spain (31 pg/mL; Rovira et al., 2022), Brazil (24.4 pg/g; Fiedler et al., 2022), Canada (35.7 ng/L; Rawn et al., 2022), and France (0.04 ng/mL; Cariou et al., 2015). The PFNA GM concentration found in this study was higher than those reported in China (0.01 ng/mL; Li et al., 2024), Lebanon (1.26 ng/L; Mahfouz et al., 2023), Spain (6.8 pg/mL; Rovira et al., 2022), Brazil (2.11 pg/g; Fiedler et al., 2022), and Canada (10.4 ng/L; Rawn et al., 2022). In contrast, the PFDeA GM concentration measured in this study was comparable to that reported in a recent study from China (0.03–0.07 ng/mL; Zhang et al., 2024) but was higher than the value reported in a previous domestic study (0.007 ng/mL; Kim et al., 2023). As ingestion is the primary route of PFAS exposure, this trend can be interpreted based on dietary and lifestyle factors (Susmann et al., 2019; Hung et al., 2020). Korean dietary habits include a higher consumption of various seafood products, such as fish, seaweed, shellfish, and cephalopods, compared with other populations (Hung et al., 2020; Huh et al., 2024). Furthermore, PFAS can accumulate throughout the food chain, leading to higher concentrations in commonly consumed predatory fish and other marine organisms (Bedi et al., 2023; Huh et al., 2024). Consequently, seafood has consistently been reported as a significant dietary source of PFAS exposure (Bedi et al., 2023; Crawford et al., 2024). Korean mothers

Moon N, et al., Fortune J Health Sci 2026
DOI:10.26502/fjhs.403

tend to consume greater quantities of seaweed-based foods after childbirth to replenish iron and other essential nutrients (Hwang et al., 2022). This postpartum dietary pattern may contribute to maternal PFAS exposure, as previous studies have identified seafood, including seaweed, as a potential bioaccumulator of these compounds (Huo et al., 2023; Wang et al., 2024). Additionally, Korea's highly industrialized and convenience-driven lifestyle has led to the widespread use of consumer products, single-use utensils, takeout containers, and food packaging materials containing coating agents (Kang et al., 2016; Serrano et al., 2021). Previous studies have shown a significant association between maternal seafood consumption, the frequent use of disposable products and coated food containers, and elevated PFAS concentrations in breast milk (Kang et al., 2016; Mahfouz et al., 2023; Kim et al., 2023). These findings suggest that dietary habits and daily exposure sources play crucial roles in the relatively high PFAS levels observed in Korean mothers.

PFAS EDI through Breast milk

The EDI values of PFOA, PFOS, and PFNA in breastfed infants vary across countries. In China, Zhang et al. (2024) reported a mean (range) EDI of 36.5 (0.52–291.7) ng/kg bw/day, while Xu et al. (2024) showed a declining trend in median EDI values by age group, with 162 ng/kg bw/day for infants under one month, 151 ng/kg bw/day for those 1–3 months, 119 ng/kg bw/day for those 3–6 months, and 89.6 ng/kg bw/day for those 6–12 months. In Australia, the reported EDI for PFOA was 28 (4.1–84) ng/kg bw/day (Hartmann et al., 2024). In contrast, a previous Korean study reported EDI values below 2.0 ng/kg bw/day (Kim et al., 2023), which are lower than the mean PFOA EDI observed in this study. The PFOS EDI values here exceeded those reported previously. In China, Zhang et al. (2024) reported a mean (range) PFOS EDI of 5.21 (0.26–32.3) ng/kg bw/day, and Xu et al. (2024) observed a declining trend over time, with median values of 18 ng/kg bw/day for infants under one month, 16.8 ng/kg bw/day for those 1–3 months, 13.2 ng/kg bw/day for those 3–6 months, and 9.96 ng/kg bw/day for those 6–12 months. In Australia, Hartmann et al. (2024) reported a mean and median PFOS EDI of 14 and 11 ng/kg bw/day, respectively. This study found comparable or higher EDI values for PFNA than those reported in previous studies. Hartmann et al. (2024) documented a mean and median PFNA EDI of 3.0 and 2.1 ng/kg bw/day, respectively, in Australia. In China, the EDI values reported by Xu et al. (2024) were lower than those in this study, with median values of 1.5 ng/kg bw/day for infants under one month, 1.4 ng/kg bw/day for those 1–3 months, 1.1 ng/kg bw/day for those 3–6 months, and 0.83 ng/kg bw/day for those 6–12 months. These comparisons indicate that the PFOS and PFNA EDI values in our study were higher than those reported in several international studies, whereas PFOA showed mixed trends depending on the country. Notably, the mean EDI values for PFOA, PFOS, and PFNA in this study were higher compared with a previous domestic study in Korea, highlighting the potential differences in exposure sources and maternal PFAS burdens over time.

