PFOA COMMUNITY HEALTH STUDIES: EXPOSURE VIA DRINKING WATER CONTAMINATED BY A TEFLON MANUFACTURING FACILITY

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Introduction

Perfluorooctanoate (PFOA, C8) is a surfactant used in the manufacturing of fluoropolymers. Common uses of fluoropolymers include non-stick cookware such as Teflon®, protective polymer for carpets and clothing, and weather-resistant coating for construction materials. PFOA is persistent in the environment, and data from the National Health and Nutrition Examination Survey (NHANES) 2003-2004 reported a median serum PFOA level in the general U.S. population of 3.9 μg/L. [1] Animal studies suggest that exposure to PFOA may cause some cancers, adverse reproductive health outcomes, and liver damage, but few human studies have been conducted to provide reliable evidence.[2] Currently, the primary routes of exposure are unknown but the U.S. EPA is working with industry and other stakeholders to improve its understanding of the sources and pathways of exposure to PFOA.[3] As part of this risk assessment work, EPA signed a Memorandum of Understanding (MOU) with DuPont (the manufacturer of Teflon®) in November 2005 for extensive environmental monitoring at and near the Washington Works facility in Parkersburg, West Virginia. The facility is located on the Ohio River and released PFOA into the air, ground water, and Ohio River from the 1950s until recently. Peak PFOA emissions occurred in the late 1990s. PFOA was detected in public drinking water districts in Ohio and West Virginia during well survey sampling in 2002.[4] A class action lawsuit brought by the surrounding communities against DuPont resulted in a settlement agreement whereby Brookmar, Inc., an independent company, conducted a year-long survey (August 2005 - July 2006) called the C8 Health Project.[5] The drinking water of the survey participants was supplied from either private wells in the contaminated area or at least one of the following public water supplies: Little Hocking Water Association, Tuppers Plains-Chester Water District, the Village of Pomeroy Public Service District, or Belpre Public Service District in Ohio; the Public Service Districts of Lubeck and Mason County in West Virginia. To investigate the health effects of PFOA released from the facility into the environment, the settlement also established a Science Panel of public health scientists to assess whether or not there is a probable link between PFOA exposure and disease in the community.[6] This paper describes the Science Panel's community health studies and examines the relationship between PFOA in drinking water and serum levels.

Materials and Methods

Our research focuses on a U.S. population exposed via drinking water to PFOA for up to several decades. The Science Panel designed a series of studies to assess exposure and investigate health effects, some based on existing data from the C8 Health Project, while others require collection of new data, including interviews and blood samples. The C8 Health Project gathered information through questionnaires and collected serum samples from over 69,000 people living near the Washington Works facility. The median PFOA serum level in the C8 Health Project population was 28.2 μg/L (range, 0.50-22412 μg/L)[7], seven times higher than the U.S. median in the 2003-2004 NHANES data.[1] The questionnaire collected information on a range of conditions including cancers, pregnancy outcomes, heart disease, diabetes and autoimmune diseases. Data on potential confounders including age, gender, body mass index, smoking and drinking habits, household income, education, and occupational history were also collected. In addition to the C8 Health Project data, Science Panel members will also gather information from the Ohio and West Virginia state health department Cancer Registries and Vital Records. Of the 69,030 participants in the C8 Health Project, over 70% consented to the Science Panel's use of their identifiable data such as name and social security number. The Science Panel will link C8 Health Project data to available disease registries, birth certificates, and death records. Residential

history and information on drinking water source and bottled water consumption from the C8 Health Project survey will be used to assess environmental exposure to PFOA.

We used a first order steady-state pharmacokinetic model to explore the relationship between PFOA drinking water concentrations and PFOA serum levels for the population living near the Washington Works facility. We estimated the concentration ratio of the serum PFOA concentration to drinking water PFOA concentration for adults assuming a half-life of 3.8 years (from an observational study of fluorochemical production workers) and 100% absorption.[8,9] The volume of distribution used was 12.7 L for men and 11.9 L for women based on results from monkey experiments scaled to humans using 70 kg and 60 kg for male and female body weights.[9] The distribution of PFOA in animal models suggests that PFOA is primarily distributed in the blood, liver, and kidney. In the absence of drinking water consumption data, we used the U.S. EPA's recommended average tap water intake rate for adults of 1.41L/day.[10]

We calculated an estimated steady-state PFOA serum concentration for the long-term C8 Health Project population by multiplying the concentration ratio and the mean PFOA water concentration for the six public water districts. We used data from well sampling conducted by DuPont for each water district to calculate PFOA water concentrations. The sampling was undertaken in response to a Consent Order issued jointly by the West Virginia Department of Environmental Protection and Bureau for Public Health.[4] The values presented in DuPont's summary reports are ammonium perfluorooctanoate (APFO) concentrations (the salt form of PFOA). We calculated the mean APFO concentrations for the six water districts, excluding any samples taken after treatment, and mathematically converted to PFOA concentrations using the ratio of PFOA:APFO molecular weights (414/431).

