HHS Public Access

Author manuscript

Environ Pollut. Author manuscript; available in PMC 2020 March 01.

Published in final edited form as:

Environ Pollut. 2019 March ; 246: 482–490. doi:10.1016/j.envpol.2018.12.018.

Cord Blood Perfluoroalkyl Substances in Mothers Exposed to the World Trade Center Disaster During Pregnancy

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Abstract

Perfluoroalkyl substances (PFAS) may have been released during the collapse of the World Trade Center (WTC) on 9/11. Evidence suggests PFAS can cross the placental barrier in humans and cause harm to the developing fetus; however, no studies have measured PFAS in mothers exposed to the WTC disaster during pregnancy. We measured PFAS in maternal plasma (n=48) or cord blood (n=231) from pregnant women in the Columbia University WTC birth cohort, enrolled between December 13, 2001 and June 26, 2002 at one of three hospitals located near the WTC site. In order to maximize sample size, we used a linear regression to transform the 48 maternal plasma samples to cord blood equivalents in our study; cord blood and transformed maternal plasma-to-cord blood samples were then analyzed together. We evaluated the association between WTC exposure and PFAS concentrations using three exposure variables: 1) living/working within two miles of WTC; 2) living within two miles of WTC regardless of work location; and 3) working but not living within two miles of WTC. Exposure was compared with those not living/working within two miles of WTC (reference group). Living/working within two miles of WTC was associated with 13% higher PFOA concentrations compared with the reference group [GMR (95% CI): 1.13 (1.01, 1.27)]. The association was stronger when comparing only those who lived within two miles of WTC to the reference group [GMR (95% CI): 1.17 (1.03, 1.33)], regardless of

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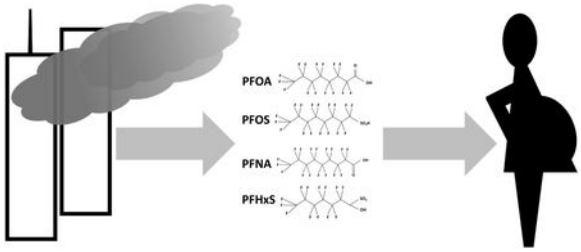
work location. Our results provide evidence that exposure to the WTC disaster during pregnancy resulted in increases in PFAS concentrations, specifically PFOA. This work identifies a potentially vulnerable and overlooked population, children exposed to the WTC disaster in utero; and highlights the importance of future longitudinal studies in this cohort to investigate later life effects resulting from these early life exposures.

Graphical Abstract



MAIN FINDING

Our work provides evidence that exposure to the World Trade Center disaster during pregnancy resulted in increases in cord blood concentrations of perfluorooctanoic acid (PFOA).

Keywords

perfluoroalkyl substances; cord blood; world trade center disaster

INTRODUCTION

It has been nearly two decades since the World Trade Center (WTC) disaster, yet questions remain regarding the health of those exposed to the dust, smoke and fumes caused by the collapse, as well as persistent fires that burned for over three months following the event. In addition to WTC responders, these concerns extend to the health of residents in lower Manhattan, particularly vulnerable populations such as pregnant women. The WTC plume was comprised of thousands of tons of toxic chemicals, many of which have been shown to adversely affect the fetus, including lead, particulate matter and numerous persistent organic pollutants (POPs).[1] Perfluoroalkyl substances (PFAS) were among various elevated POPs reported in personnel responding to the WTC disaster compared to nationally representative samples.[2] Further, a recent study reported higher PFAS in WTC-exposed children compared to a matched comparison group, providing evidence for WTC-related residential PFAS exposures.[3]

PFAS are a synthetic subgroup of organic compounds known for their oil and water repelling properties; these qualities have led to their widespread use as surfactants and stain-resistant coatings on numerous products, including carpets, food packaging, textiles, leather, cleaning products, pesticides, non-stick cookware and notably, in fire-fighting foam.[4] Due to their persistence in the environment and human health concerns, efforts have been made to reduce production of the two most widely used PFAS, perfluorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA).[4] In 2002, the main PFOS manufacturer in the US

voluntarily phased out PFOS production and in 2006, eight major PFAS companies joined a global stewardship program aimed at eliminating PFOA emissions.[4] Still, exposure to these compounds continues to be a pertinent public health issue as measurements of PFAS in humans remain at levels that have been associated with adverse health effects, including increases in lipids and liver enzymes, as well as cancer, immune suppression and thyroid disorders.[5, 6] Further, studies have shown PFAS can cross the placental barrier in humans and have been associated with reductions in fetal growth.[7, 8] Despite this, few studies have evaluated levels of PFAS in cord blood; just two of which were conducted in a US population.[9, 10] In this study, we analyzed four PFAS [PFOS, PFOA, perfluorohexanesulfonic acid (PFHxS) and perfluorononanoic acid (PFNA)] in the cord blood of a population at risk for high exposure to PFAS: women residing in lower Manhattan during and following the WTC disaster when fires continued to burn, releasing large quantities of toxic chemicals. We attempt to better understand the contribution of the WTC disaster to PFAS levels by evaluating their association with WTC exposure. Further, we add to the limited research characterizing trends in cord blood PFAS levels across sociodemographic subgroups in the US.

MATERIAL AND METHODS

Study Population

Data for this work came from a Columbia University birth cohort designed to study the effects of WTC exposures on pregnancy outcomes and development. Detailed methods have been described previously.[11] Briefly, 329 women with singleton pregnancies were enrolled between December 13, 2001 and June 26, 2002 at one of three hospitals located near the WTC site: Beth Israel, St. Vincent's, and New York University Downtown. Eligibility requirements included: ages between 18 and 39 years, had not smoked (>1 cigarette/at any time) during pregnancy, and self-report of no diabetes, hypertension, HIV infection or AIDS, and no use of illegal drugs in the last year. Participants provided at least one blood sample (maternal blood at the time of delivery and/or cord blood), access to their medical record and their newborn's medical record, and completion of a 30- to 45-minute interview after delivery. Participants missing data on PFAS concentrations in either cord blood or maternal plasma (n=27), race (n=19), body mass index (BMI) (n=3) and parity (n=1) were excluded, resulting in a sample size of 279 participants for analyses (Figure 1).

