



December 30, 2021

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Chair  
PFAS Review Panel  
Science Advisory Board  
US Environmental Protection Agency  
1200 Pennsylvania Avenue, NW  
Washington, DC 20460

Re: Proposed approaches to the derivation of draft maximum contaminant level goals for perfluorooctanoic acid and perfluorooctane sulfonic acid

Dr. Chiu:

The American Chemistry Council (ACC) provides the following comments on the draft documents provided to the PFAS Review Panel (the Panel) for review relating to derivation of maximum contaminant level (MCL) goals for perfluorooctanoic acid (PFOA) and perfluorooctane sulfonic acid (PFOS). In light of the abbreviated review period that has been provided, we have highlighted the significant issues we have identified in the drafts to be reviewed. These issues include the following –

- The epidemiology data do not provide clear evidence of an association between PFOA or PFOS exposure and reduced vaccine response in children;
- The evidence for an increase in infection rates among children exposed to PFOA and PFOS is conflicting;
- USEPA has mischaracterized the evidence for other non-cancer endpoints
- There is a lack of consistent response in the human and animal evidence for the carcinogenic potential of PFOA;
- USEPA has not made the details of the benchmark dose and pharmacokinetic modeling available for stakeholder review and comment; and
- The relative source contribution of PFOA and PFOS in drinking water is considerably higher than the default assumption of twenty percent.



## Reduced Vaccine Response in Children

Budtz-Jorgensen and Grandjean (2018)<sup>1</sup> report two findings from the study of diphtheria and tetanus antibody concentrations associations among Faroe Islands children –

- An association between prenatal exposure to PFOA/PFOS and antibody concentrations at 5 years of age, and
- An association between PFOA/PFOS serum concentrations at age 5 and antibody concentrations at age 7.<sup>2</sup>

In an earlier publication by Grandjean et al. (2012),<sup>3</sup> however, this research group did not observe an association between maternal PFOA/PFOS serum concentrations and antibody concentrations at age 5 in a cohort of children born between 1997 and 2000. Although the researchers reported an association in a cohort of Faroe Islands children born from 2007 and 2009, serum concentrations were lower than in the earlier cohort (see Table 1).

**Table 1. Comparison of Serum Concentrations at Birth and 60 months in the Studies of Faroe Islands Children**

	Median Concentration (Interquartile Range)			
	1997-2000 Cohort <sup>a</sup>		2007-2009 Cohort <sup>b</sup>	
	At birth	At 60 months	At birth	At 60 months
PFOS (ng/ml)	27.3 (23.2,33.1)	16.7 (13.5,21.1)	n/a	4.7 (3.5,6.3)
PFOA	3.20 (2.6,4.0)	4.1 (3.3,4.9)	n/a	2.2 (1.8,2.8)
<sup>a</sup> Source: Table 2, Grandjean <i>et al.</i> 2012;				
<sup>b</sup> Source: Table 1, Grandjean <i>et al.</i> 2017a <sup>4</sup>				

<sup>1</sup> Budtz-Jorgensen E and Grandjean P. Application of benchmark analysis for mixed contaminant exposures: mutual adjustment of perfluoroalkyl substances associated with immunotoxicity. *PLoS ONE* 13:e0205388 (2018).

<sup>2</sup> The draft approaches select the benchmark dose modeling results for the serum levels at age 5 and antibody levels at age 7 from the cohort of children born between 1997-2000 to calculate the reference doses.

<sup>3</sup> Grandjean P *et al.* Serum vaccine antibody concentrations in children exposed to perfluorinated compounds. *J Amer Med Assn* 307(4):391-397 (2012).

<sup>4</sup> Grandjean P *et al.* Estimated exposures to perfluorinated compounds in infancy predict antibody concentrations at age 5 years. *J Immuno* 14(1):188-195 (2017a). Maternal serum concentrations are not provided



Among 7-year olds, the Faroe Islands researchers did not find an association between serum concentrations at 7 and antibody levels after excluding children suspected of receiving additional antibodies (*i.e.*, no booster, ER visit, or unexplained antibody increase).<sup>5</sup> Although the 2012 publication reports an association between serum levels of PFOA at age 5 and tetanus antibody concentrations at age 7,<sup>6</sup> the analysis does not control for children receiving additional antibodies between ages 5 and 7. Given the results of the prior analysis, this would appear to be a significant oversight that raises additional questions about the broad conclusion that exposure to PFOA or PFOS reduces vaccine response in children.