We conducted a sensitivity analysis to comprehensively interpret the effects of PFAS exposure on infant growth and the relative comparison of daily oral exposure through breastfeeding. The analysis revealed that the PFAS concentration in breast milk was the most influential factor affecting oral exposure levels, which aligns with the higher PFOS and PFNA concentrations observed in this study than in previous studies. Given these findings, reducing the contaminant concentrations in orally consumed food and water sources is the most critical factor in minimizing the risk of exposure to pollutants in infants. To achieve this, institutional and societal efforts are essential for implementing effective policies and strategies aimed at lowering contamination levels through the oral pathway. As part of these efforts, it is crucial to recognize that recent international guidelines have introduced strict tolerable weekly intake (TWI) limits for PFAS. The European Food Safety Authority (EFSA) has set a combined threshold of 4.4 ng/kg bw/week for PFOA, PFOS, PFHxS, and PFNA, reflecting heightened regulatory concerns over PFAS accumulation and potential health risks (EFSA, 2020). Both this study and previous studies reported exposure levels that substantially exceeded this benchmark. Given that infants under one year of age have low body weight and rapid growth rates, there is a pressing need for more rigorous regulatory measures and enhanced monitoring to mitigate potential health risks. In particular, previous studies assessing PFAS-related health risks have applied older international standards, such as the 2008 EFSA guidelines, which allowed much higher exposure limits of 150 ng/kg bw/day for PFOS and 1500 ng/kg bw/day for PFOA (EFSA, 2008; Černá et al., 2020). The substantial gap between outdated and current guidelines underscores the necessity for future research to adopt an updated TWI to ensure a more precise and conservative risk assessment.

Association between PFAS exposure and infant growth: role of maternal and infant factor

In this study, PFOS and PFNA concentrations in breast milk from obese mothers were negatively associated with infant growth under six months of age, with significant differences observed based on infant sex. According to a previous study, mothers with high PFAS levels exhibited altered lipid profiles in breast milk, which were negatively associated with weight gain in infants under six months of age (Lamichhane et al., 2021). Similarly, in a Chinese study, C_8–C_{10} perfluorocarboxylic acids were significantly correlated with length gain rate in five-month-old infants (Jin et al., 2020). Moreover, some studies revealed a significant

Moon N, et al., Fortune J Health Sci 2026
DOI:10.26502/fjhs.403

association between maternal blood PFAS concentrations and infant growth, particularly sex-specific differences (Starling et al., 2019; Gao et al., 2022; Zhang et al., 2024). A study in the United States reported that maternal blood PFOS and PFHxS concentrations were associated with lower WFL in female infants (Starling et al., 2019). Another Chinese cohort study found that boys showed higher sensitivity to prenatal maternal blood PFAS exposure than girls in terms of weight-for-age z-scores and WFL trajectories (Gao et al., 2022). Based on the findings of this study and previous studies, two major aspects emerged as crucial for the risk assessment and intervention of PFAS in both mothers and infants. First, the finding that the effects of PFAS exposure through breast milk were significant only in infants born to obese mothers highlights the critical role of maternal metabolic health in PFAS transfer and metabolism. Obese individuals often exhibit altered lipid metabolism and systemic inflammation, which may influence PFAS distribution and excretion (Monteiro and Azevedo, 2010; Fuentes et al., 2013; Tan et al., 2023). The mechanisms driving increased PFAS transfer in obese mothers remain unclear. However, potential explanations include greater PFAS mobilization from the adipose tissue during lactation, leading to higher levels in breast milk; altered hepatic metabolism affecting PFAS bioaccumulation; and chronic low-grade inflammation in obesity, modifying PFAS binding affinity and transport (Tan et al., 2023; Cinzori et al., 2024; Vujic et al., 2024). These findings emphasize the necessity of considering maternal metabolic status in risk evaluations and suggest that obese mothers may constitute a high-risk group for PFAS-mediated health effects in infants.

In this study, PFAS concentrations in breast milk and infant growth showed some association with maternal age but no significant relationship with parity. Previous research has shown that for each increase in maternal age, the daily PFOS intake through breastfeeding in infants increased by 0.086 ng/kg (van Beijsterveldt et al., 2022). In that study, researchers concluded that older mothers had higher PFAS concentrations in their serum due to the bio-accumulation of PFAS, resulting in higher levels in breast milk (Luzardo et al., 2019; van Beijsterveldt et al., 2022). Another study by Kingsley et al. (2018) found that maternal age at delivery was positively associated with PFAS levels in infant serum but showed no association with maternal serum PFAS levels, suggesting a potential effect of breastfeeding (Kingsley et al., 2018). Parity is a factor that should be considered along with maternal age. In this study, no clear association was found between PFAS concentrations, infant growth, and maternal parity. However, previous studies reported some degree of association. For example, according to a previous study, women with more than one child had lower serum PFAS levels than primiparous mothers; however, no further reduction in PFAS concentrations was observed after the first child (Kingsley et al., 2018). The study also found that children born second or later in birth order had significantly lower serum PFAS levels than those born first (Kingsley et al., 2018). A Canadian study reported that primiparous and younger mothers had higher PFAS concentrations in their breast milk than multiparous and older mothers (Rawn et al., 2022). Similarly, a domestic study revealed a significant correlation between maternal parity and PFAS concentrations in breast milk (Lee et al., 2018). According to a previous review that discussed these conflicting findings regarding maternal characteristics, maternal PFAS levels may increase with age due to a decline in kidney function (McAdam and Bell, 2023). However, this effect could be counterbalanced by a higher number of pregnancies and breastfeeding episodes, which facilitate PFAS excretion (McAdam and Bell, 2023). This suggests a complex relationship between maternal age and PFAS concentrations. Considering these findings, maternal age and parity may influence PFAS concentrations and infant growth in a complex manner. Therefore, women of reproductive age should actively take steps to reduce their PFAS exposure before pregnancy. This is particularly important for first-time mothers, as they may transfer accumulated PFAS to their firstborn. To mitigate this risk, both individual and societal efforts are necessary to minimize overall PFAS exposure before conception.