Sociodemographic and Exposure Variables

The postpartum interview was administered at the hospital in the woman's preferred or native language (English, Spanish, or Chinese). Information on maternal education, date of birth, race, parity, marital status, home smoking exposure and residential and work addresses was elicited through the questionnaire. Residential and work addresses (for the 4 weeks starting on and following 9/11) were geocoded at the Center for International Earth Science Information Network of Columbia University's Earth Institute, using geographic information system (GIS) software from Environmental Systems Research Institute (Redlands, CA), including ArcGIS 8.3 and the Street Map 2003 extension.[11] Using these data, multiple exposure categories were created: 1) women who lived within 2 miles of the WTC site regardless of where they worked; 2) women who worked but did not live within 2

miles of the WTC site; 3) women that lived or worked within 2 miles of the WTC site; and 4) women that neither worked nor lived within 2 miles of the WTC site (reference group). Two miles was selected to delineate the exposure radius based on previous findings of an association between this exposure group and birth outcomes;[11] as well as for consistency with the World Trade Center Health Registry definition of the WTC disaster area, which includes the area of Manhattan south of Houston Street and any block of Brooklyn that is within a 1.5-mile radius of the former World Trade Center site.[12] Maternal pre-pregnancy BMI was calculated using weight in kilograms divided by height in meters squared, both abstracted from participants' medical chart. In the case of missing height (n=36) or weight (n=49) from the medical record, self-reported information on these variables from the hospital interview were used. Child sex and date of birth were abstracted from child's medical record. Gestational age in days was also abstracted from the medical record (if missing (n=15), date of last menstrual period from interview minus child's date of birth was used). Gestational age on 9/11, used to determine trimester during the WTC disaster, was created by subtracting days since the 9/11 disaster on child's date of birth from child's gestational age in days at birth. Mothers were classified as being in their first trimester on 9/11 if their child had a gestational age of ≤91 days on 9/11, and in their second or third trimester if their child had a gestational age >91 days. 18 participants were not pregnant yet on 9/11 but still included in the study in the first trimester group, as exposures to the disaster persisted for months following the initial collapse. Maternal age at delivery was determined by subtracting the child's date of birth from the mother's date of birth.

PFAS Measurements

Twelve PFAS [PFOS; PFOA; perfluorobutanesulfonic acid (PFBS); perfluorohexane sulfonate (PFHxS); perfluorodecanesulfonate (PFDS); perfluorooctane sulfonamide (PFOSA); perfluorohexanoic acid (PFHxA); perfluoroheptanoic acid (PFHpA); perfluorodecanoic acid (PFDA); perfluoroundecanoic (PFUnDA), perfluorododecanoate (PFDoDA) and perfluorononanoic acid (PFNA)] were measured in maternal plasma (n=48) and cord blood (n=231) using a solid phase extraction (SPE) procedure and high-performance liquid chromatograph interfaced with an electrospray tandem mass spectrometer at the New York State Department of Health Wadsworth Center Laboratory, using methods similar to prior studies.[13, 14] Internal standards for 13 C-labeled PFAS were added into plasma samples prior to the addition of reagents for extraction.[15] Solvents and method blanks (blinded to the laboratory) were tested for the presence of the PFAS. Target chemicals were not found in procedural blanks at concentrations above the limits of quantification (LOQs). The LOQs of target chemicals ranged from 0.08 to 0.20 ng/mL. A standard reference material from the National Institute of Standards and Technology (NIST) was analyzed with every batch of 50 samples and recoveries of target chemicals were between 90% and 115% of the certified values. Recoveries of target chemicals passed through the entire analytical procedure ranged between 100% and 124%. Quantification was by isotope dilution and target chemicals were monitored by multiple reaction monitoring mode under negative ionization.

Statistical Analyses

All statistical analyses were conducted in R software (version 3.5.1; R Project for Statistical Computing). PFAS assessment was restricted to compounds quantified in ≥50% of samples (PFOS, PFOA, PFNA, PFHxS and PFDS). To maximize sample size, we used both maternal plasma and cord blood concentrations in analyses. However, to account for differences in maternal versus cord blood samples, we used 78 paired cord blood and maternal plasma PFAS samples from a US-based cohort with comparable PFAS concentrations, the HOME study[9], to create cord blood concentration predictions from our 48 maternal samples. We were unable to create prediction models from our own data because we did not have paired samples. Separate prediction models were run for PFOS, PFOA, PFNA and PFHxS (Supplemental Table 1). Prediction models were not available for PFDS because this compound was not measured in the HOME study and was therefore not included in the main analyses for this study despite being detected in >50% of samples. Exploratory analyses evaluating PFDS only in cord blood revealed no significant associations with the four WTC exposure variables defined previously (data not shown). To maximize sample size, all analyses report concentrations and associations using both transformed maternal plasma and cord blood concentrations together. PFOS, PFOA, PFNA and PFHxS were log-transformed to account for right-skewed distributions. Both PFOA and PFOS were detected in 100% of samples. Two samples (<1%) were below the LOQ (0.08 ng/ml) for PFHxS and 40 samples (14%) were below the LOQ (0.20 ng/ml) for PFNA. In accord with published practices,[16] samples <LOQ were imputed as the LOQ divided by √2.

Sociodemographic variables were compared in the final analysis dataset versus the overall dataset. Spearman correlations between log-transformed PFAS variables were explored using a correlation matrix. PFAS levels in their original scale were compared across sociodemographic variable subgroups using Kruskal-Wallis tests for subgroups with greater than two categories [race (Black, White, Asian, Native American/other); maternal pre-pregnancy BMI (underweight: BMI<18.5 kg/m^2, normal: BMI≥18.5 & <25 kg/m^2, overweight/obese: BMI≥25 kg/m^2); education (< high school degree, high school degree, > high school degree)], and using Mann-Whitney U tests for subgroups with two categories [maternal age (<30, ≥30); child sex; trimester on 9/11 (≤91 days, >91 days); home smoking exposure (no reported family member smoking in household, any reported family member smoking in household); marital status (not married, married); and parity (primiparous, multiparous)]. Results were displayed using forest plots.

