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Polyfluorinated compounds in dust from homes, offices, and vehicles as predictors of concentrations in office workers' serum

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ABSTRACT

We aimed to characterize levels of polyfluorinated compounds (PFCs) in indoor dust from offices, homes, and vehicles; to investigate factors that may affect PFC levels in dust; and to examine the associations between PFCs in dust and office workers' serum. Dust samples were collected in 2009 from offices, homes, and vehicles of 31 individuals in Boston, MA and analyzed for nineteen PFCs, including perfluorooctanoate (PFOA), perfluorooctane sulfonate (PFOS), fluorotelomer alcohols (FTOHs), and sulfonamidoethanols (FOSEs). Serum was collected from each participant and analyzed for eight PFCs including PFOA and PFOS. Perfluorononanoate, PFOA, perfluoroheptanoate, perfluorohexanoate, PFOS and 8:2 FTOH had detection frequencies >50% in dust from all three microenvironments. The highest geometric mean concentration in office dust was for 8:2 FTOH (309 ng/g), while PFOS was highest in homes (26.9 ng/g) and vehicles (15.8 ng/g). Overall, offices had the highest PFC concentrations, particularly for longer-chain carboxylic acids and FTOHs. Perfluorobutyrate was prevalent in homes and vehicles, but not offices. PFOA serum concentrations were not associated with PFC dust levels after adjusting for PFC concentrations in office air. Dust concentrations of most PFCs are higher in offices than in homes and vehicles. However, indoor dust may not be a significant source of exposure to PFCs for office workers. This finding suggests that our previously published observation of an association between FTOH concentrations in office air and PFOA concentrations in office workers was not due to confounding by PFCs in dust.

1. Introduction

Polyfluorinated compounds (PFCs) are used in a variety of commercial applications due to desirable properties such as water and oil repellency, thermal stability, and resistance to biotic, chemical or mechanical degradation. Since the 1940s, PFCs have been used in applications such as fire-fighting foams and pesticides, in the production of protective sprays and coatings for fabrics, carpets, and clothing, and more recently in food-contact paper and non-stick cookware (Kissa, 2001; Prevedouros et al., 2006). In fact, their widespread use is such that PFCs have been detected in wildlife, humans, water, air and soil (Barber et al., 2007; Kato et al., 2011; Lau et al., 2007; Prevedouros et al., 2006; Rumsby et al., 2009; Shoeib et al., 2006; Strynar et al., 2012). Low-level body burdens of some PFCs, such as perfluorooctanoate (PFOA) and perfluorooctane sulfonate (PFOS), are ubiquitous in the general human population and raise concern about the potential toxicity of these persistent organic pollutants (Kato et al., 2011; Lau et al., 2007).

Animal studies have identified PFOA and PFOS to be potent peroxisome proliferators that are associated with liver toxicity, developmental delays, immune system effects and endocrine disruption (DeWitt et al., 2009; Jensen and Leffers, 2008; Lau et al., 2007). Emerging epidemiologic research suggests that PFOA and PFOS may be associated with lowered birth weight (Apelberg et al., 2007; Fei et al., 2007; Stein et al., 2009), increased cholesterol (Nelson et al., 2010; Steenland et al., 2009), and attention deficit hyperactivity disorder (ADHD) (Hoffman et al., 2010; Stein and Savitz, 2011). Almost no data exist on the toxicity of other PFCs including longer-chain length (>C8) PFCs and a multitude of precursor compounds such as fluorotelomer alcohols (FTOHs), fluorinated sulfonamides (FOSAs) and sulfonamidoethanols (FOSEs).

Over the past few years, research examining PFC exposure pathways has increased dramatically with detectable levels being found in indoor air (Barber et al., 2007; Haug et al., 2011b; Shoeib et al., 2005, 2011), indoor dust (Björklund et al., 2009; Goosey and Harrad, 2011; Kato et al.,

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2009; Strynar and Lindstrom, 2008), and foods such as meat, fish, and dairy products (Ericson et al., 2008; Tittlemier et al., 2007). Still, it remains largely unclear whether ingestion of contaminated food and water, inhalation of indoor and ambient air, ingestion of indoor dust, or direct contact with PFC-containing consumer products is the largest contributor to human body burdens of PFCs. The majority of previous exposure studies have focused on PFOA and PFOS in dust and diet, estimating that dietary sources dominate exposure in adults except in some worst-case scenario estimates that use a high dust ingestion factor and maximum PFC dust concentrations (Björklund et al., 2009; Egeghy and Lorber, 2011; Lorber and Egeghy, 2011; Zhang et al., 2010). However, the true contribution of different exposure pathways to PFC body burdens remains unclear due to limited data on adult dust ingestion rates, absorption capacities, PFCs levels in indoor air, and PFCs levels in the air and dust of places where people spend significant amounts of time other than their homes (e.g., offices). Additionally, and importantly, the role of precursor compounds such as FTOHs, FOSAs and FOSEs in either air or dust has yet to be adequately assessed.

Accordingly, the primary objective of this study was to address these gaps by building on our previous work in which we found that concentrations of FTOHs in indoor air were particularly high in offices of a newly constructed building and significantly associated with serum PFOA (Fraser et al., 2012). In the current paper, we report on PFC concentrations in the dust of those same offices as well as in dust collected from the homes and vehicles of the same participants. We assess the relationships between PFC dust concentrations in these three microenvironments and levels of PFCs in the office workers' serum. Lastly, we compare exposure to FTOHs via office dust and office air with respect to their ability to predict concentrations of PFOA and perfluorononanoate (PFNA) in the office workers' serum.