The observation that PFAS exposure through breastfeeding influences infant growth, particularly in a sex-specific manner, and is notably pronounced in infants born to obese mothers underscores the importance of a refined approach for evaluating PFAS-related health risks, taking into account both biological sex differences and maternal metabolic conditions. PFASs are well-known endocrine disruptors that interfere with the thyroid and growth hormone axes, and are critical for infant growth and metabolism (Sun et al., 2022; Yao et al., 2022). According to previous studies, PFAS may enhance inflammatory responses by altering regulatory T-cell and Th17-cell activities, contributing to growth inhibition via the inflammation-mediated suppression of cellular growth and differentiation at the growth plates (Kirk et al., 2021; Liu et al., 2024). The observed sex-specific differences in sensitivity to PFAS exposure, with boys being more susceptible to growth impairment, may be attributed to differences in hormone regulation and epigenetic responses (McComb et al., 2020). In previous studies, boys were shown to exhibit higher sensitivity to PFAS, potentially owing to differences in androgen receptor activity and slower maturation of the male immune system (Behr et al., 2018; McComb et al., 2020). Additionally, other studies have suggested that PFAS exposure may induce sex-specific epigenetic modifications that affect genes related to growth hormone signaling and insulin-like growth factor 1 (IGF-1) pathways, further influencing growth outcomes (Luo et al.,

2021; Cai et al., 2025). Another contributing factor may be lipid metabolism pathways (Szilagyi et al., 2020; Kirk et al., 2021). PFASs have been shown to interfere with peroxisome proliferator-activated receptor pathways, which regulate adipocyte differentiation and lipid metabolism during child growth and development (Szilagyi et al., 2020; Kirk et al., 2021). Overall, the observed sex- and obesity-related interactions with PFAS exposure have critical implications for public health strategies and regulatory frameworks. Current risk assessment models should incorporate sex-based analyses to capture differential vulnerabilities. Furthermore, guidelines for PFAS exposure mitigation should be tailored to high-risk populations, including obese mothers and their infants. Future studies should explore the precise mechanisms underlying these disparities, particularly in relation to endocrine-disrupting properties and metabolic influences.

Strengths and Limitations

This study identified the hidden influence of high-risk maternal factors (age, BMI, and parity) on the association between PFAS exposure and infant growth. These factors should be carefully considered when evaluating the causal relationships between PFAS exposure and infant growth. Additionally, a key strength of this study is that it focused on a cohort of healthy mother-infant pairs and relied on individual measurements rather than literature-based estimates. Specifically, we directly measured the PFAS concentrations in the breast milk of each mother, assessed the individual infant breast milk intake, and recorded the actual infant weight. This approach allowed for more precise and reliable findings compared with studies relying solely on estimated or population-level data. Nevertheless, this study had some limitations. First, PFAS concentrations in breast milk were measured only once, failing to account for temporal variations in exposure levels. Second, the sample size was relatively small (n = 201), which may have limited the generalizability of the findings to the entire Korean population. Future studies with longitudinal measurements and larger and more representative cohorts are required to better understand the long-term trends and broader implications of PFAS exposure during breastfeeding.

Conclusion

This study found that while PFAS concentrations in breast milk have decreased for some substances in Korea, those for certain compounds continued to increase, with levels remaining relatively high compared to those in other countries. Notably, PFOS and PFNA concentrations were higher in obese mothers, leading to more pronounced growth impairments in male infants. Maternal age was associated with PFAS levels, whereas parity did not exhibit a clear relationship. Strengthened regulations and targeted interventions for high-risk groups are essential to reduce PFAS exposure. From a public health perspective, reducing PFAS exposure in high-risk mothers, particularly in obese women, could contribute to better infant growth outcomes. Future research should focus on longitudinal studies and larger, more diverse cohorts to further clarify the long-term effects of PFAS exposure and to develop effective intervention strategies.

Glossary

BMI, Body mass index; GM, Geometric means; LOD, Limits of detection; PFASs, Per- and polyfluoroalkyl substances; PFDeA, Perfluorodecanoic acid; PFHxS, Perfluorohexane sulfonate; PFNA, Perfluorononanoic acid; PFOA, Perfluorooctanoic acid; PFOS, Perfluorooctane sulfonate; WFL, weight-for-length

Author contributions: CRediT

Ju Hee Kim: Conceptualization, Validation, Investigation, Resources, Writing – Original draft, Writing – review & editing, Supervision, Project administration.

Nalae Moon: Conceptualization, Methodology, Software, Validation, Formal analysis, Investigation, Data curation, Writing – Original draft, Writing – review & editing, Visualization.

SungChul Seo: Conceptualization, Validation, Supervision, Writing – Original draft, Writing – review & editing.

Funding: None.

Acknowledgments: None.