Linear regression models were used to evaluate the association between WTC exposure variables and log-transformed PFAS concentrations. The first exposure variable (“Home or Work”) categorized exposed participants (n=120) as those that either lived or worked within 2 miles of the WTC site. Participants that did not live or work within 2 miles of the disaster were categorized as the reference group (n=159). The second exposure variable (“Home versus Work”) attempted to better understand the contribution of home versus work exposures in PFAS levels. This exposure variable partitioned the exposed group in the “Home or Work” variable into 2 subgroups: 1) those that lived within 2 miles of the WTC site regardless of where they worked (n=75); and 2) those that worked but did not live within 2 miles of the WTC site (n=45). The unexposed group remained the same as the previous

exposure variable, and included those that did not live or work within 2 miles of the site (n=159). Models report log-unit changes in PFAS concentrations by exposure category; results are exponentiated and therefore reflect geometric mean ratios (GMRs). Model 1 was unadjusted. Model 2 was adjusted for sociodemographic variables previously associated with PFAS exposure including: child sex; maternal age, education, BMI, marital status and parity; trimester pregnant during the 9/11 disaster and home smoking exposure. Model 3 further adjusted for maternal race.

To confirm findings, several sensitivity analyses were conducted. First, we ran analyses using just PFAS concentrations measured in cord blood (n=231) to evaluate consistency with analyses using maternal-to-cord transformed concentrations. Second, to check whether using a complete case analysis biased our analyses, we also ran analyses filling in missing values in our covariates using multiple imputation using Fully Conditional Specification (FCS) implemented by the MICE (multivariate imputation by chained equations) algorithm. Analyses were conducted using the "MICE" r package with the number of imputed datasets set to 10. Finally, among a subset of participants (n=165) with information on local fish intake, we ran analyses additionally adjusting for this variable to better understand whether other sites of PFAS contamination (e.g,, contaminated waterways, and in turn contaminated local fish) might confound associations.

RESULTS

Participant Characteristics

Cord blood PFAS geometric means were generally lower than maternal plasma concentrations (Table 1). Our maternal-to-cord transformed concentrations plus cord blood concentrations together were slightly lower than cord blood concentrations alone. Geometric mean (range) concentrations of PFAS variables (cord blood plus maternal-to-cord transformed) were 6.03 (1.05, 33.7)ng/ml for PFOS, 2.31 (0.18, 8.14)ng/ml for PFOA, 0.43 (<LOQ, 10.3)ng/ml for PFNA and 0.67 (<LOQ, 15.8) ng/ml for PFHxS (Table 1). All PFAS variables were significantly correlated (p = <0.01) with each other. Correlations ranged from 0.17 between PFOA and PFNA and 0.70 between PFNA and PFOS (Figure 2). Median (interquartile range (IQR)) age of the study population was 31.0 (27.3-34.6) years (Table 2). Roughly half of newborns were female (52.3%); 43.7% of the population was White, 36.2% were Asian, 16.1% were Black and 3.9% were Native American/other. The majority of participants had a normal BMI (72%), with 18.6% overweight/obese and just 9.3% underweight. Slightly more participants were delivering their first child (56.6%) than their second or more. Most of the women were married (82.4%), reported no family smoking during pregnancy (81.7%) and were in their first trimester (68.5%) on 9/11. The majority of participants reported more than a high school degree (63.8%), with roughly the same proportion reporting just a high school degree (17.2%) or less than a high school degree (19.0%). Participants in the overall study population (n=329) versus the analysis population (n=279) were similar across most study variables; however, participants in the analysis population had slightly higher PFNA concentrations (0.37 versus 0.36), were slightly older (31.0 versus 30.3 years) and were more likely to be in their second/third trimester (31.5%

versus 29.2%) on 9/11 compared to those participants in the overall study population (Table 2).

Comparing PFAS concentrations across sociodemographic subgroups revealed some differences (Figure 3). Older age was associated with higher PFHxS concentrations and female newborns had higher PFOA concentrations. PFNA concentrations were higher in women exposed to the WTC disaster during their first trimester compared with women who were later on in their pregnancy on 9/11. A post hoc sensitivity analysis conducted to evaluate PFNA concentrations in the 18 women who were not yet pregnant on 9/11 revealed that PFNA concentrations were highest in this subgroup (data not shown). A higher pre-pregnancy BMI was associated with lower PFOS and PFNA concentrations. Higher education was also associated with lower PFOS and PFNA, but with higher PFHxS. Race was associated with all four PFAS concentrations. Asian race was associated with higher PFOS and PFNA, but lower PFOA concentrations. Black race was associated with lower concentrations for all four PFAS variables. White race was associated with lower PFOS and PFNA but higher PFOA and PFHxS. Native American/other was associated with higher PFOA and PFHxS. Home smoking exposure during pregnancy, marital status and parity were not significantly associated with any PFAS concentrations.

Associations between WTC Exposure and PFAS concentrations

In fully adjusted models (Table 3), living or working within two miles of the WTC site in the four weeks following the event, was associated with 13% higher PFOA concentrations compared with those not living or working within two miles of the site (reference group) [GMR (95% CI): 1.13 (1.01, 1.27)]. Further, living within two miles of the WTC site regardless of work location, was associated with a 17% higher PFOA concentration compared with the reference group [GMR (95% CI): 1.17 (1.03, 1.33)]. Working but not living within two miles of the site was associated with a non-significant increase in PFOA concentrations [GMR (95% CI): 1.07 (0.92, 1.25)]. There were no other significant associations between WTC exposure group and PFAS concentration; however, there was a general trend of increases in the other three PFAS (PFOS, PFHxS and PFNA) concentrations for those living within two miles of the WTC site compared with those not living or working within two miles of the site. Of note, this trend was not apparent when those who worked but did not live within two miles of the WTC site were compared to the reference group.

