



PERFLUOROCTANOIC ACID (PFOA) IN NEW JERSEY DRINKING WATER: OCCURRENCE, EXPOSURE SIGNIFICANCE, AND HEALTH-BASED GUIDANCE

Gloria B. Post¹, Keith R. Cooper², Betty Jane Boros-Russo³, Judith B. Louis¹, and R. Lee Lippincott¹

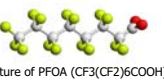
Division of Science, Research and Technology¹
Bureau of Safe Drinking Water¹
New Jersey Department of Environmental Protection, Trenton, NJ

Department of Biochemistry and Microbiology²
Rutgers University, New Brunswick, NJ

ABSTRACT

Perfluorooctanoic acid (PFOA) is ubiquitous in the blood of humans, with a mean serum concentration in the US of about 4 ug/L. Sources of PFOA exposure include food, house dust, consumer products, and drinking water. Human exposure is of concern because of PFOA's long half-life and toxicity. Little is known about the general occurrence of PFOA in drinking water or its contribution to total human exposure. PFOA was found in 24 of 30 (80%) of NJ public water supplies tested at levels ranging from <0.004-0.19 ug/L. These concentrations may contribute significantly to total human exposure, based on an approximate 100:1 ratio between the concentration of PFOA in serum and in drinking water observed in OH and WV communities with both high (>3 ug/L) and low (<0.1 ug/L) drinking water levels. A lifetime drinking water guidance for PFOA was developed based on evaluation of NOAELs and LOAELs, as well as cancer data, from animal studies identified in a USEPA draft risk assessment (2005). Since the half-life of PFOA in humans is much longer than in animals, the drinking water guidance was based on comparison of blood levels in animals and humans, rather than on administered doses. The 100:1 ratio between serum and drinking water concentrations of PFOA was used to develop health-based drinking water concentrations for non-cancer and cancer endpoints. The most sensitive endpoints were decreased body weight and hematological effects in a chronic study in female rats. The guidance value based on these endpoints is 0.04 ug/L, while the drinking water concentration based on cancer at the 1 x 10⁻⁶ risk level is 0.06 ug/L. Recent data from animal and human studies not considered in the USEPA risk assessment, including developmental effects in mice and decreased birth weight and other measures of fetal growth in humans, further support this health-based drinking water guidance. While PFOA in most NJ water supplies was below the health-based drinking water guidance of 0.04 ug/L, several NJ water supplies exceeded this concentration.

The views expressed in this presentation are those of the authors and do not represent the policies of the New Jersey Department of Environmental Protection.



Structure of PFOA (CF3(CF2)6COOH)

OCCURRENCE OF PFOA IN NEW JERSEY PUBLIC DRINKING WATER SUPPLIES

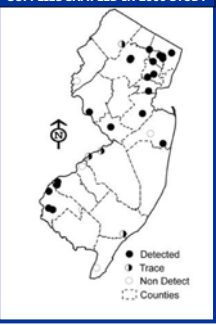
Initial Detections

- In 2006, PFOA was detected at up to 0.018 ug/L and 0.19 ug/L in wells of two public water systems located near a New Jersey facility that used PFOA in manufacturing and processed waste containing PFOA.
 - A median ratio of about 1:100 between PFOA concentration in drinking water and serum in Little Hocking, Ohio, a community served by drinking water with a high level (~3.5 ug/L) of PFOA was reported by Emmett et al. (2006).
 - Data from other communities served by drinking water with lower PFOA concentrations were evaluated to determine whether the 1:100 ratio is valid at lower drinking water concentrations such as those found in New Jersey.
- 2006 New Jersey Department of Environmental Protection (NJDEP) Study
- PFOA was detected at >0.004 ug/L in 20 (69%) of 29 samples from 15 (65%) of 23 additional public water systems in a study conducted by NJDEP in 2006. The highest concentration detected was 0.039 ug/L. Trace levels (<0.004 ug/L) were found in 4 samples from 3 systems.
 - The study included ground water and surface water systems. Some systems had a history of contamination by synthetic organic chemicals or were located near facilities where PFOA may have been present. Other systems had no history of contamination or were chosen to expand the geographical extent of the study.