### Infection Rates Among Children

In the draft documents for PFOA and PFOS, EPA suggests that a decrease in antibody concentrations may reduce the prevention of diphtheria and tetanus in children. Results of associations between PFOA exposure and childhood infection are mixed, however, with studies reporting both increased and decreased associations with reported infections.<sup>7</sup> As a result, the National Toxicology Program (NTP) concluded that there is low confidence that exposure to either substance is associated with an increased incidence of infectious disease or a lower ability to resist or respond to infectious disease.<sup>8</sup>

The epidemiological evidence for an association between PFOA and PFOS exposure and hypersensitivity and autoimmune disease is also mixed. Studies that observed significant associations with “ever” or “current” asthma were seen primarily in sex- or age-specific subgroups but were null or insignificant in whole study analyses. For allergy and eczema outcomes, results were inconsistent across studies. Studies of PFOS exposure and autoimmune condition in humans are limited, and the results from studies of PFOA exposure and human autoimmune disease are mixed. While Steenland *et al.* reported an association with ulcerative colitis,<sup>9</sup> the analysis did not adequately control for confounding factors such as gastrointestinal infection and family history.<sup>10</sup>

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<sup>5</sup> Grandjean P *et al.* Serum vaccine antibody concentrations in adolescents exposed to perfluorinated compounds. *Environ Health Perspect* 125:077018 (2017b).

<sup>6</sup> No association is observed between PFOS serum concentrations at age 5 and diphtheria antibody concentrations at age 7, after adjusting for the antibody concentration at age 5.

<sup>7</sup> Steenland K *et al.* Review: Evolution of evidence on PFOA and health following the assessments of the C8 Science Panel. *Environ Int* 145: 106125 (2020).

<sup>8</sup> NTP. Immunotoxicity Associated with Exposure to Perfluorooctanoic acid or Perfluorooctane Sulfonate. NTP Monograph. US Department of Health and Human Services. (September 2016)

<sup>9</sup> Steenland K *et al.* Ulcerative colitis and perfluorooctanoic acid (PFOA) in a highly exposed population of community residents and workers in the mid-Ohio valley. *Environ Health Perspect* 121: 900-905 (2013).

<sup>10</sup> <http://www.c8sciencepanel.org/study.html>.



## Evidence for Other Non-Cancer Endpoints

In addition to vaccine antibody response, EPA calculates candidate reference doses (RfDs) for PFOA and PFOS based on recent epidemiological studies reporting an association between prenatal exposure to the two substances and decreased birth weight. Although EPA does not calculate a candidate RfD for cardiovascular disease (CVD), the Agency has developed a draft analysis of the potential for reducing CVD risks as the result of implementation of drinking water standards for PFOA and PFOS.

### Reduced Birth Weight

As noted in the draft documents, several human studies have investigated PFOA and PFOS exposure and birth outcomes, including birth weight. Most of these studies did not find an association between maternal serum levels and birth weight.<sup>11</sup> Among the negative studies was an occupational exposure study in which female workers were exposed to high levels of PFOS.<sup>12</sup> In many of those studies reporting an inverse relationship, moreover, the effect was small and limited to a single sex or exposure group.

Among the five studies for which EPA conducted benchmark dose modeling to develop a candidate RfD based on reduced birth weight, two did not report a significant association with maternal serum concentrations of PFOA or PFOS – Govarts *et al.* 2016<sup>13</sup> and Sagiv *et al.* 2017.<sup>14</sup> Moreover, Starling *et al.* (2017)<sup>15</sup> did not observe a significant association with serum concentration of PFOS and reported an association only in the highest tertile of PFOA concentration. In the study by Chu *et al.* (2020), the association was not significant in the analysis by serum concentration quartiles for either substance or in the continuous serum

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<sup>11</sup> Agency for Toxic Substances and Disease Registry (ATDSR). Toxicological Profile for Perfluoroalkyls. US Department of Health and Human Services (May 2021).