Findings from **s**ensitivity analyses conducted in just cord blood samples (Supplemental Table 2), among participants with local fish intake data available additionally adjusted for that variable (data not shown) and using multiple imputation to fill in missing values in covariates (data not shown) were consistent with main analyses.

DISCUSSION

In this study of women who delivered children in hospitals near the WTC disaster, who were pregnant during or within the weeks following the 9/11/2001 event, we observed significant increases in PFOA concentrations among participants who worked or lived within two miles of the WTC site compared with those who did not. Further, we observed stronger associations for those who lived within two miles of the site, regardless of where they

worked, compared to those who worked but did not live within two miles of the site. These findings add to the minimal existing research available on trends in cord blood PFAS concentrations across sociodemographic variables in the US. In addition, our results provide evidence that exposure to the WTC disaster during pregnancy resulted in increases in cord blood PFAS concentrations, specifically PFOA. This work identifies a potentially vulnerable and overlooked population, children exposed to the WTC in utero, who should be monitored for later life effects resulting from these early life exposures.

Our study's finding of detectable PFOA and PFOS concentrations in all cord blood samples and in the majority of samples for PFNA and PFHxS, confirm previous evidence[9, 17-21] that PFAS can cross the placental barrier, potentially harming the developing fetus. Indeed, in addition to substantial experimental evidence on the developmental toxicity of PFOS,[22, 23] numerous epidemiological studies have reported associations between prenatal PFAS exposure and adverse health outcomes, including reductions in birth weight[8, 23, 24], birth length[25], abdominal circumference[25], ponderal index[24] and head circumference[24]; immune effects[26-31]; and hormonal[21, 32, 33], behavioral[34, 35] and neurological[36-38] outcomes.

In response to their toxicity as well as their ubiquity and persistence in the environment and in humans, substantial efforts have been made to reduce their production.[4] Still, while an analysis of exposure trends using NHANES data from 1999-2008 showed marked reductions in PFOS over time, decreases in PFOA and PFHxS were minimal, and increases were seen for PFNA.[39] Food and drinking water ingestion, followed by dust inhalation, have been identified as main pathways of exposure for PFOA and PFOS, but less is known regarding other PFAS including PFNA and PFHxS.[40] Further, patterns and other determinants of exposure are incompletely understood, particularly for cord blood. The limited research and the unique sensitivity of the gestational period to chemical exposures, highlights the importance of additional studies on this topic. Our finding of higher PFOS in Asian participants compared to Black, White or other, is consistent with the only other study to evaluate trends in cord blood PFAS in the US.[9] We also found higher PFNA cord blood in Asian participants compared to other races, which is a novel finding for cord blood but consistent with analyses in pregnant women.[41] The observed higher concentrations of PFNA and PFOS in Asian participants versus other races is also consistent with national trends for these PFAS in adult serum samples.[42] Our results of lower PFOA and PFHxS in black versus white participants are consistent with studies evaluating trends by race in pregnant women[10, 41] and children[43] in the US. In addition, our findings of lower PFOS and PFNA with higher BMI confirm a previous US-based study in pregnant women.[10] Trends in PFAS concentrations across education subgroups varied by compound in our study: we report lower PFNA and PFOS but higher PFHxS with higher education, in agreement with some[10, 41, 43, 44] but not all[43] studies. In regard to WTC-related exposure, our finding of higher PFNA in women who were exposed to the WTC disaster earlier in their pregnancy may reflect the longer duration of exposure following 9/11 before delivery, compared with women who were further along in their pregnancy during the disaster. This is supported by the finding that women who were not yet pregnant on 9/11 had the highest concentrations of PFNA. Still it's unclear why this finding would be evident for PFNA and not the other PFAS. Our finding of higher PFOA in female cord blood is consistent with a previous US-based cord blood PFAS analysis.[9] The mechanism behind the higher levels seen in female

cord blood is unclear. Further, in nationally representative analyses in adults, the inverse of this trend has been consistently reported.[39, 42] Finally, our finding of higher PFHxS with higher maternal age is consistent with previous studies.[9, 10, 43, 45] It is interesting to note the apparent similarities in trends across education, BMI and race between PFOS and PFNA versus PFOA and PFHxS in our population. More research is needed to determine whether these data suggest a shared source between the similar PFAS that is driving these trends.

In addition to differences in PFAS concentrations across sociodemographic variables, we observed a significant association between living or working within 2 miles of the WTC site and higher PFOA concentrations. This finding contrasts with two previous studies evaluating other chemical exposures in WTC-exposed pregnant women: WTC exposure during pregnancy was not associated with significant differences in blood mercury[46] or polybrominated diphenyl ethers (PBDEs).[47] However, it is consistent with two recent studies that found elevated concentrations of PFAS[3] and dioxin[48] in adolescents exposed to the WTC event as children; as well as a study that found higher polycyclic aromatic hydrocarbons-DNA adducts in mothers and newborns residing within one mile of the WTC disaster.[49] It is not completely clear why we observed an association with PFOA and not the other PFAS measured. Studies have suggested that PFOA crosses the placental barrier more easily than PFOS, resulting in higher transplacental transfer efficiency.[17, 50, 51] In turn, higher transfer efficiency has been indicated as an explanation for significant associations between PFOA and adverse health outcomes in the absence of significant findings for other PFAS.[50] It is possible a high transplacental transfer efficiency for PFOA, resulting in cord blood levels more influenced by maternal exposures, might explain the association we see between WTC exposure and cord blood PFOA versus the other PFAS evaluated in this study. We were unable to evaluate transplacental transfer efficiency for the PFAS in our population because we did not have access to paired maternal-cord samples.