PFOA in New Jersey Public Water Supplies Sampled by NJDEP in 2006

Concentration	Number of Samples (n=29)	Percent of Samples
Not Detected	3	10%
Trace (<0.004 ug/L)	4	14%
>0.004 - 0.01 ug/L	7	24%
>0.01 - 0.02 ug/L	2	7%
>0.02 - 0.03 ug/L	7	24%
>0.03 - 0.04 ug/L	4	14%

LOCATIONS OF NJ PUBLIC WATER SUPPLIES SAMPLED IN 2006 STUDY



BACKGROUND

Environmental Occurrence and Fate

- PFOA has been used for about 50 years as a processing aid in the manufacture of fluoropolymers. It is also a breakdown product and impurity of fluorinated telomers.
- Uses of fluoropolymers and fluorotelomers include non-stick cookware, waterproof breathable clothing, various industrial applications, fire fighting foams, and stain resistant coatings for textiles, food packaging paper, and carpets.
- PFOA has been emitted to air and water from industrial facilities. It was also formerly released through use in fire fighting foams.
- Groundwater contamination near industrial facilities has occurred through releases to air—soil deposition—migration to groundwater.
- PFOA does not degrade in the environment, and it has been detected in surface water, ground water, and drinking water, as well as in wildlife and other environmental media worldwide.
- Use of PFOA is currently being phased out, but fluorotelomer alcohols, related products that can degrade to PFOA in the environment and in the body, are not.

Human Exposure and Health Effects

- Sources of human exposure include consumer products, house dust, food, and drinking water. The relative contributions of these sources are not fully characterized.
- PFOA is found in the blood of the general population, with a median US serum level of about 4 ug/L (Calafat et al., 2007). It is persistent in the body, with a half-life of several years in humans.
- Unlike most bioaccumulative organic chemicals, PFOA is water soluble and accumulates in the blood rather than in the fat.
- In a community served by drinking water with a high level of PFOA (>3 ug/L), the median serum level was about 100-fold greater than the drinking water concentration.
- In animal studies, PFOA caused developmental effects, liver toxicity, effects on lipid metabolism, neurobehavioral effects, and immunotoxicity, and it induced tumors at several sites.
- In humans, serum PFOA levels have been associated with several effects including increased risk of elevated cholesterol and uric acid, decreased fetal growth, infertility (associated with PFOA serum levels in women), and decreased number of normal sperm in young men. These associations were seen at the levels of exposure in the general population or in communities with contaminated drinking water.

PFOA Detections Above Health-based Guidance (0.04 ug/L) in New Jersey Public Water Supplies, 2007-2009

Public Water Supply	Source Water	Sample Type	Number of Samples	Average (ug/L)	Range (ug/L)
1	Groundwater	PCE 1	4	0.038	0.028-0.048
2	Groundwater	PCE 2	4	0.019	0.014-0.027
3	Groundwater	PCE 3	3	0.029	0.018-0.040
4	Groundwater	PCE 4	5	0.063	0.042-0.1
5	Groundwater	PCE 5	6	0.078	0.055-0.11
6	Groundwater	PCE 6	6	0.079	0.053-0.11
7	Surface water	PCE 7	1	0.049	0.049
8	Groundwater	PCE 8	4	0.018	0.012-0.026

OBJECTIVES

- To study the occurrence of PFOA in New Jersey public drinking water systems, following detection of PFOA in a public water system at up to 0.19 ug/L in 2006.
- To evaluate the exposure contribution from drinking water at concentrations found in New Jersey relative to total human exposure to PFOA.
- To develop a health-based drinking water guidance protective for lifetime exposure, in order to assess the potential health significance of PFOA detected in New Jersey drinking water.

CONTRIBUTION TO PFOA EXPOSURE FROM DRINKING WATER

- Water supplies in several communities in Ohio and West Virginia have been contaminated by PFOA emissions from a manufacturing facility.
- A median ratio of about 1:100 between PFOA concentration in drinking water and serum in Little Hocking, Ohio, a community served by drinking water with a high level (~3.5 ug/L) of PFOA was reported by Emmett et al. (2006).
- Data from other communities served by drinking water with lower PFOA concentrations were evaluated to determine whether the 1:100 ratio is valid at lower drinking water concentrations such as those found in New Jersey.