References

1. Bedi M, Sapozhnikova Y, Taylor RB, et al. Per- and polyfluoroalkyl substances (PFAS) measured in seafood from a cross-section of retail stores in the United States. J. Hazard. Mater 459 (2023): 132062.
2. Behr AC, Lichtenstein D, Braeuning A, et al. Perfluoroalkylated substances (PFAS) affect neither estrogen and androgen receptor activity nor steroidogenesis in human cells in vitro. Toxicol. Lett 291 (2018): 51–60.
3. Bernasconi S, Street ME, Iughetti L, et al. Chemical contaminants in breast milk: A brief critical overview. Glob. Pediatr 2 (2022).
4. Blomberg AJ, Haug LS, Lindh C, et al. Changes in perfluoroalkyl substances (PFAS) concentrations in human milk over the course of lactation: A study in Ronneby mother-child cohort. Environ. Res 219 (2023): 115096.
5. Brantsæter AL, Whitworth KW, Ydersbond TA, et al. Determinants of plasma concentrations of perfluoroalkyl

substances in pregnant Norwegian women. Environ. Int 54 (2013): 74–84.

6. Cai A, Portengen L, Govarts E, et al. Prenatal exposure to persistent organic pollutants and changes in infant growth and childhood growth trajectories. Chemosphere 314 (2023): 137695.
7. Cai D, Li SP, Guo YT, et al. Effects of serum insulin and insulin-like growth factor 1 levels on the association between fetal growth and per- and polyfluoroalkyl substance exposure based on a nested case-control study. Environ. Sci. Technol 59 (2025): 3841–3852.
8. Cariou R, Veyrand B, Yamada A, et al. Perfluoroalkyl acid (PFAA) levels and profiles in breast milk, maternal and cord serum of French women and their newborns. Environ. Int 84 (2015): 71–81.
9. Černá M, Grafnetterová AP, Dvořáková D, et al. Biomonitoring of PFOA, PFOS and PFNA in human milk from Czech Republic, time trends and estimation of infant's daily intake. Environ. Res 188 (2020): 109763.
10. Cinzori ME, Pacyga DC, Rosas L, et al. Associations of per- and polyfluoroalkyl substances with maternal metabolic and inflammatory biomarkers in early-to-mid-pregnancy. Environ. Res 250 (2024): 118434.
11. Costello E, Rock S, Stratakis N, et al. Exposure to per- and polyfluoroalkyl substances and markers of liver injury: A systematic review and meta-analysis. Environ. Health Perspect 130 (2022): 46001.
12. Crawford KA, Gallagher LG, Giffard NG, et al. Patterns of seafood consumption among New Hampshire residents suggest potential exposure to per- and polyfluoroalkyl substances. Expo. Health 16 (2024): 1501–1517.
13. Daniels SR, Hassink SG, Committee on Nutrition, et al. The role of the pediatrician in primary prevention of obesity. Pediatrics 136 (2015): e275–e292.
14. Fenton SE, Ducatman A, Boobis A, et al. Per- and polyfluoroalkyl substance toxicity and human health review: Current state of knowledge and strategies for informing future research. Environ. Toxicol. Chem 40 (2021): 606–630.
15. Fiedler H, Sadia M, Krauss T, et al. Perfluoroalkane acids in human milk under the global monitoring plan of the Stockholm Convention on Persistent Organic Pollutants (2008–2019). Front. Environ. Sci. Eng 16 (2022).
16. Fuentes E, Fuentes F, Vilahur G, et al. Mechanisms of chronic state of inflammation as mediators that link obese adipose tissue and metabolic syndrome. Mediators Inflamm 2013 (2013): 136584.
17. Gao Y, Luo J, Zhang Y, et al. Prenatal exposure to per- and polyfluoroalkyl substances and child growth trajectories in the first two years. Environ. Health Perspect 130 (2022): 37006.
18. Gomis MI, Vestergren R, Borg D, et al. Comparing the toxic potency in vivo of long-chain perfluoroalkyl acids and fluorinated alternatives. Environ. Int 113 (2018): 1–9.
19. Hartmann C, Kaiser AM, Moche W, et al. Persistent organic pollutants in Austrian human breast milk collected between 2013 and 2016. J. Xenobiot 14 (2024): 247–266.
20. Hoadley L, Watters M, Rogers R, et al. Public health evaluation of PFAS exposures and breastfeeding: A systematic literature review. Toxicol. Sci 194 (2023): 121–137.
21. Høyer BB, Bonde JP, Tøttenborg SS, et al. Exposure to perfluoroalkyl substances during pregnancy and child behaviour at 5 to 9 years of age. Horm. Behav 101 (2018): 105–112.
22. Huh SW, Cho SY, Yoon S, et al. Relationship between crustacean consumption and serum perfluoroalkyl substances (PFAS): The Korean National Environmental Health Survey (KoNEHS) cycle 4. Ann. Occup. Environ. Med 36 (2024): e12.
23. Hung MD, Jung HJ, Jeong HH, et al. Perfluoroalkyl substances (PFASs) in special management sea areas of Korea: Distribution and bioconcentration in edible fish species. Mar. Pollut. Bull 156 (2020): 111236.
24. Huo X, Liang W, Tang W, et al. Dietary and maternal sociodemographic determinants of perfluoroalkyl and polyfluoroalkyl substance levels in pregnant women. Chemosphere 332 (2023): 138863.
25. Hwang J, Shin D, Kim H, et al. Association of maternal dietary patterns during pregnancy with small-for-gestational-age infants: Korean Mothers and Children's Environmental Health (MOCEH) study. Am. J. Clin. Nutr 115 (2022): 471–481.
26. Hyötyläinen T, Ghaffarzadegan T, Karthikeyan BS, et al. Impact of environmental exposures on human breast milk lipidome in future immune-mediated diseases. Environ. Sci. Technol 58 (2024): 2214–2223.
27. Jensen RC, Andersen MS, Larsen PV, et al. Prenatal exposures to perfluoroalkyl acids and associations with markers of adiposity and plasma lipids in infancy: An Odense child cohort study. Environ. Health Perspect 128 (2020): 77001.
28. Jin H, Mao L, Xie J, et al. Poly- and perfluoroalkyl substance concentrations in human breast milk and their associations with postnatal infant growth. Sci. Total Environ 713 (2020): 136417.