PFAS was not measured in WTC dust,[1] however, it is also possible dust and smoke from the WTC collapse and subsequent fires resulted in greater relative increases in PFOA than other PFAS due to differences in exposure sources related to the disaster across PFAS compounds. For example, PFAS are the active ingredient in aqueous film-forming foam (i.e., fire-fighting foam), which would likely have been used to help extinguish fires resulting from the WTC disaster. It's difficult to know which PFAS may have been more concentrated in the foam used during the WTC collapse due to the proprietary nature of fire-fighting foam mixtures, as well as the changes in formulations by year of production and manufacturer[52]; however, it's possible PFOA was the dominant PFAS. This may also be true of other PFAS exposure sources specific to the WTC collapse. Dust samples from office buildings have been reported to have higher PFAS concentrations than residences and vehicles[53]; further, PFAS precursors in office air samples have been associated with PFOA serum concentrations, but not PFOS, in office workers.[54] A study evaluating serum PFAS in WTC first responders reported 2-fold higher PFOA and PFHxS concentrations, yet lower PFOS, than the general US population, suggesting higher WTC-related PFOA and PFHxS exposures compared to PFOS.[2] Still, a recent study conducted in WTC-exposed children, reported significantly higher concentrations of all four PFAS compared to a matched unexposed control group.[3] Just two other studies have evaluated cord blood PFAS in US populations: cord blood PFOA in our population (median = 2.38 ng/mL) was similar (including the "exposed" population)

to concentrations in both Baltimore, MD (geometric mean = 1.6 ng/mL)[9] and Cincinnati, Ohio (median = 3.1 ng/mL)[10] birth cohorts. Despite comparable overall PFOA levels to other studied cities, the significant increases in PFOA concentrations observed with WTC exposure still warrant attention. Indeed, increases in PFOA at levels in the range of our cohort was associated with significant reductions in fetal growth, providing evidence that even minor increases in prenatal exposure may cause harm.[24]

Our findings also highlight the often overlooked importance of indoor exposures as a source of exposure in the event of both manmade and natural environmental disasters. Further, the stronger association that we found with home exposure versus just work exposure, emphasizes the significance, in particular, of home exposures to chemical body burdens in the event of an environmental disaster. This is supported by other WTC studies, which have found higher PFAS in children exposed to WTC-related home dust exposure versus dust cloud exposure,[3] as well as higher dioxin levels in children exposed to WTC house dust even after adjustment for dust cloud exposure.[48]

This study has limitations that should be considered when interpreting findings. Given the long half-life of PFAS and the single measurement of PFAS concentrations used in this study, we cannot rule out another source of exposure that could explain the observed differences reported for PFOA or could have masked differences we did not observe for the other PFAS. However, contaminated drinking water, a significant potential source of PFAS, would not be an exposure source for this population as NYC drinking water is drawn from upstate watersheds, isolated from the WTC disaster. Further, a sensitivity analysis adjusting for local fish intake to account for potential confounding by this exposure source, yielded consistent results with main analyses. Finally, our unexposed comparison group resided in New York City and may have been exposed to WTC dust to some degree; this may have attenuated differences in PFAS concentrations between comparison groups and contributed to the null findings we observed for other PFAS

CONCLUSIONS

Sociodemographic trends in PFAS vary by compound, with certain similarities in trends for PFOA and PFHxS and for PFOS and PFNA, potentially suggesting shared exposure sources that should be researched further. In addition, living or working within 2 miles of the WTC site was associated with increases in PFOA cord blood among women who were pregnant at the time of, or within the weeks following, the WTC disaster. These results identify a vulnerable population that should be monitored for the development of later-life health effects arising from these early life exposures; highlighting the importance of future longitudinal analyses in this cohort. Home dust exposures, specifically, appear to be a driving factor in elevating internal levels of PFAS, and potentially other chemicals, and should be an important focus of cleanup in the event of other environmental disasters.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

Funding: This work was supported by grants 1U01-OH010714-01A1, 1U01-OH010394-01A1, 1U01-OH011299-01A1 from NIOSH; the September 11th Fund of the New York Community Trust and United Way of New York City; the New York Times 9/11 Neediest Fund; the National Philanthropic Trust; and the National Institute of Environmental Health Sciences grant ES09089.

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HIGHLIGHTS

- Perfluoroalkyl substances (PFAS) may have been released during the 9/11 disaster
- PFAS can cross the placenta potentially harming the fetus
- Proximity to the 9/11 site was associated with increases in a cord blood PFAS
- Longterm health effects in children exposed to 9/11 in utero should be evaluated