<sup>12</sup> Grice MM *et al.* Self-reported medical conditions in perfluorooctanesulfonyl fluoride manufacturing workers. *J Occup Environ Med* 49(7):722-729 (2007).

<sup>13</sup> Govarts E *et al.* Combined effects of prenatal exposures to environmental chemicals on birth weight. *Int J Environ Res Public Health* 13:495 (2016).

<sup>14</sup> Sagiv SK *et al.* Early Pregnancy Perfluoroalkyl Substance Plasma Concentrations and Birth Outcomes in Project Viva: Confounded by Pregnancy Hemodynamics? *Am J Epidemiol* 187: 793-802 (2017). The association with PFOS was not significant after adjusting for potential confounders.

<sup>15</sup> Starling AP *et al.* Perfluoroalkyl substances during pregnancy and offspring weight and adiposity at birth: Examining mediation by maternal fasting glucose in the healthy start study. *Environ Health Perspect* 125: 067016 (2017).



concentration analysis for PFOA.<sup>16</sup> The final study by Wikstrom *et al.* (2019)<sup>17</sup> reported an association with PFOA and PFOS concentration in the highest quartile of girls; no association was observed in infant boys. Calculating an RfD from these epidemiology studies is inappropriate based on the higher degree of uncertainty in the findings.

### Cardiovascular Disease

Most of the research on cardiovascular disease (CVD) associated with PFOA and PFOS has focused on blood pressure in the general adult population. These studies do not provide consistent evidence for an association between exposure to the two substances and blood pressure. Similarly, the evidence for an association between PFOA or PFOS and an increased risk of hypertension is inconsistent. Evidence for other CVD-related outcomes across all study populations is limited and inconsistent. Although there is some evidence for an association with a modest increase in cholesterol, the increase does not correlate with increased CVD. Accordingly, the C8 Science Panel found no evidence of a link with CVD, raising the distinct possibility that people with high cholesterol may retain PFOA, rather than PFOA being responsible for an increase in cholesterol.<sup>18</sup>

### **Human and Animal Evidence for the Carcinogenic Potential of PFOA**

EPA has developed a cancer slope factor for PFOA based on elevated levels of kidney cancer (renal cell carcinoma, or RCC) reported by Shearer *et al.* (2021).<sup>19</sup> The Agency concluded that the available data do not support the development of a cancer estimate for PFOS.

Shearer *et al.* (2021) identified 324 cases of renal cell carcinoma (RCC) among 75,000 participants of a multi-site study from medical centers in 10 US cities.<sup>20</sup> The subjects had baseline serum collected during 1993-2002, although the samples were not analyzed for PFOA and other PFAS until 2018. The cases were diagnosed with RCC subsequent to serum collection.

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<sup>16</sup> Chu C *et al.* Are perfluorooctane sulfonate alternatives safer? New insights from a birth cohort study. *Environ Intl* 135: 105365 (2020). While the OR for continuous serum concentration (per nanogram/milliliter) did not include 1, the confidence interval is quite wide (1.08, 5.47).

<sup>17</sup> Wikström, S *et al.* Maternal serum levels of perfluoroalkyl substances in early pregnancy and offspring birth weight. *Pediatric Res* 87: 1093-1099 (2019).

<sup>18</sup> Fletcher T *et al.* Probable Link Evaluation for heart disease (including high blood pressure, high cholesterol, coronary artery disease). C8 Science Panel (2012).  
[http://www.c8sciencepanel.org/pdfs/Probable\\_Link\\_C8\\_Heart\\_Disease\\_29Oct2012.pdf](http://www.c8sciencepanel.org/pdfs/Probable_Link_C8_Heart_Disease_29Oct2012.pdf)

<sup>19</sup> Shearer JJ *et al.* Serum concentrations of per- and polyfluoroalkyl substances and risk of renal cell carcinoma. *J Natl Cancer Inst* 113:580-587 (2021).

<sup>20</sup> The total population of 150,00 individuals was divided into two groups – screening and control. RCC cases and controls were identified from the screening group.