For comparison, we predicted adjusted geometric mean PFOA serum levels using a multivariate linear regression model. The model examined data from a subset of C8 Health Project participants with valid serum measurements. We excluded participants who reported using private wells for drinking water, having worked at the DuPont Washington Works facility or those with missing covariate data. We estimated the concentration ratio at steady-state, so only participants with at least 10 years of service from one of the six public water supplies at the time of the survey were included. $\log_{10}$[PFOA] was modeled as a function of water district coded using indicator variables and adjusted for age, sex, and body mass index (BMI). We used the resulting model to predict PFOA serum levels using the population average age/sex/BMI values.

Results and Discussion

A series of ten different studies were designed to provide a comprehensive understanding of PFOA exposure and human health in the population surrounding the Washington Works facility. They are discussed in detail at www.c8sciencepanel.org and briefly described here. The series consists of two cross-sectional studies using the existing C8 Health Project data to investigate (1) cardiovascular risk factors measured in blood such as total cholesterol, triglycerides, uric acid, red blood cell counts, glucose and (2) cancer, immune function, liver and hormone disorders; two prospective studies that follow until 2010 (3) a subset of the C8 Health Project participants and (4) DuPont Washington Works plant workers; two birth outcomes studies that (5) use birth records from exposed and unexposed geographical areas in the Mid-Ohio Valley and (6) use C8 Health Project participant data linked to birth records; two studies that involve re-contacting C8 Health Project participants for additional blood samples and interview data to conduct (7) a much more complete set of tests of the immune, liver, and endocrine systems and (8) a pharmacokinetic study to determine the rate of PFOA removal from the body; (9) an assessment of PFOA exposure by modeling levels in groundwater using historical meteorological and hydrogeological data, emissions records from the facility, and reconstruction of the historic water district service areas; (10) a geographic cancer incidence study relating cancer incidence rates to PFOA exposures by area; and (11) a neurobehavioral development study. Results of the exposure assessments will be used by all of the health studies. Some of these studies, including the pharmacokinetic study, are already underway. This is the largest survey ever conducted in a community with elevated exposure to PFOA, offering a valuable opportunity to assess relationships with a wide range of clinical parameters as well as diagnosed disease.

The PFOA steady-state concentration ratio estimated using a simple first order pharmacokinetic model was 222. This result is comparable to other analyses employing similar models.[11,12] Hinderliter and Jepson (2001) calculated a steady state concentration ratio of 300 using a half-life of 1 year, a volume of distribution of 3.5 L (assumed PFOA was distributed only in the blood compartment), and an ingestion rate of 2 L/day. Gray (2005) calculated a steady state concentration ratio of 150 using a half-life of 4.4 years and an ingestion rate of 2 L/day. To estimate the steady-state PFOA serum level, we multiplied the concentration ratio of 222 by the mean PFOA water concentrations for the six water districts.

The adjusted geometric mean PFOA serum concentrations by water district for a subset of the long-term C8 Health Project population were similar to the averages published for the whole population.[7] The ratio of the geometric mean PFOA serum concentrations to measured water concentrations in these long-term residents is comparable to our estimated steady-state concentration ratio of 222, although there is variation by water district. The predicted adjusted geometric means were lower than the modeled steady-state estimates, but within the same order of magnitude, suggesting the pharmacokinetic model provides a reasonable first order estimate.

Reasons for differences between the estimated steady-state and the predicted concentration ratios include uncertainty about drinking water consumption (e.g, use of bottled water due to knowledge of the contamination), changing water concentrations over time (so that the serum/water ratio is not at steady state), and use of a very simple pharmacokinetic model. Another uncertain parameter is the half-life of PFOA. The Science Panel studies will provide valuable information on these variables including the secular trends in water concentrations and the rate of PFOA removal in this population.

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