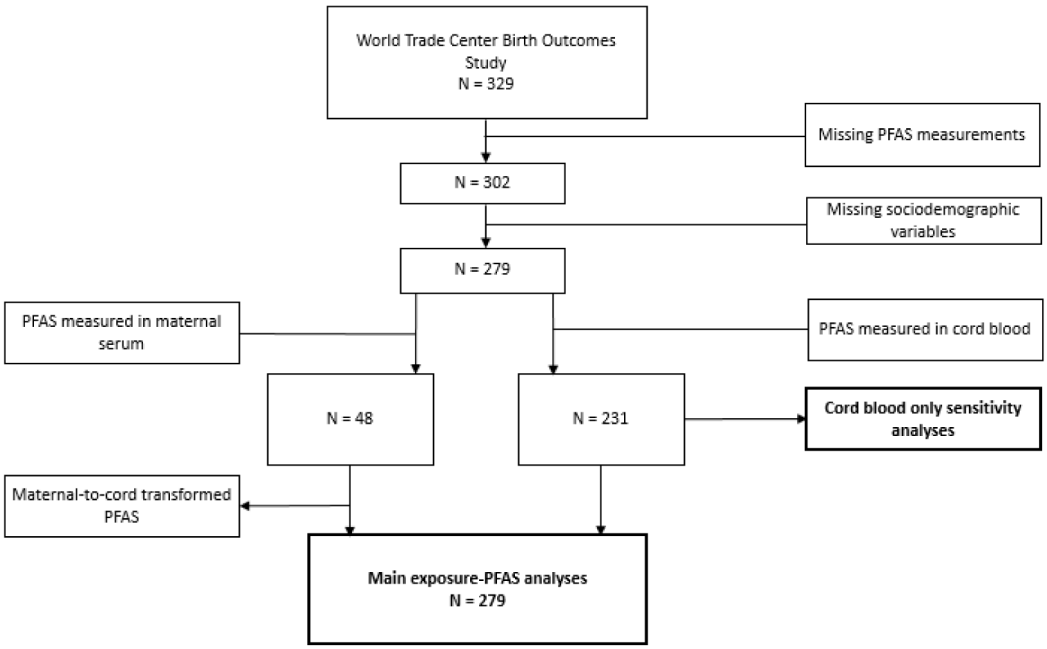

Figure 1. Study Flow Diagram.
Abbreviations: Perfluroalkyl substances (PFAS)

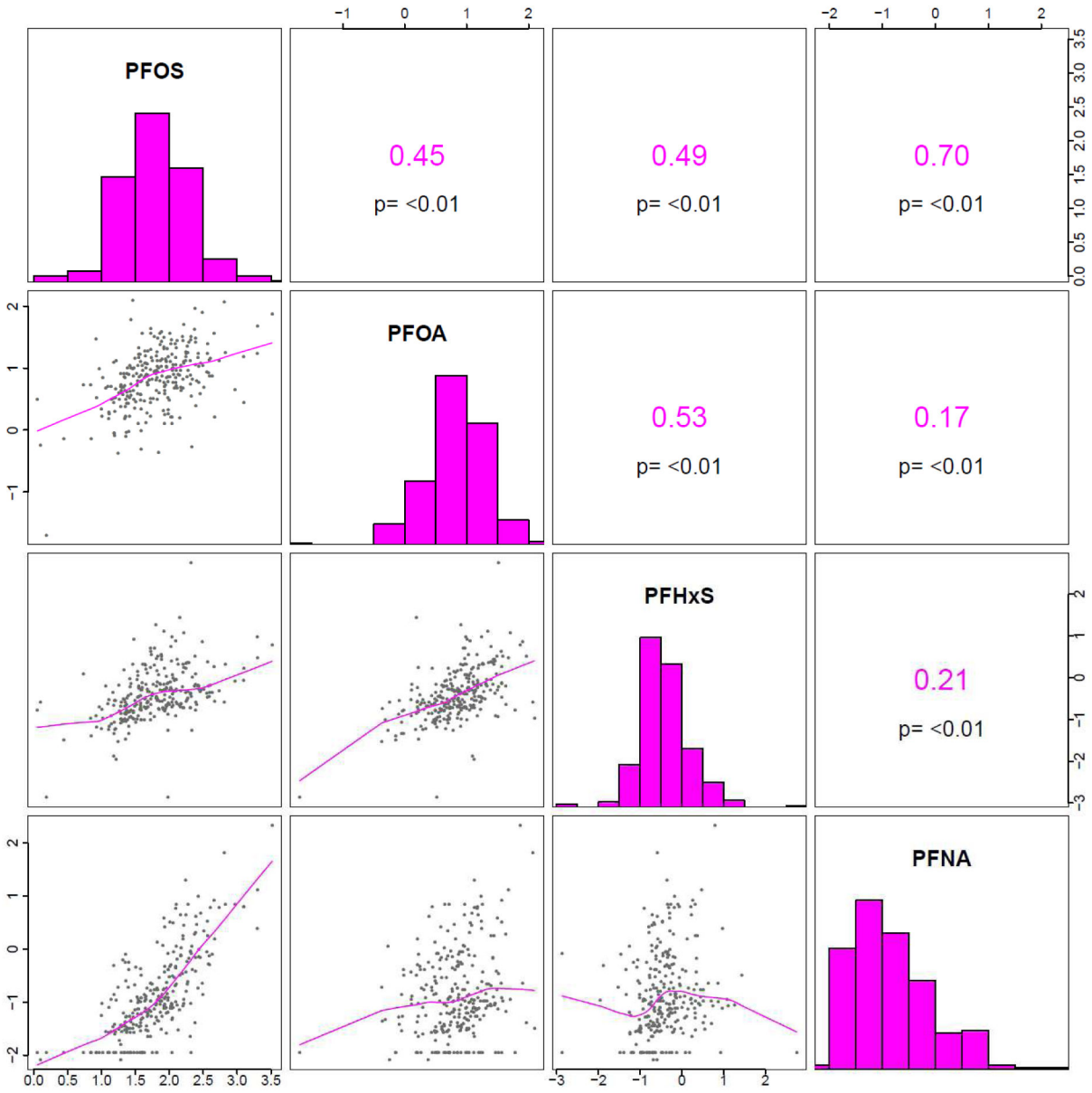

Figure 2. Correlation Matrix of Perfluroalkyl Substances.

Histograms of, and spearman correlations between, log-transformed perfluroalkyl substances. Abbreviations: perfluorohexanesulfonic acid (PFHxS), perfluorononanoic acid (PFNA), perfluorooctane sulfonate (PFOS), perfluorooctanoic acid (PFOA)