29. Jusko TA, Oktapodas M, Palkovičová Murinová L, et al. Demographic, reproductive, and dietary determinants of perfluorooctane sulfonic (PFOS) and perfluorooctanoic acid (PFOA) concentrations in human colostrum. Environ. Sci. Technol 50 (2016): 7152–7162.

30. Kang H, Choi K, Lee HS, et al. Elevated levels of short carbon-chain PFCAs in breast milk among Korean women: Current status and potential challenges. Environ. Res 148 (2016): 351–359.

31. Kim JH, Moon N, Lee JW, et al. Time-course trend and influencing factors for per- and polyfluoroalkyl substances in the breast milk of Korean mothers. Chemosphere 310 (2023): 136688.

32. Kim SK, Lee KT, Kang CS, et al. Distribution of perfluorochemicals between sera and milk from the same mothers and implications for prenatal and postnatal exposures. Environ. Pollut 159 (2011): 169–174.

33. Kim SK, Yoon J, et al. Chronological trends of emission, environmental level and human exposure of POPs over the last 10 years (1999–2010) in Korea: Implication to science and policy. Sci. Total Environ 470–471 (2014): 1346–1361.

34. Kingsley SL, Eliot MN, Kelsey KT, et al. Variability and predictors of serum perfluoroalkyl substance concentrations during pregnancy and early childhood. Environ. Res 165 (2018): 247–257.

35. Kirk AB, Michelsen-Correa S, Rosen C, et al. PFAS and potential adverse effects on bone and adipose tissue through interactions with PPARgamma. Endocrinology 162 (2021): bqab194.

36. LaKind JS, Verner MA, Rogers RD, et al. Current breast milk PFAS levels in the United States and Canada: After all this time, why don't we know more? Environ. Health Perspect 130 (2022): 25002.

37. Lamichhane S, Siljander H, Duberg D, et al. Exposure to per- and polyfluoroalkyl substances associates with an altered lipid composition of breast milk. Environ. Int 157 (2021): 106855.

38. Lee S, Kim S, Park J, et al. Perfluoroalkyl substances (PFASs) in breast milk from Korea: Time-course trends, influencing factors, and infant exposure. Sci. Total Environ 612 (2018): 286–292.

39. Li Q, Zhang Y, Chen C, et al. Association between prenatal exposure to per- and poly-fluoroalkyl substances from electronic waste disassembly areas and steroid hormones in human milk samples. GeoHealth 8 (2024): e2024GH001142.

40. Liu Y, Wang Y, Ren T, et al. Unraveling the long-term gastrointestinal impact of perinatal perfluorobutane sulfonate exposure on rat offspring: Intestinal barrier dysfunction and Th17/Treg imbalance. Sci. Total Environ 955 (2024): 176858.

41. Luo D, Wu W, Pan Y, et al. Associations of prenatal exposure to per- and polyfluoroalkyl substances with the neonatal birth size and hormones in the growth hormone/insulin-like growth factor axis. Environ. Sci. Technol 55 (2021): 11859–11873.

42. Luzardo OP, Badea M, Zumbado M, et al. Body burden of organohalogenated pollutants and polycyclic aromatic hydrocarbons in Romanian population: Influence of age, gender, body mass index, and habitat. Sci. Total Environ 656 (2019): 709–716.

43. Mahfouz M, Harmouche-Karaki M, Matta J, et al. Maternal serum, cord and human milk levels of per- and polyfluoroalkyl substances (PFAS), association with predictors and effect on newborn anthropometry. Toxics 11 (2023): 455.

44. McAdam J, Bell EM, et al. Determinants of maternal and neonatal PFAS concentrations: A review. Environ. Health 22 (2023): 41.

45. McComb J, Mills IG, Berntsen HF, et al. Human-based exposure levels of perfluoroalkyl acids may induce harmful effects to health by disrupting major components of androgen receptor signalling in vitro. Expo. Health 12 (2020): 527–538.

46. Mogensen UB, Grandjean P, Nielsen F, et al. Breastfeeding as an exposure pathway for perfluorinated alkylates. Environ. Sci. Technol 49 (2015): 10466–10473.

47. Mondal D, Weldon RH, Armstrong BG, et al. Breastfeeding: A potential excretion route for mothers and implications for infant exposure to perfluoroalkyl acids. Environ. Health Perspect 122 (2014): 187–192.

48. Monteiro R, Azevedo I, et al. Chronic inflammation in obesity and the metabolic syndrome. Mediators Inflamm 2010 (2010): 289645.