A control group of 324 individuals who had never had RCC was selected from among the same study participants – matched to the RCC cases by age (>50 years of age), sex, ethnicity, study center, and year of blood draw.

The researchers calculated odds ratios (ORs) for exposure quartiles and for continuous exposure, controlling for multiple potential confounding factors<sup>21</sup> in addition to the case-control matching factors. The quartiles were assigned based on serum concentrations of PFOA among controls, resulting in an uneven distribution in the ranges of the quartiles, which can skew the analyses for exposure-response trends. Unfortunately, it is unclear whether the covariates were addressed one at a time (varying each potential confounder, to see how the fit of the model changed) or all at once. No equation was presented in Shearer *et al.* (2021) to help understand their view of the interactions of all the confounders present when assessing the correlations with RCC.

As shown in **Table 2** and as emphasized with shading, the data do not support a positive dose-response relationship (CI includes 1.0) and would be considered not significantly elevated

**Table 2. Odds ratios and 95% confidence intervals (CIs) evaluating PFOA serum concentration and risk of renal cell carcinoma (Shearer *et al.* 2021)<sup>22</sup>**

Serum Concentration Quartile (micrograms/Liter)	Controls	Cases	OR	95% CI
<4.0	81	47	1.00	Reference
>4.0-5.5	79	83	1.41	0.69, 2.90
>5.5-7.3	83	69	1.12	0.52, 2.42
>7.3-27.2	81	125	2.19	0.86, 5.61
Continuous <sup>23</sup>			1.68	1.07, 2.63

\* Shading is applied to demonstrate that the 95%CI range includes the odds of 1.00, meaning the finding is *not statistically significant* and is not found to be a significantly elevated odds ratio.

<sup>21</sup> These included body mass index, smoking status, hypertension, prior freeze-thaw cycle, year of blood draw, estimated glomerular filtration rate (eGFR), and exposure to other PFAS. Several of these confounders are on their own dose-response continuum, rather than a simple yes/no comparison, which further complicates the ability to pinpoint the effects of PFOA exposure.

<sup>22</sup> Source: Table 2 of Shearer *et al.* 2021.

<sup>23</sup> Continuous OR is in relation to a 1-unit increase in serum PFOA concentration on the log base 2 scale.

for the three higher exposure quartiles after adjusting for other PFAS exposure. The results also do not suggest a dose-response pattern, and the p value for a positive trend was not statistically significant ( $p=0.13$ ) according to the researchers.

Although the OR for the continuous exposure analysis was statistically significant, questions remain about the meaning of this finding. Of primary concern is whether the single serum measurement taken prior to RCC diagnosis (1993-2002) is an appropriate measure of PFOA exposure.

Conducting an analysis for continuous exposure, in addition to the quartile analysis, helps to address the disparity in the range of the exposures in the quartiles. However, questions remain about the distribution of exposures between the two groups. The supplemental information<sup>24</sup> provided by the authors suggests that the range of serum levels was only slightly higher among the cases compared to the controls, with the exception of a serum level nearly 10 times the high end of the range in the case group. While this value may explain the use of a log base 2 scale for the continuous analysis, Shearer *et al.* do not explain the potential effect of this outlier on their results. However, the broad confidence interval in the highest exposure quartile suggests that such an explanation is necessary to adequately interpret the findings. Typical publications of this type will generally develop an equation that explains the relationship between the continuous variables, as well as provide a robust uncertainty or sensitivity analysis. These elements are missing from the Shearer *et al.* (2021) publication and would be considered “best practice” for epidemiology that is expected to become the basis for a public health regulation.

Although the researchers were able to use several factors to match controls to the RCC cases, the decision to select an equal number of controls may also limit the significance of the continuous exposure finding. While the number of controls selected per case may vary, it is common in the nested case-control literature to find four or five controls per case.<sup>25</sup> The researchers do not provide an explanation for the decision to identify only 324 controls, particularly given the fact that they appear to have had such a large pool of individuals for whom a serum sample had been collected.