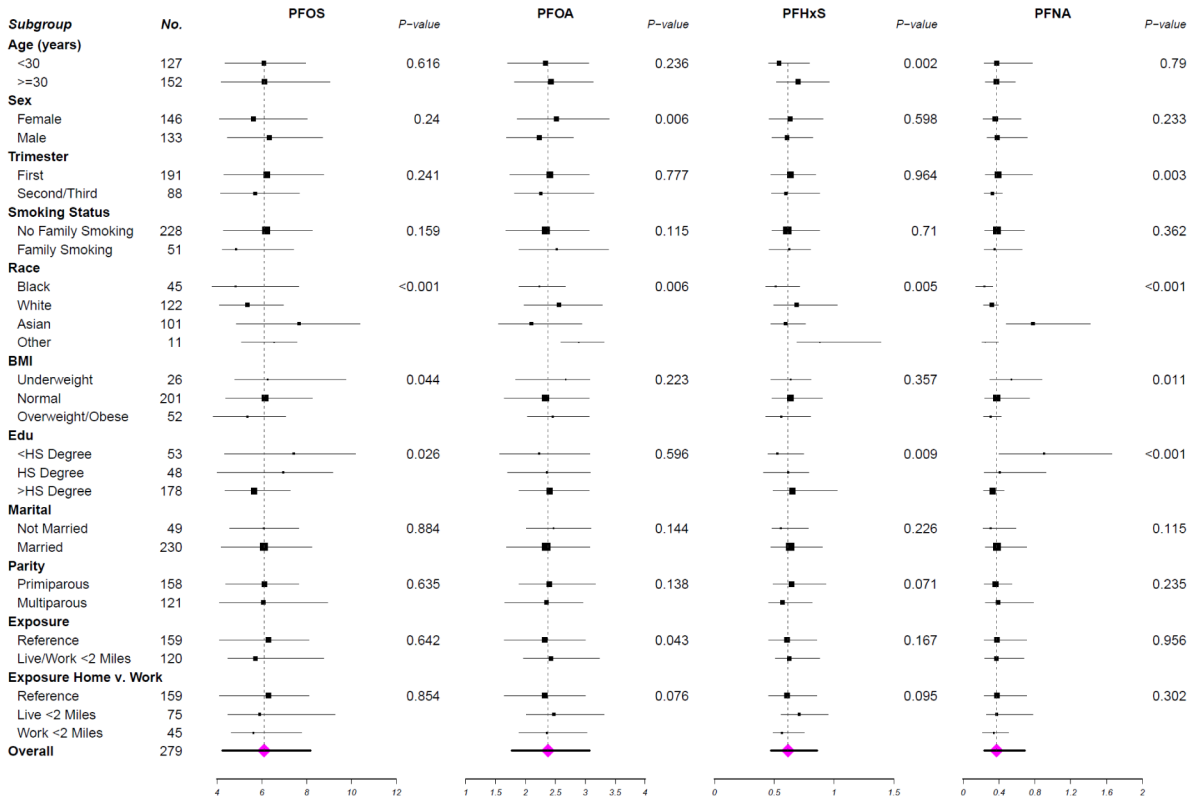

Figure 3. Population Characteristics by Perfluroalkyl Substance Concentrations.

Squares represent the median levels of metabolites and lines represent the interquartile range in each subcategory. P-values calculated from Mann-Whitney U Tests for subgroups with two categories and Kruskal-Wallis Tests for subgroups with three categories. Abbreviations: Body Mass Index (BMI), High School (HS), perfluorohexanesulfonic acid (PFHxS), perfluorononanoic acid (PFNA), perfluorooctane sulfonate (PFOS), perfluorooctanoic acid (PFOA)

Table 1.

Geometric Mean Concentrations (ng/mL) and Percent Above Detection of Perfluoroalkyl Substances (PFAS) in Cord Blood, Maternal Serum and Cord + Transformed Maternal Serum

PFAS	N	LOQ	% Above LOQ	Geometric Mean (Range)[a]
PFOS				
Cord Blood	231	0.20	100%	6.27 (1.05, 33.7)
Maternal Serum	48	0.20	100%	11.9 (2.90, 30.9)
Cord Blood + Transformed Maternal Serum	279	0.20	100%	6.03 (1.05, 33.7)
PFOA				
Cord Blood	231	0.08	100%	2.37 (0.18, 8.14)
Maternal Serum	48	0.08	100%	2.42 (0.88, 5.06)
Cord Blood + Transformed Maternal Serum	279	0.08	100%	2.31 (0.18, 8.14)
PFNA				
Cord Blood	231	0.20	86%	0.45 (<LOQ, 10.3)
Maternal Serum	48	0.20	96%	0.45 (<LOQ, 1.93)
Cord Blood + Transformed Maternal Serum	279	0.20	88%	0.43 (<LOQ, 10.3)
PFHxS				
Cord Blood	231	0.08	99%	0.69 (<LOQ, 15.8)
Maternal Serum	48	0.08	100%	0.94 (0.35, 3.20)
Cord Blood + Transformed Maternal Serum	279	0.08	99%	0.67 (<LOQ, 15.8)
PFDS				
Cord Blood	231	0.08	97%	0.13 (<LOQ, 0.64)
Maternal Serum	48	0.08	98%	0.16 (<LOQ, 0.82)
PFOSA				
Cord Blood	231	0.08	0%	<LOQ
Maternal Serum	48	0.08	0%	<LOQ
PFBS				
Cord Blood	231	0.08	<1%	<LOQ (<LOQ, 0.28)
Maternal Serum	48	0.08	0%	<LOQ
PFHxA				
Cord Blood	231	0.08	15%	<LOQ (<LOQ, 10.8)
Maternal Serum	48	0.08	19%	<LOQ (<LOQ, 6.01)
PFHpA				
Cord Blood	231	0.08	36%	<LOQ (<LOQ, 0.59)
Maternal Serum	48	0.08	23%	<LOQ (<LOQ, 0.23)
PFDA				
Cord Blood	231	0.08	45%	<LOQ (<LOQ, 1.69)
Maternal Serum	48	0.08	75%	0.13 (<LOQ, 0.75)
PFUnDA				
Cord Blood	231	0.20	20%	<LOQ (<LOQ, 3.27)
Maternal Serum	48	0.20	13%	<LOQ (<LOQ, 0.87)
PFDoDA				

PFAS	N	LOQ	% Above LOQ	Geometric Mean (Range)[a]
Cord Blood	231	0.20	7%	<LOQ (<LOQ, 0.63)
Maternal Serum	48	0.20	0%	<LOQ

Abbreviations: Limit of Quantification (LOQ); Interquartile Range (IQR); perfluorobutane sulfonate (PFBS); perfluorodecane sulphonate (PFDS); perfluorodecanoate (PFDA); perfluorododecanoic acid (PFDoDA); Perfluoroheptanoic acid (PFHpA); perfluorohexanesulfonic acid (PFHxS); Perfluorohexanoic acid (PFHxA); perfluorononanoic acid (PFNA); Perfluorooctanesulfonamide (PFOSA); perfluorooctane sulfonate (PFOS); perfluorooctanoic acid (PFOA); perfluoroundecanoic acid (PFUnDA)

[a]Geometric mean is listed as <LOQ if >50% of observations are <LOD.