PFOA Concentrations in Water and Serum in Water Districts in Ohio and West Virginia

Water District	PFOA Levels Reported by Water District (ug/L) (Anderson-Mahoney et al., 2008)	Median Serum PFOA Concentration (ug/L) (Steenland et al., 2008a)
Little Hocking, OH	1.7-4.3	234
Labuck, WV	0.4-3.9	70
Tuppers Plains, OH	0.25-0.37	35
City of Belgov, OH	0.06-0.13	37
Mason County, WV	0.06-0.1	12
Village of Pomroy, OH	0.06-0.07	12

CONTRIBUTION TO PFOA EXPOSURE FROM DRINKING WATER (Continued)

- The mean serum concentration in all six water districts was above the US median of 4 ug/L.
- The median serum concentration increased with increasing drinking water concentration.
- The lower bound on the ratio of serum to drinking water concentrations can be estimated by assuming that none of the background level of 4 ug/L comes from drinking water. For the two districts with the lowest serum level (12 ug/L), subtracting the background of 4 ug/L gives an estimate of 8 ug/L from drinking water exposure.
- The average drinking water concentrations in these districts are estimated as 0.065 ug/L and 0.08 ug/L, and the median serum: drinking water ratios for these two districts are estimated as 123:1 and 100:1.
- The 100:1 ratio is also supported by the predictions of a published one-compartment pharmacokinetic model for PFOA (Harada et al., 2005).
- These results indicate that the 100:1 estimated ratio is valid for lower drinking water concentration such as those detected in New Jersey.
- Based on the 100:1 ratio, exposure to PFOA from drinking water at levels commonly detected in New Jersey can be significant compared to the PFOA exposure in the general population. For example, drinking water with 0.01 ug/L would contribute about 1 ug/L to PFOA in serum, or about 25% of the median serum level in the US population of 4 ug/L.

Derivation of Health-Based Drinking Water Concentrations for PFOA from Endpoints in Animal Studies

Species	Study (Year)	Endpoint	NOAEL or LOAEL (ug/L)	Average Serum Level (ug/L)	Uncertainty Factor	Target Human Serum Level (ug/L)	Health Based Concentration (ug/L)
Adult female rat	Shaw (1999, 2001)	Body Weight, Hematology	NOAEL 2.5 mg/kg/day (28 ug/L)	200 (based on median AOC)	100 (10 interspecies, 10 intraspecies)	18	0.04
Adult male rat	Reproductive reproductive, Swanson et al. (2002), Berooff et al. (2004)	Body Weight, Hematology, Weight of P-5 generation	NOAEL 1.5 mg/kg/day	40,000 (100% males)	1000 (10 interspecies, 10 intraspecies, 10 toxicokinetic, 10 toxicodynamic)	42	0.08
Non-human primate	Emmett, male cynomolgus monkeys, Emmett/Harada, 2003, Berooff et al. (2004)	Increased liver weight and prostatic metastasis	NOAEL 1 mg/kg/day	77,000 (measured)	1000 (10 interspecies, 10 intraspecies, 10 toxicokinetic, 10 toxicodynamic)	26	0.05
Pregnant female rat	Reproductive reproductive, Swanson et al. (2002), Berooff et al. (2004)	Body Weight in male P-5 pups during post-weaning	NOAEL 1 mg/kg/day	3000 (based on median AOC)	100 (10 interspecies, 10 intraspecies)	15	0.07
Male rat pups, post-weaning	Reproductive reproductive, Swanson et al. (2002), Berooff et al. (2004)	Body Weight in male P-5 pups during post-weaning	NOAEL 1 mg/kg/day	6200 (based on median AOC at week 6)	100 (10 interspecies, 10 intraspecies)	12	0.18
Female rat pups, post-weaning	Reproductive reproductive, Swanson et al. (2002), Berooff et al. (2004)	Body Weight in female P-5 pups during post-weaning	NOAEL 20 mg/kg/day	13,000 (based on median AOC at week 7)	100 (10 interspecies, 10 intraspecies)	130	0.26
Male rats, young	Shaw et al. (2002), Berooff et al. (2004)	Liver weight, hematology, and liver lesions	NOAEL 1.0 mg/kg/day (100 ug/L)	572,000 ug/L (>100% males)	100 (10 interspecies, 10 intraspecies)	5720	0.04