49. Ng CA, Hungerbühler K, et al. Bioconcentration of perfluorinated alkyl acids: How important is specific binding? Environ. Sci. Technol 47 (2013): 7214–7223.

50. Panieri E, Baralic K, Djukic-Cosic D, et al. PFAS molecules: A major concern for human health and the environment. Toxics 10 (2022): 44.

51. Papadopoulou E, Haug LS, Sakhi AK, et al. Diet as a source of exposure to environmental contaminants for pregnant women and children from six European countries. Environ. Health Perspect 127 (2019): 107005.

52. Pham PC, Taylor M, Nguyen GTH, et al. Binding of per- and polyfluoroalkyl substances to beta-lactoglobulin from bovine milk. Chem. Res. Toxicol 37 (2024): 757–770.

53. Rawn DFK, Dufresne G, Clément G, et al. Perfluorinated alkyl substances in Canadian human milk as part of the Maternal-Infant Research on Environmental Chemicals (MIREC) study. Sci. Total Environ 831 (2022): 154888.

54. Rovira J, Martínez MÁ, Mari M, et al. Mixture of environmental pollutants in breast milk from a Spanish cohort of nursing mothers. Environ. Int 166 (2022): 107375.

55. Serrano L, Iribarne-Durán LM, Suárez B, et al. Concentrations of perfluoroalkyl substances in donor breast milk in Southern Spain and their potential determinants. Int. J. Hyg. Environ. Health 236 (2021): 113796.

56. Starling AP, Adgate JL, Hamman RF, et al. Prenatal exposure to per- and polyfluoroalkyl substances and infant growth and adiposity: The Healthy Start Study. Environ. Int 131 (2019): 104983.

57. Starling AP, Engel SM, Whitworth KW, et al. Perfluoroalkyl substances and lipid concentrations in plasma during pregnancy among women in the Norwegian Mother and Child Cohort Study. Environ. Int 62 (2014): 104–112.

58. Sun M, Cao X, Wu Y, et al. Prenatal exposure to endocrine-disrupting chemicals and thyroid function in neonates: A systematic review and meta-analysis. Ecotoxicol. Environ. Saf 231 (2022): 113215.

59. Susmann HP, Schaider LA, Rodgers KM, et al. Dietary habits related to food packaging and population exposure to PFASs. Environ. Health Perspect 127 (2019): 107003.

60. Szilagyi JT, Avula V, Fry RC, et al. Perfluoroalkyl substances (PFAS) and their effects on the placenta, pregnancy, and child development: A potential mechanistic role for placental peroxisome proliferator–activated receptors (PPARs). Curr. Environ. Health Rep 7 (2020): 222–230.

61. Tan Y, Taibl KR, Dunlop AL, et al. Association between a mixture of per- and polyfluoroalkyl substances (PFAS) and inflammatory biomarkers in the Atlanta African American maternal-child cohort. Environ. Sci. Technol 57 (2023): 13419–13428.

62. Tao H, Zhang Y, Yao Q, et al. Per- and polyfluoroalkyl substances exposure through human milk, health risk assessment and physical growth in one-year-old children in Shandong, China. Environ. Chem. Ecotoxicol 7 (2025): 539–546.

63. van Beijsterveldt IALP, van Zelst BD, de Fluiter KS, et al. Poly- and perfluoroalkyl substances (PFAS) exposure through infant feeding in early life. Environ. Int 164 (2022): 107274.

64. Verner MA, Loccisano AE, Morken NH, et al. Associations of perfluoroalkyl substances with lower birth weight: An evaluation of potential confounding by glomerular filtration rate using a physiologically based pharmacokinetic model (PBPK). Environ. Health Perspect 123 (2015): 1317–1324.

65. Victora CG, Bahl R, Barros AJD, et al. Breastfeeding in the 21st century: Epidemiology, mechanisms, and lifelong effect. Lancet 387 (2016): 475–490.

66. Vujic E, Ferguson SS, Brouwer KLR, et al. Effects of PFAS on human liver transporters: Implications for health outcomes. Toxicol. Sci 200 (2024): 213–227.

67. Wang Y, Gui J, Howe CG, et al. Association of diet with per- and polyfluoroalkyl substances in plasma and human milk in the New Hampshire Birth Cohort Study. Sci. Total Environ 933 (2024): 173157.

68. Xu Y, Sui X, Li J, et al. Early-life exposure to per- and polyfluoroalkyl substances: Analysis of levels, health risk and binding abilities to transport proteins. Eco Environ. Health 3 (2024): 308–316.

69. Yao J, Dong Z, Jiang L, et al. Emerging and legacy perfluoroalkyl substances in breastfed Chinese infants: Renal clearance, body burden, and implications. Environ. Health Perspect 131 (2023): 37003.

70. Yao Q, Vinturache A, Lei X, et al. Prenatal exposure to per- and polyfluoroalkyl substances, fetal thyroid hormones, and infant neurodevelopment. Environ. Res 206 (2022): 112561.

71. Zhang X, Zhou X, Chen H, et al. Changes in concentrations of polyfluoroalkyl substances in human milk over lactation time and effects of maternal exposure via analysis of matched samples. Environ. Sci. Technol 58 (2024): 4115–4126.

72. Zhang Y, Pan C, Ren Y, et al. Association of maternal exposure to perfluoroalkyl and polyfluoroalkyl substances with infant growth from birth to 12 months: A prospective cohort study. Sci. Total Environ 806 (2022): 151303.