Table 2.

Participant Characteristics in Full versus Final Study Dataset

Variable	Full	Final Dataset	P-value
N (%)	329 (100)[a]	279 (84.8)	
PFOS ng/ml, median (IQR)	6.07 (4.18-8.1)	6.10 (4.24-8.18)	0.112
PFOA ng/ml, median (IQR)	2.39 (1.77-3.07)	2.38 (1.77-3.07)	0.715
PFNA ng/ml, median (IQR)	0.36 (0.24-0.67)	0.37 (0.24-0.69)	0.032
PFHxS ng/ml, median (IQR)	0.61 (0.46-0.85)	0.62 (0.47-0.86)	0.084
Maternal Age	30.3 (26.7-34.5)	31.0 (27.3-34.6)	0.001
Gender, n (%)			
Female	168 (51.1)	146 (52.3)	0.352
Male	161 (48.9)	133 (47.7)	
Race, n (%)			
Black	50 (16.2)	45 (16.1)	0.933
White	133 (43.2)	122 (43.7)	
Asian	113 (36.7)	101 (36.2)	
Other	12 (3.9)	11 (3.9)	
BMI, n (%)			
Underweight	31 (9.5)	26 (9.3)	0.489
Normal	231 (70.9)	201 (72.0)	
Overweight/Obese	64 (19.6)	52 (18.6)	
Parity, n (%)			
Primiparous	188 (57.3)	158 (56.6)	0.658
Multiparous	140 (42.7)	121 (43.4)	
Marital Status, n (%)			
Single	64 (19.5)	49 (17.6)	0.064
Married	265 (80.5)	230 (82.4)	
Trimester on 9/11, n (%)			
≤91 GA in days	233 (70.8)	191 (68.5)	0.04
>91 GA in days	96 (29.2)	88 (31.5)	
Education, n (%)			
< High School Degree	61 (18.5)	53 (19.0)	0.836
High School Degree	56 (17.0)	48 (17.2)	
> High School Degree	212 (64.4)	178 (63.8)	
Smoking Exposure, n (%)			
No Family Smoking	270 (82.1)	228 (81.7)	0.852
Any Family Smoking	59 (17.9)	51 (18.3)	

[a]Sample size in full dataset varies by some characteristics (PFASs variables: n=302; Race: n=308; BMI: n=326; Parity: n=328) Abbreviations: body mass index (BMI), gestational age (GA), perfluorohexanesulfonic acid (PFHxS), perfluorononanoic acid (PFNA), perfluorooctane sulfonate (PFOS), perfluorooctanoic acid (PFOA)

Table 3.

Geometric Mean Ratios of Perfluroalkyl Substances World Trade Center Exposure Categories

PFAS	Model 1	Model 2	Model 3
	PFOA		
Reference (n=159)	1	1	1
Live 2 Miles (n=75)	1.18 (1.04, 1.35)	1.17 (1.03, 1.33)	1.17 (1.03, 1.33)
Work 2 Miles (n=45)	1.09 (0.93, 1.27)	1.05 (0.89, 1.22)	1.07 (0.92, 1.25)
Reference (n=159)	1	1	1
Live or Work < 2 Miles (n=120)	1.15 (1.03, 1.28)	1.12 (1.00, 1.25)	1.13 (1.01, 1.27)
	PFOS		
Reference (n=159)	1	1	1
Live 2 Miles (n=75)	1.06 (0.92, 1.22)	1.05 (0.92, 1.21)	1.02 (0.89, 1.18)
Work 2 Miles (n=45)	1.00 (0.85, 1.19)	1.00 (0.85, 1.19)	1.00 (0.84, 1.18)
Reference (n=159)	1	1	1
Live or Work < 2 Miles (n=120)	1.04 (0.92, 1.17)	1.03 (0.92, 1.17)	1.01 (0.90, 1.14)
	PFHxS		
Reference (n=159)	1	1	1
Live 2 Miles (n=75)	1.16 (0.98, 1.37)	1.12 (0.95, 1.33)	1.12 (0.95, 1.32)
Work 2 Miles (n=45)	1.00 (0.82, 1.22)	0.93 (0.75, 1.14)	0.95 (0.78, 1.17)
Reference (n=159)	1	1	1
Live or Work < 2 Miles (n=120)	1.09 (0.95, 1.27)	1.05 (0.90, 1.21)	1.06 (0.91, 1.22)
	PFNA		
Reference (n=159)	1	1	1
Live 2 Miles (n=75)	1.13 (0.91, 1.41)	1.13 (0.93, 1.38)	1.06 (0.89, 1.26)
Work 2 Miles (n=45)	0.83 (0.64, 1.08)	0.93 (0.73, 1.18)	0.89 (0.72, 1.09)
Reference (n=159)	1	1	1
Live or Work < 2 Miles (n=120)	1.01 (0.83, 1.22)	1.05 (0.88, 1.25)	0.99 (0.85, 1.15)

Model 1: Unadjusted

Model 2: Adjusted for Maternal Age, Child Gender, Trimester on 9/11, Maternal Pre-pregnancy BMI, Maternal Edu, Parity, Marital Status, and Family Smoking Status

Model 3: Model 2 adjustments + Race

Abbreviations: perfluorohexanesulfonic acid (PFHxS), perfluorononanoic acid (PFNA), perfluorooctane sulfonate (PFOS), perfluorooctanoic acid (PFOA)