DEVELOPMENT OF HEALTH-BASED DRINKING WATER GUIDANCE FOR PFOA

- The starting point for the guidance is the endpoints identified in the USEPA Draft PFOA Risk Assessment (USEPA, 2005a).
- The guidance considers the large differences in half-life between humans and experimental animals by comparing animal exposures to human exposures on the basis of internal dose (serum level) rather than administered dose.
- The guidance is intended to be protective for lifetime (chronic) exposure, as are all health-based drinking water standards, drinking water guidance values, and ground water criteria developed by NJDEP.
- The guidance does not consider recent studies that were not evaluated by USEPA (2005a), including mouse developmental studies, other recent animal studies, recent human clinical data, and human birth weight studies.
- USEPA (2005) Draft PFOA Risk Assessment
- Evaluates the significance of the general population's exposure to PFOA.
- Does not address the external dose in humans (from water, food, soil, or air) which would result in a certain internal dose (serum level).
- Identifies LOAELs and NOAELs for non-cancer effects in several animal studies, but does not develop a Reference Dose or cancer slope factor.
- Develops Margins of Exposure between animal NOAELs/LOAELs and exposure of general population, based on comparison of serum levels in animal studies and in humans.
- Classified PFOA as having "Suggestive Evidence of Carcinogenic Potential" under the 2005 USEPA Guidelines for Carcinogen Risk Assessment (USEPA, 2005b).
- The USEPA Science Advisory Board (2006) disagreed and classified PFOA as "Likely to Be Carcinogenic to Humans."

Approach for Development of Health-based Guidance

- Non-cancer Endpoints:
- The approach is based on the same principles used to develop a Reference Dose, but results in a drinking water concentration (ug/L) rather than a Reference Dose (mg/kg/day).
- The starting point is internal dose (PFOA serum level in ug/L) in animals at the NOAEL or LOAEL, not the external dose (mg/kg/day) of PFOA.
- Because half-lives are so different in humans and animals, the same dose in mg/kg/day will give very different serum levels.
- Standard uncertainty factors are applied to animal serum level at the NOAEL or LOAEL to determine the target human serum level. (Similar to application of uncertainty factors to NOAEL or LOAEL to determine a Reference Dose.)
- The serum: drinking water ratio of 100:1 is used to calculate drinking water concentrations from target human serum levels. (Using the ratio of half-lives in animals and humans is an alternative approach used by others to account for animal/human pharmacokinetic differences.)
- The Default Relative Source Contribution factor of 20% is used for non-cancer endpoints.
- Cancer Endpoints:
- The target serum level at the 10⁻⁶ risk level was estimated by linear extrapolation from the serum level at which there was 10% tumor incidence in a chronic rat study.
- The 100:1 serum:drinking water ratio was used, as above, to calculate the drinking water concentration at the 10⁻⁶ risk level.
- No Relative Source Contribution factor is used for cancer endpoint.
- Cancer is not the most sensitive endpoint.

CALCULATION OF DRINKING WATER CONCENTRATION FROM TOXICOLOGICAL ENDPOINTS EXAMPLE

- LOAEL in adult male rat is **1 mg/kg/day** (4 body wt., 4 liver and kidney wt.) in two generation reproductive study.
- Blood concentration in rat at 1 mg/kg/day is modeled at **42,000 ug/L**.
- Uncertainty factor of 1000 (chronic LOAEL) is applied to serum level → 42 ug/L target human serum concentration.
- 42 ug/L x 0.2 (Relative Source Contribution factor) = **8 ug/L** (Target contribution to serum concentration from drinking water exposure)
- Concentration factor between serum and drinking water is 100:1 → Target drinking water concentration is **0.08 ug/L**.

DETERMINATION OF HEALTH-BASED DRINKING WATER GUIDANCE FOR PFOA

- A similar calculation was performed for each endpoint.
- The most sensitive endpoints were decreased body weight and hematological effects in females in chronic rat study.
- The NOAEL for female rats in the chronic study gives a target human serum level of 18 ug/L and a drinking water concentration of 0.04 ug/L.
- Endpoints from all studies evaluated give similar drinking water concentrations: 0.04, 0.05, 0.06 (cancer), 0.07, 0.08, 0.18, and 0.26 ug/L.
- The additional uncertainty factor normally used for suggestive or possible carcinogens is not needed, because risk assessment based on non-cancer endpoints is protective for cancer risk at the 10⁻⁶ risk level.
- The health-based drinking water guidance is 0.04 ug/L based on target human serum level of **18 ug/L**.