2. Materials and methods

2.1. Study design

We recruited a convenience sample of 31 office workers who live and work in the greater Boston, MA area. Participants were 90% white, 84% female and ranged in age from 25 to 64 years. Dust samples were collected from each participant's home, office, and vehicle (where available), while indoor air samples were collected from offices only. The field investigation also included the collection of a blood sample and administration of a questionnaire to gather information on demographics, microenvironment characteristics (including information about renovations), time spent in different microenvironments, and diet. All samples were collected between January and March of 2009.

Study participants worked in separate offices that were located throughout seven buildings, which were categorized into three groups: Building A ($n = 6$), Building B ($n = 17$), and Other ($n = 8$). Building A was newly built approximately one year before the study began and contained new carpeting throughout hallways and offices, as well as new upholstered furniture in each of the offices. Building B was partially renovated approximately one year before the study began, including the installation of new carpeting throughout hallways and in approximately 10% of offices. The five remaining buildings (Other building category) were not known to have undergone recent renovation. All offices contained four solid walls and a door that was closed each evening. Two-thirds of offices contained at least one window and the average office size was 38 m^2. No two participants shared an office.

Air samples were collected from the 31 offices during a four-day period between Monday at 8 AM and Friday at 8 AM using an active air sampling pump and polyurethane foam (PUF)/XAD-2 cartridges. A detailed description of the methods for the collection and analysis of office air samples were reported previously (Fraser et al., 2012).

On the last day of air sampling and after removal of air sampling equipment, dust was collected from each of the offices and a blood sample was collected from each participant by a trained phlebotomist. Arrangements were made through each building's facilities management office to ensure that study offices were not vacuumed during the sampling week. Participants were also asked not to dust or vacuum their homes and vehicles for at least one week prior to the home sampling visit, which occurred either mid-week during the sampling period or, occasionally, during the following week. Informed consent was obtained from each participant prior to data collection and the study was approved by the Boston University Medical Center's Institutional Review Board. The involvement of the Centers for Disease Control and Prevention (CDC) and the U.S. Environmental Protection Agency (EPA) laboratories were determined not to constitute engagement in human subject research.

2.2. Dust sampling

The dust sampling media consisted of a cellulose extraction thimble (Whatman International) inserted between the crevice tool and vacuum tube extender of a Eureka Mighty-Mite vacuum cleaner (Allen et al., 2008). Offices and the main living area of homes were vacuumed for approximately 10 min, covering the entire floor surface area including accessible floor space under desks and the tops of immovable furniture. Vehicles were also vacuumed for approximately 10 min, covering the entire surface area of the front and back seats. Vehicle floorboards and dashboards were not vacuumed. A total of 12 dust field blanks (six from offices and six from homes) were collected using sodium sulfate powder as a surrogate for dust.

After vacuuming, sample thimbles were removed, wrapped in aluminum foil, sealed in polyurethane zip-lock bags, and stored at room temperature for an average of 2 months until sieving. Dust samples were sieved to a particle size of <500 μm, placed in clean amber glass jars, and stored at -20 °C until they were shipped to the National Exposure Research Laboratory at the U.S. EPA for analysis.

2.3. Analysis of dust samples

Approximately 50 mg of each dust sample were sonic extracted with methanol and centrifuged to pelletize the dust. The supernatant was passed through a 3 cm^3 Supelclean ENVI-Carb 250 mg phase cartridge (Supelco, Bellefonte, PA) that was pre-treated with 5 mL of methanol (2×). The eluate was captured and evaporated to approximately 0.5 mL and prepared for ultra performance liquid chromatography tandem mass spectrometry (UPLC/MS/MS) analysis by mixing the methanolic extract with 2 mM ammonium acetate at a 60:40 ratio. Analytical batches consisted of method blanks, solvent blanks, QA/QC samples (NIST SRM 2583), calibration curve samples, and unknown samples. All samples underwent the same extraction procedure.

Analytes included 3 perfluorinated sulfonates (perfluorobutane sulfonate [PFBS], perfluorohexane sulfonate [PFHxS], and PFOS) and 9 perfluorinated carboxylates (C4–C12: perfluorobutyrate [PFBA], perfluoropentanoate [PFPeA], perfluorohexanoate [PFHxA], perfluoroheptanoate [PFHpA], PFOA, PFNA, perfluorodecanoate [PFDA], perfluoroundecanoate [PFUnA], and perfluorododecanoate [PFDoA]). In addition, samples were analyzed for fluorotelomer alcohols (6:2, 8:2 and 10:2 FTOH), FOSE alcohols (N-Me FOSE and N-Et FOSE), and C13 (perfluorotridecanoate [PFTrDA]) and C14 (perfluorotetradecanoate [PFTeDA]) carboxylic acids using the same extraction procedure as above with the following differences. Samples were prepared 80:20 (methanol:2 mM ammonium acetate) and analyzed via high performance liquid chromatography time-of-flight mass spectrometry (HPLC/TOFMS).

Samples were analyzed for C4–C12, PFBS, PFHxS, and PFOS using a Waters Acquity UPLC interfaced with a Quatro Premier XE triple quadruple mass spectrometer (Waters Corp. Milford, MA). Samples were analyzed for the remaining analytes (FTOHs, FOSE alcohols, PFTrDA, and PFTeDA) using an Agilent 1100 HPLC interfaced with an Agilent 6200 TOF-MSD (Agilent Technologies, Palo Alto, CA). Additional details