Finally, a key topic related to the variety of RCC subtypes that can be diagnosed is the differentiation in tumor type, by genetic basis. An analysis of the subtype of RCC has been a

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<sup>24</sup> <https://academic.oup.com/inci/article/113/5/580/5906528#supplementary-data>

<sup>25</sup> Ernster VL. Nest case-control studies. *Prevent Med* 23(5):587-590 (1994).  
<https://doi.org/10.1006/pmed.1994.1093>



topic of recent interest<sup>26</sup> due to the variable survival rates and seemingly different course of both development and treatment. Not all RCC are the same which raises concern that any study linking PFOA to generic RCC could be conflating correlation with causation artificially, by not evaluating by RCC subtype. Analysis of the raw data by subtype may yield a different conclusion, and also provide clues to where to look in the animal data for subtle mode-of-action data that could clear up the discordance between human and laboratory animal kidney disease attributed to PFOA.

Two other publications explore the incidence of kidney cancer among residents of the Mid-Ohio Valley exposed to PFOA in drinking water – Vieira *et al.* (2023)<sup>27</sup> and Barry *et al.* (2013).<sup>28</sup> The study by Barry *et al.* was conducted in the same study area as Vieira *et al.* and likely included many of the same participants. However, Barry *et al.* included information from additional years of follow-up and provides a more recent analysis of cancer incidence in the Mid-Ohio River Valley. Also, as indicated above and as described in more detail below, Barry *et al.* includes a more comprehensive assessment of exposure. Moreover, Barry *et al.* included an analysis of cancer incidence among the workers of the manufacturing facility, whereas the previous study of these workers by Steenland and Woskie (2012)<sup>29</sup> was limited to cancer mortality.

The cohort assembled by Barry *et al.* included 28,541 residents and 3,713 workers who participated in at least one of the follow-up surveys conducted between 2008 and 2011 and for whom an exposure estimate was available. A total of 105 cases of kidney cancer were identified with a complete data set within the cohort – 87 among the residents and 18 among the workers. Barry *et al.* developed estimates of the cumulative PFOA serum concentration using the same model as Vieira *et al.*, but accounted for each participant's reported residential history, drinking water source, tap water consumption, and workplace water consumption.<sup>30</sup> The researchers calculated hazard ratios (HRs) for an increase in kidney cancer among

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<sup>26</sup> Wang Z *et al.* Cause-specific mortality among survivors from T1N0M0 renal cell carcinoma: a registry-based cohort study. *Frontiers in Oncology* (2021). <https://doi.org/10.3389/fonc.2021.604724>

<sup>27</sup> Vieira VM *et al.* Perfluorooctanoic acid exposure and cancer outcomes in a contaminated community: a geographic analysis. *Environ Health Perspect* 121: 318-323 (2013).

<sup>28</sup> Barry V *et al.* Perfluorooctanoic acid (PFOA) exposures and incident cancers among adults living near a chemical plant. *Environ Health Perspect* 121: 1313-1318 (2013).

<sup>29</sup> Steenland K and Woskie S. Cohort mortality study of workers exposed to perfluorooctanoic acid. *Am J Epidemiol* 176: 909-917 (2012).

<sup>30</sup> Based on measurements taken in 2005-2006, mean serum concentrations were 0.024 mg/L for community residents and 0.113 mg/L for workers.





residents, workers, and the combined group cohort for both continuous and quartiles of PFOA serum concentration.<sup>31</sup>

**Table 3. Exposure quartiles and continuous log estimated cumulative PFOA serum concentration and risk of kidney cancer risk with a 10-year lag (Barry *et al.* 2013)<sup>32</sup>**

Serum Concentration Quartile	Residents		Workers		Total	
	HR (95% CI)	p-Value	HR (95% CI)	p-Value	HR (95% CI)	p-Value
Quartile 1	1.0		1.0		1.0	
Quartile 2	0.94 (0.45, 1.99)	0.02	1.22 (0.28, 5.3)	0.42	0.99 (0.53, 1.85)	0.34
Quartile 3	1.08 (0.52, 2.25)		3.27 (0.76, 14.10)		1.69 (0.93, 3.07)	
Quartile 4	1.50 (0.72, 3.13)		0.99 (0.21, 4.68)		1.43 (0.76, 2.69)	
Continuous	1.11 (0.96, 1.29)	0.17	0.99 (0.67, 1.46)	0.97	1.09 (0.97, 1.21)	0.15

As a result of the additional follow up, refined exposure assessment, and larger cohort size in the analysis by Barry *et al.*, the association between PFOA exposure and risk of kidney cancer is substantially reduced. Significantly, the hazard ratio is weakest for workers with a significantly higher median estimated exposure.