RECENT DATA NOT CONSIDERED IN DEVELOPMENT OF HEALTH-BASED DRINKING WATER GUIDANCE

- Mouse Developmental Studies**
- Earlier rat developmental studies showed little effects. The female rat is not a good model due to its very short half-life for PFOA (2-4 hours) which prevents continuous PFOA exposure to the fetus.
- The mouse is a good model for developmental studies because the longer half-life in the female mouse (17 days) results in continuous PFOA exposure to the fetus.
- Developmental effects seen in mice include full litter resorptions, fetal death, neonatal mortality, inhibition of postnatal growth and development, effects on maternal and pup mammary gland development, increased pup liver weight, and metabolic effects in adults exposed only prenatally (Reviewed by Lau et al., 2007).
- Liver Cancer in Rainbow Trout**
- The human relevance of liver tumors from PFOA in rodents has been questioned because they may occur through a peroxisome proliferation mechanism that may not be relevant to humans.
- Rainbow trout is a model for human liver cancer because both humans and rainbow trout are insensitive to peroxisome proliferation.
- PFOA enhances liver cancer in rainbow trout through an estrogenic mechanism which may be relevant to human carcinogenic potential (Tilton et al., 2008).

Health Effects Studies in Communities with Drinking Water Exposure TO PFOA

- A health study of ~70,000 Ohio and West Virginia residents exposed to ~0.06 ug/L to >3 ug/L PFOA in drinking water is currently being conducted (CS Health Project, 2009).
- It is a unique study because of the large number of subjects, and because effects are correlated with internal dose (serum level) of PFOA, rather than with drinking water concentrations.
- Serum concentrations span the range from those seen in the general population (<0.5 ug/L) to very elevated (100's and 1000's of ug/L). The median PFOA serum concentration in this population is 28 ug/L.
- Mean serum levels in the first and second deciles are 6 ug/L and 9.80 ug/L, within the range prevalent in the general population.
- The risk of clinically elevated cholesterol and uric acid in adults was significantly associated with increased PFOA serum levels in a dose-related fashion. The risks were significantly increased even in the second quartile, where serum PFOA levels were about 13 - 30 ug/L (Steenland et al., 2008b; Steenland et al., 2009). Associations of increased serum cholesterol and uric acid with serum PFOA have also recently been reported in exposed workers (Costa et al., 2009).
- Preliminary data suggest associations of serum PFOA levels with other biological endpoints, including liver enzymes, electrolytes, and indicators of inflammatory and immune response. These associations appear to occur over the entire range of serum levels including the lowest deciles. These data have not yet been adjusted for age, sex, and other factors (CS Health Project, 2009).

Other Effects Associated with PFOA Exposure in the General Population

- Maternal PFOA serum levels in the general population were associated with decreased birth weight and effects on ponderal index, head circumference, and birth length in two populations (Apelberg et al., 2007; Fei et al., 2007, 2008) although other studies did not show these effects.
- Serum PFOA levels in women in the general population were associated with infertility as measured by time to pregnancy (Fei et al., 2009).
- Serum PFOA levels in men in the general population were associated with significantly decreased number of normal sperm (Joesens et al., 2009).

POTENTIAL IMPLICATIONS OF RECENT DATA FOR HEALTH-BASED GUIDANCE

- Mouse developmental studies reveal effects of serious concern not considered in the New Jersey drinking water guidance. These studies and other recent animal studies have not yet been quantitatively evaluated.
- The health-based drinking water guidance for PFOA is based on a conservative approach using standard uncertainty factors for Reference Dose development and considering interspecies half-life differences. It is intended to protect against adverse effects from a lifetime of exposure. Drinking water concentrations based on such an approach are normally anticipated to be far below the level at which any human effects occur.
- Associations with elevated cholesterol and uric acid are seen in the range of the target serum level of 18 ug/L that is the basis for the 0.04 ug/L health-based guidance. Preliminary data suggest associations with other biological endpoints at serum levels below the target serum level of 18 ug/L.
- Associations with measures of fetal growth, infertility, and decrease in number of normal sperm have also been observed within the range of serum levels found in the general population (<10 ug/L), although other studies have not confirmed the fetal growth results.
- Thus, health-based drinking water concentrations developed from animal data using standard values for uncertainty factors and considering interspecies differences in half-life may not provide a margin of exposure from biological effects associated with PFOA in humans.

