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EXPOSURE OF NORWEGIAN TODDLERS TO PERFLUOROALKYL SUBSTANCES (PFAS): THE ASSOCIATION WITH BREASTFEEDING AND MATERNAL PFAS CONCENTRATIONS

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Introduction

Perfluoroalkyl substances (PFASs) are synthetic fluorinated organic compounds used in industrial and consumer products over the last 50 years due to their chemical and thermal stability and water and oil repellency. Adults are exposed to PFASs through ambient indoor air, house dust and drinking water, though the main route is through food (Fromme et al., 2009; Vestergren and Cousins 2009). During pregnancy, maternal PFASs are transferred through the placenta resulting in prenatal exposure of the fetus (Gutzkow et al., 2012). The infant is born with approximately 30-50% of the mother's blood concentrations for PFOS and 60-80% of the mother's PFOA concentrations (Beesoon et al., 2011; Gutzkow et al., 2012;). After birth, the breastfed infant is continuously exposed to PFASs through consumption of breast milk (Kim et al., 2011). Even though the PFAS concentrations in breast milk are lower than in maternal serum, 6 months of breastfeeding can substantially increase the PFAS body burden of the infant. It has been shown that 90% of infant's PFAS exposure can be attributed to breastfeeding (Haug et al., 2011).

Maternal PFAS concentrations are positively correlated with PFAS concentrations in cord blood, newborn blood and breast milk and this has been well documented for the most abundant congeners. However, the relationship between maternal PFASs and PFAS concentrations in young children is scarcely studied (Mondal et al., 2012; Mondal et al., 2014). Infants (0-1 year) and toddlers (1-3 years) have been exposed to PFASs both in-utero and postnatally through breastfeeding, while other postnatal exposure patterns, through diet, indoor air and dust, might also occur for toddlers. Given the emerging evidence of adverse health effects related to high PFASs exposure of young children, it is important to report the PFAS concentrations in toddlers and investigate the determinants of PFASs exposure in this age group.

The aims of this study are to examine the PFAS blood concentrations in Norwegian toddlers and to assess the relationship with maternal PFAS levels in pregnancy and breastfeeding duration.

Materials and methods

The Norwegian birth cohort BraMat is a sub-cohort of the Norwegian Mother and Child Cohort Study (MoBa). MoBa is a prospective population-based pregnancy cohort study conducted by the Norwegian Institute of Public Health (Magnus et al., 2006; Ronningen et al., 2006). Participants were recruited from all over Norway from 1999-2008. The women consented to participation in 40.6% of the pregnancies. The cohort now includes 114.500 children, 95.200 mothers and 75.200 fathers. Eligible participants in the BraMat sub-cohort were women with singleton pregnancies, already recruited in the MoBa study and who were scheduled to give birth at the Oslo University Hospital Ullevål or the Akershus University Hospital, in Oslo area, Norway. The recruitment took place between April 2007 and March 2008 and women were contacted at the 37th week of pregnancy, thus only full-term pregnancies were included (37th – 42nd week of pregnancy). The recruitment rate in BraMat was 25% (n=205). The BraMat study was approved by the Norwegian Regional Committee for Medical and Health Research Ethics and the Data Inspectorate.

Blood samples were collected from mothers around delivery (0-3 days after birth) and 112 (56%) children at the age of three years. This study includes 99 mothers and 112 3-year old children (mean age: 34.4 months, SD: 2.5), of whom 55 are mother-child pairs. Concentrations of nineteen PFASs were investigated in maternal plasma and children's serum samples, using high-performance tandem mass spectrometry at the Norwegian Institute of Public Health by a previously described method (Haug et al., 2009). Statistical analyses were limited to six PFASs that were quantifiable in more than 50% of both maternal and children samples. The compounds finally included in our study were: PFHxS, PFHpS, PFOS, PFOA, PFNA, and PFUnDA. The limit of quantification (LOQ) was 0.05 ng/mL for all measured PFASs. Values below LOQ were replaced with LOQ divided by the square root of two

for further statistical analysis. All PFAS distributions failed the Shapiro-Wilk test of normality, thus the logarithm (base 10) of the values was used in subsequent analyses.

Information on maternal and pregnancy related characteristics that have been identified as determinants of PFAS blood concentrations in mothers, including maternal age and education, pre-pregnancy body mass index (BMI), parity, history of breastfeeding in previous pregnancies and type of delivery were obtained through questionnaires administered at 15th and 30th week of gestation and the Medical Birth Registry of Norway. Child's gender, gestational age and birth weight were also included as potential predictors of PFAS blood concentrations in children (Mondal et al., 2012; Mondal et al., 2014). Information on duration of breastfeeding and attendance to day-care center were obtained by questionnaires administered at the follow-up of the BraMat study at 3-years of age, as proxies of postnatal exposure pathways.

Results and discussion

The 3-years old children had higher concentrations of PFASs in blood than pregnant women, particularly due to higher PFOA, PFNA and PFHxS concentrations (Figure 1). These differences were confirmed when comparing mother-child pairs.

Multiple linear regression analysis showed that every month of breastfeeding was associated with 3% to 5% increase in PFOS, PFOA, PFHxS and PFHpS in children independently of maternal prenatal PFAS concentrations.

Median PFAS child:mother ratios followed this increasing sequence: PFOS< PFUnDA< PFHpS< PFHxS< PFOA< PFNA (range: 0.9-3.6). Moderate positive correlations between maternal and children concentrations of PFHxS, PFHpS, PFOS and PFOA were found (range: 0.50-0.66). No correlations between maternal and child concentrations of PFNA and PFUnDA were observed, and the levels of the mother did not contribute to the observed variation of PFNA and PFUnDA in children, as showed by regression analysis. Among mother-child pairs, all children had higher PFNA concentrations than their mothers and 24% of the children had PFNA concentrations more than 10 times higher than their mothers.

Our findings suggest that transplacental transfer, prenatally, and breastfeeding, postanatally, are among the main determinants of PFOS, PFOA, PFHxS and PFHpS concentrations in toddlers.The absence of association between prenatal levels of PFNA and PFUnDA and breastfeeding duration with PFNA and PFUnDA in toddlers suggests a different postnatal source of exposure.

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Figure 1. Geometric means of PFAS concentrations of 99 maternal and 112 children samples.