Considering the uncertainty in the epidemiological database, it is important to look at the results of cancer studies in laboratory animals. While several bioassays have been conducted, none have reported an increase in kidney cancer among the exposed animals. Reported cancers have included liver, pancreas, and Leydig cell cancers. The most recent of these studies was conducted by the National Toxicology Program (NTP).<sup>33</sup> In addition, no plausible biological basis for the development of tumors from PFOA exposure has been reported. Without it, there does not appear to be sufficient information to establish causation.

<sup>31</sup> The cutoffs for the exposure quartiles are not provided in the publication of supplemental material. The model was adjusted for the same potential confounders as in the analysis by Vieira *et al.*

<sup>32</sup> Source: Barry *et al.* 2013 and supplemental material available at <https://ehp.niehs.nih.gov/doi/suppl/10.1289/ehp.1306615>.

<sup>33</sup> NTP. Technical report on the toxicology and carcinogenesis studies of perfluorooctanoic acid administered in feed to Sprague-Dawley rats. Technical Report 598. Department of Health and Human Services. Research Triangle Park, North Carolina (2019).



## Review of Benchmark Dose and Pharmacokinetic Models

In calculating the RfDs for vaccine antibody response, EPA used the results of benchmark dose (BMD) model presented by Budtz-Jorgensen and Grandjean (2018). The details of the model are not available for review by stakeholders and the validity of the model is questionable. Significantly, the dose-response relationship reported is driven by statistical, rather than clinical, significance. There is a clinical cut-off level that exists for antibody concentrations that represent long-term protection. Instead of using dichotomous antibody concentrations in the model, based on the clinical cut-off, the authors used continuous antibody concentration in order to detect evaluate a dose-response relationship.

Moreover, the estimated BMD and lower limit of the BMD (BMDL) obtained from the model are unstable. The authors reported BMD and BMDL estimates for PFOA, PFOS, and three other PFAS. Estimates for PFOA are unaffected by the mutual adjustment for other substances. For the other 4 PFAS, however, mutual adjustment yields unstable estimates that included infinity values. In addition, PFOA and PFOS have been observed to be highly correlated, but the model shows no indication of the interactions between these two compounds.

For its analysis, EPA selects the lowest BMDL from the Budtz-Jorgensen and Grandjean analysis using three models (piecewise, linear, conservative) adjusted and unadjusted for other PFAS. The dose-response relationship is only available for the linear and piecewise models from an earlier publication. Estimated BMDs and BMDLs obtained from the conservative model are almost 10 times higher compared to those of the piecewise model. The conservative model assumes no effect below the lowest observed concentration and therefore yields the highest plausible benchmark results that agree with the data.

The authors suggested a BMDL of 1 nanograms per milliliter (ng/mL) serum for both PFOS and PFOA, and that an uncertainty factor of 10 (accounted for vulnerable population) be applied as serum-based target reference concentration of 0.1 ng/mL. The suggestion of 1 ng/mL of serum PFOA is arbitrary and has no statistical or clinical significance. The uncertainty adjustment seems excessive since the data is from a vulnerable population of children. A factor of 1 or 3 would be more consistent with standard risk assessment practices.