COMPARISON OF NEW JERSEY HEALTH-BASED GUIDANCE WITH USEPA PROVISIONAL HEALTH ADVISORY

- The USEPA Office of Water (2009) recently developed a **short term Provisional Health Advisory for PFOA in drinking water of 0.4 ug/L**.
- It is based on systemic effects (increased liver weight) in pregnant rats exposed in a 17 day study, which is shorter than subchronic in duration. It is not intended to be protective for longer term or lifetime exposures.
- It accounts for the difference in half-lives between rats and humans by using the ratio of half-lives instead of the standard uncertainty factor for interspecies pharmacokinetic differences. Based on pharmacokinetic principles, this approach should give consistent results with the 100:1 ratio between serum and drinking water used in the New Jersey guidance. Risk assessments previously developed by USEPA and Minnesota Dept. of Health using the ratio of half-lives were consistent with the New Jersey guidance.
- It does not consider potential carcinogenic effects of PFOA either by low dose extrapolation of tumor data or by incorporation of an additional uncertainty factor for possible carcinogenic effects.
- Continued exposure to PFOA in drinking water at the provisional Health Advisory of 0.4 ug/L is expected to increase serum levels by about **40 ug/L**, or by about **10 times** the median level of 4 ug/L in the US general population (see above). Serum levels below 40 ug/L have been associated with adverse health effects in human populations (see below).
- The **New Jersey health-based guidance of 0.04 ug/L** is intended to be protective for lifetime exposure, as are all health based standards and guidance for drinking water and ground water developed by NJDEP.
- It considers both non-cancer endpoints and cancer risk at the 10⁻⁶ risk level.
- It accounts for the difference in half-lives in animals and humans by using the 100:1 ratio observed for serum to drinking water concentrations. Based on pharmacokinetic principles, this approach should give consistent results with the ratio of half-lives used by USEPA (2009). Risk assessments previously developed by USEPA and Minnesota Dept. of Health using the ratio of half-lives were consistent with the New Jersey guidance.
- Continued exposure to PFOA in drinking water at the guidance level of 0.04 ug/L is expected to increase serum levels by about **4 ug/L**, or to about **twice** the median level in the US general population (see above).

CONCLUSIONS

- PFOA was commonly detected in New Jersey public drinking water systems using both surface and ground water sources.
- PFOA concentrations in serum from drinking water in a ratio of about 100:1 over a range of concentrations detected in drinking water.
- The contribution to exposure from low levels of PFOA in drinking water (e.g. 0.01 ug/L) may be significant relative to the total exposure to PFOA in the U.S. general population.
- Health-based drinking water guidance of 0.04 ug/L was developed based on non-cancer and cancer endpoints from animal studies.
- The primary difference between the New Jersey chronic health-based guidance and the USEPA short term Provisional Health Advisory of 0.4 ug/L is the exposure duration for which it is intended to protect.
- While PFOA in most New Jersey public water systems was below the health-based guidance, several systems consistently exceeded the guidance.
- Recent animal studies have identified additional effects not considered in developing the guidance, particularly developmental effects in mice.
- Recent human studies suggest that health-based drinking water concentrations developed from animal data using standard uncertainty factors and other health protective assumptions may not provide a margin of exposure from effects that have been associated with PFOA in humans.

REFERENCES

Anderson-Mahoney, R., Kotterman, J., Taltav, H., Gray, D., and Dalgin, J. (2008). Self-reported health effects among community residents exposed to perfluorooctanoic acid. *Environ. Health Perspect.* 116: 129-143.

Becker, J.S., Wilson, P.A., Hechtman, J.B., Calafat, A.M., Henderson, L.L., and Goldman, L.R. (2007). Cord serum concentrations of perfluorooctanoic acid (PFOA) and perfluorooctane sulfonate (PFOS) in relation to weight and size at birth. *Environ. Health Perspect.* 115: 1030-1035.

CS Health Project (2009). *WVA Data Request Website*. West Virginia University School of Medicine, Department of Community Health.

Calafat, A.M., Wong, L.Y., Kuklenyik, Z., Reidy, J.A., and Newby, L.L. Halocarbonated chemicals in the U.S. population: data from the National Health and Nutrition Examination Survey (NHANES) 2001-2004 and comparison with NHANES 1999-2000 (2007). *Environ. Health Perspect.* 115: 1596-1602.