73. Zhou Y, Li Q, Wang P, et al. Associations of prenatal PFAS exposure and early childhood neurodevelopment: Evidence from the Shanghai Maternal-Child Pairs Cohort. Environ. Int 173 (2023): 107850.

Citation: Dr. Pratyush Kumar, Dr. Amitrajit Pal, Dr. Dattatray Pawar, Dr. Akhilesh Sharma. A Retrospective Real-World Evidence Study Evaluating the Clinical Effectiveness and Safety of Cefixime in Managing Typhoid Fever in Indian Population. Fortune Journal of Health Sciences. 9 (2026): 132-148.

Moon N, et al., Fortune J Health Sci 2026
DOI:10.26502/fjhs.403

Supplementary Table 1. Concentrations of Per- and Polyfluoroalkyl Substances (PFAS) and in breast milk and differences according to the sub-groups (maternal age, parity, and BMI) (n=201, ng/mL)

Sub-groups		Mean (SD)											
		PFDeA			PFNA			PFOA			PFOS		
Maternal age	≥ 35	<35	*p*	≥ 35	<35	*p*	≥ 35	<35	*p*	≥ 35	<35	*p*
	0.045 (0.087)	0.046 (0.163)	0.961	0.034 (0.038)	0.028 (0.022)	0.17	0.119 (0.088)	0.118 (0.098)	0.975	0.083 (0.057)	0.086 (0.077)	0.784
Parity	Primi	Multi	*p*	Primi	Multi	*p*	Primi	Multi	*p*	Primi	Multi	*p*
	0.045 (0.098)	0.046 (0.184)	0.956	0.033 (0.035)	0.026 (0.019)	0.134	0.127 (0.098)	0.103 (0.083)	0.08	0.089 (0.076)	0.077 (0.052)	0.201
Maternal pre-pregnancy BMI (kg/m^2)	≥ 25	< 25	*p*	≥ 25	< 25	*p*	≥ 25	< 25	*p*	≥ 25	< 25	*p*
	0.032 (0.054)	0.049 (0.151)	0.453	0.035 (0.043)	0.029 (0.025)	0.245	0.139 (0.134)	0.112 (0.077)	0.086	0.091 (0.110)	0.083 (0.051)	0.492

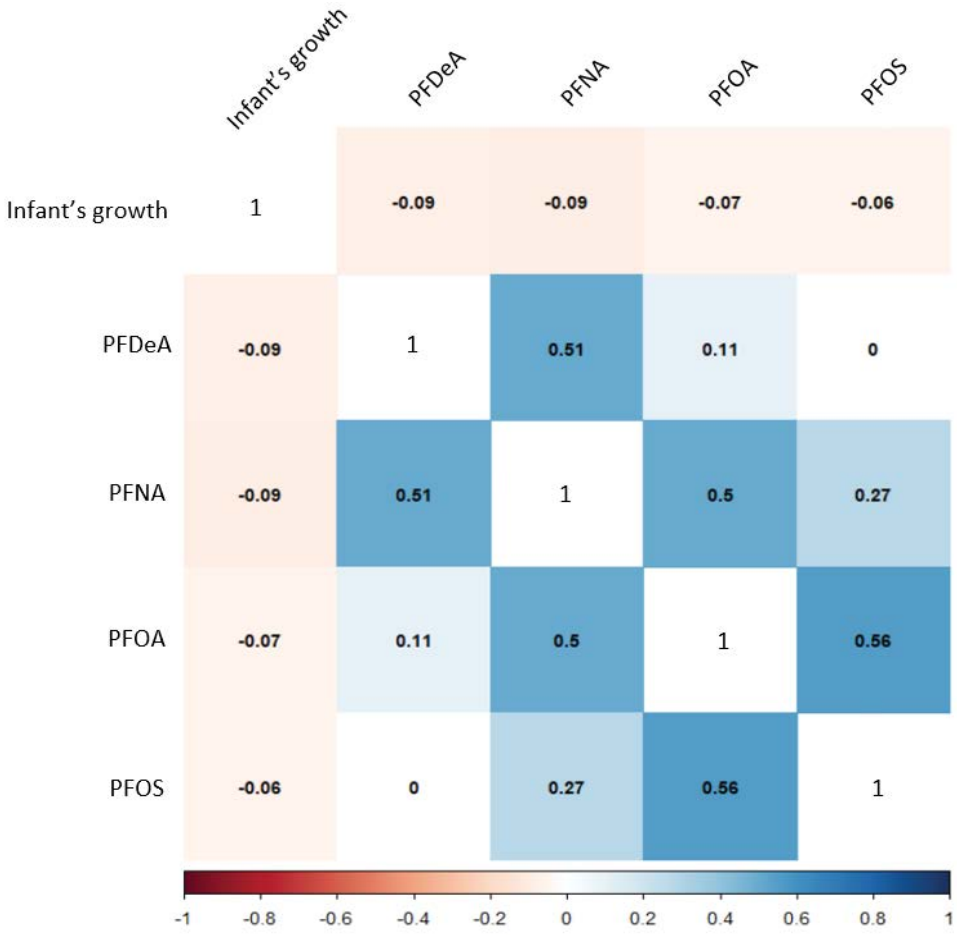
PFDeA, Perfluorodecanoic acid; PFHxS, Perfluorohexane sulfonic acid; PFNA, Perfluorononanoic acid; PFOA, Perfluorooctanoic acid; PFOS, Perfluorooctane sulfonic acid; BMI, body mass index; SD, standard deviation; p, p-value.