Costa, G., Santoro, S., and Casanova, D. (2009). Thirty years of medical surveillance in perfluorinated acid production workers. *J. Occ. Environ. Med.* 51 (in press).

Fei, C., McLaughlin, J.L., Santoro, R.E., and Olsen, J. (2007). Perfluorinated chemicals and fetal growth: a study within the Danish National Birth Cohort. *Am. J. Epidemiol.* 166: 66-72.

Fei, C., McLaughlin, J.L., Santoro, R.E., and Olsen, J. (2009). Neonatal levels of perfluorinated chemicals and subfertility. *Hum. Reprod.* Advance Access published online on January 28, 2009. <http://dx.doi.org/10.1093/humrep/dpn049>

Emmett, E., Shultz, E.S., Zhang, H., Freeman, S., Oles, C., and Shaw, L.M. (2006). Community exposure to perfluorooctanoate: relationships between serum concentrations and exposure sources. *J. Occup. Environ. Med.* 48: 759-776.

Harada, H., Inoue, E., Horikawa, A., Watanabe, T., Sakai, H., and Kasuya, A. (2005). Neonatal clearance of perfluorooctanoate and perfluorooctane sulfonate in humans and their species-specific excretion. *Environ. Res.* 99: 253-61.

Joesens, L.H., Bies, L., Luffels, H., Jansen, A.A., Skaalwijk, H.G., and Jongenbos, H. (2009). Do perfluorinated compounds impair human sperm quality? doi: 10.1186/14752875 (available at <http://dx.doi.org/10.1186/14752875>).

Lau, C., Arnold, E., Hodler, C., Liu, D., and Pflieger-Hutchins, A. and Sand, L. (2007). Perfluorinated acids: A review of monitoring and toxicological findings. *Reprod. Sci.* 10: 366-394.

Steenland, K., Pfoet, T., and Santoro, D. (2008a). CS Science Panel. Status report: Factors associated with PFOA levels in a community surrounding a chemical plant. http://www.dboconcern.org/pdfs/Status_Report_factors_associated_with_pfoa_levels_042008.pdf

Steenland, K., Pfoet, T., and Santoro, D. (2008b). CS Science Panel. Status report: Association of perfluorooctanoic acid (PFOA) and perfluorooctane sulfonate (PFOS) with lipid levels among adults in a community with high exposure to PFOA. http://www.dboconcern.org/pdfs/Status_Report_Lipid_levels_042008.pdf

Steenland, K., Pfoet, T., and Santoro, D. (2009). CS Science Panel. Status report: Association of perfluorooctanoic acid (PFOA) and perfluorooctane sulfonate (PFOS) with uric acid among adults with elevated community exposure to PFOA. http://www.dboconcern.org/pdfs/Status_Report_Uric_acid_042009.pdf

Tilton, S.C., Omer, G.A., Berninghoff, A.D., Carpenter, M.H., Hendricks, J.D., Prentice, C.B., and Williams, D.E. (2006). Genetic profiles reveal an alternate mechanism for hepatic tumor promotion by perfluorooctanoic acid in rodent liver. *Environ. Health Perspect.* 116: 1047-55.

USEPA (2005). *Draft Risk Assessment of the Potential Human Health Effects Associated with Exposure to Perfluorooctanoic Acid (PFOA) and Perfluorooctane Sulfonate (PFOS)*. <http://www.epa.gov/ospp/pfoa/pfoa/pfoa.pdf>

USEPA (2005). *Guidelines for Carcinogen Risk Assessment - Risk Assessment Manual*. USEPA, Washington, DC. EPA/600/P-03/0017. <http://www.epa.gov/ncra/crtr/riskassess/pfoa.html>

USEPA and the State. http://www.epa.gov/pfoa/pfoa_06_006.pdf

USEPA (2009). *Provisional Health Advisory for Perfluorooctanoic Acid (PFOA) and Perfluorooctane Sulfonate (PFOS)*. Office of Water. http://www.epa.gov/watersciencecriteria/criteria/drinking/pfoa_pfos.pdf

FOR MORE INFORMATION:

Contact - Gloria B. Post, PhD, DABT, DABT at pfoa@dep.state.nj.us
New Jersey Health-based Guidance for PFOA in Drinking Water - [http://www](http://www.nj.gov/dep/watersupply/pfoa_dwguidance.pdf)