Supplementary Table 2: Comparison of concentrations of PFAS in breast milk reported in previous studies

No	Author (year)	Country	sample size	LOD or LOQ	unit	sampling period	PFOA				PFOS				PFNA				PFDeA			
							DF(%)	Mean (range)	med (IQR)	GM	DF(%)	Mean (range)	med (IQR)	GM	DF(%)	Mean (range)	med (IQR)	GM	DF(%)	Mean (range)	med (IQR)	GM
1	Zhang et al (2024)	China	76	LOD:	ug/L	0.5 month after delivery	92.1		0.11 (0.27)		96.1		0.11 (0.22)		23.7		<LOQ		76.3		0.03 (0.10)	
				0.003-0.098		1 month after delivery	94.7		0.16 (0.54)		98.7		0.22 (0.27)		46.1		<LOQ		60.5		0.03 (0.12)	
						3 months after delivery	100		0.14 (0.55)		100		0.22 (0.3)		47.4		<LOQ (0.03)		76.3		0.07 (0.17)	
2	Wang et al (2024)	China	243	LOD: 0.01	ng/mL	6 weeks after delivery			0.02 (0.01, 0.03)				0.02 (0.02-0.04)									
				LOQ 0.01~0.025																		
3	Li et al (2024)	China	150	LOD: 0.005-0.1	ng/mL	within 4 weeks after delivery	97.3	0.35			30.7	0.05			10	0.01			0	(n.d.-8.7)		
4	Xu et al (2024)	China	551	LOD: 0.01-0.11,	ng/mL	2-3 days after delivery	86.6		1.08 (0.41,2.32)	0.69	49.9		0.12 (-,0.47)	0.17	35.4		-, (-, 0.05)	0.02	35.4		n.d. (n.d., 0.05)	0.02
5	Hartmann et al (2024)	Australia	40	LOD: 0.01-13	ng/L	average 1 month after delivery	100	0.03 (<LOQ-0.91)	0.025		97.5	0.015 (<LOD-0.55)	0.012		90	(n.d.-15)			45			
6	Hall et al (2024)	Canada	664	LOD:	pg/g	2~10 weeks after delivery	99.7		34.1 (21.9, 51)	33.7	99.4		30 (21, 45.6)	30.1	66.9		6.1 (<LOD, 6.1)	5.6				
				05-Oct																		
7	Kim et al (2023)	Korea	207	LOQ: 0.003-0.033	ng/mL	within 3 months after delivery	100	(0.033-1.37)	0.1 (0.07, 0.14)	0.114	100	(0.017-0.512)	0.05 (0.04, 0.07)	0.058	87	(<LOQ-0.178)	0.007 (0.005,0.011)	0.009	100	(0.002-0.076)	0.007 (0.005, 0.009)	0.007
8	Mahfouz et al (2023)	Lebanon	41	LOD: 0.6,	ng/L	3-8 weeks after delivery		34.29	42			79.3	91			1.26	1.3					
				LOQ 0.3ng/mL																		
9	Rovira et al (2022)	Spain	60	LOD: 10	pg/mL	0~9 month after delivery	12	8.2	<10		87	31	28		25	6.8	<10		0			
10	Fiedler et al (2022)	Brazil	86	LOQ:	pg/g	NA		22.1 (<6.2-63.4)	18.6			24.4 (<6.2-212)	18.9			2.11				0.195 (0-8.68)	0	
				1.2-6.2																		
11	Rawn et al (2022)	Canada	664	LOD:	ng/L	2~10 weeks after delivery	99.5	41.4	34.2	33.8	95.9	35.7	30.2	30.6	61	10.4	7.6	8.2				
				05-Oct																		
12	Kang et al(2016)	Korea	264	LOQ:	ng/mL	two weeks after the delivery	98.5		0.07 (0.05, 0.11)	0.071	98.5		0.05 (0.03, 0.08)	0.049	27.3		<LOD (<LOD, 0.022)					
				0.1-2.0																		
13	Cariou et al(2015)	France	61	LOQ: 0.03-0.05	ng/mL	4th and 5th day after delivery	77	0.041 (<LOD-0.308)	<LOD		82	0.040 (<LOD-0.376)	<LOQ		0				0			

Citation: Dr. Pratyush Kumar, Dr. Amitrajit Pal, Dr. Dattatray Pawar, Dr. Akhilesh Sharma. A Retrospective Real-World Evidence Study Evaluating the Clinical Effectiveness and Safety of Cefixime in Managing Typhoid Fever in Indian Population. Fortune Journal of Health Sciences. 9 (2026): 132-148.

Moon N, et al., Fortune J Health Sci 2026
DOI:10.26502/fjhs.403



Supplementary Figure 1: Correlation between PFAS in breast milk and infant's growth - weigh for length (WFL). ln, natural log transformation; PFDeA, Perfluorodecanoic acid; PFHxS, Perfluorohexane sulfonic acid; PFNA, Perfluorononanoic acid; PFOA, Perfluorooctanoic acid; PFOS, Perfluorooctane sulfonic acid.