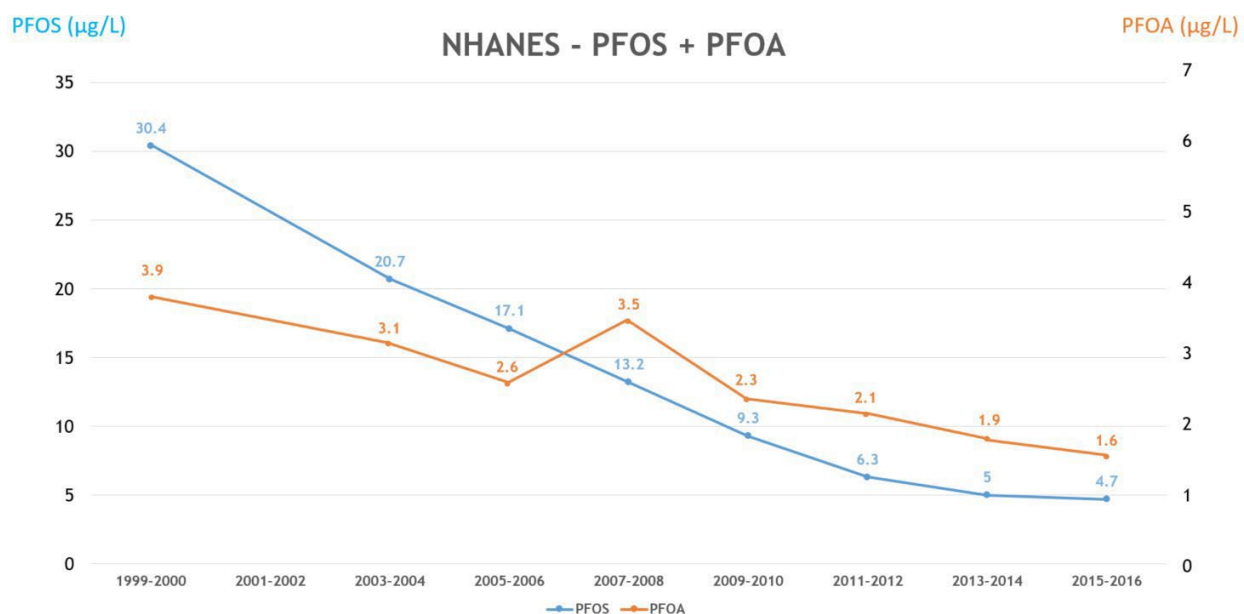
Using a no effect level of 0.1 ng/mL blood concentration in humans from the Budtz-Jorgensen publication, EPA applied a physiologically based pharmacokinetic (PBPK) model to determine what dose results in a blood concentration of 0.1 ng/ml. Despite the availability of several PBPK models in the peer reviewed literature, EPA chose to modify one of the existing model, including converting the model from one programming language to another, without submitting the new model for peer review or even making the model code publicly available.



## Relative Source Contribution

After presenting a detailed review of the potential sources of exposure to PFOA and PFOS, EPA proposes to apply a default relative source contribution (RSC) of 20 percent in developing the MCL goals – meaning that 80 percent of exposure to these substances comes from sources other than drinking water – mainly from diet and dust. However, in a 2021 survey of nationally distributed processed foods, including several baby foods, conducted by the Food and Drug Administration PFOA was not detected and PFOS was detected in only 3 of the 167 foods sampled. Moreover, while PFOA and PFOS are often detected in dust samples, the concentrations are generally not correlated with serum concentrations.

**Figure 1. Serum levels of PFOA and PFOS available from CDC.<sup>34</sup>**



The available evidence suggests that other sources of potential exposure to PFOA and PFOS have declined drastically as a result of the phaseout of these substances. According to data collected by the Center for Disease Control and Prevention (CDC), mean serum levels of PFOS declined by 85 percent in the US population since 1999.<sup>35</sup> According to CDC, mean serum levels of PFOA declined by 60 percent over the same time frame (see **Figure 1**). Given those

<sup>34</sup> Figure 1 does not include data available for 2017-18, which continues to show a decline in serum levels.

<sup>35</sup> CDC. Fourth national report on human exposure to environmental chemicals, updated tables (January 2019). <https://www.cdc.gov/exposurereport/index.html>



dramatic declines, it is inappropriate to assume that 80 percent of exposure to these substances comes from sources other than drinking water. While a few other states have assumed an RSC of 50 or 60 percent, it is likely that the contribution of drinking water to overall exposure is even higher – particularly in areas where drinking water contamination has been detected.

ACC urges the Panel to consider the information provided above as part of its careful review of the draft approach documents provided by the Agency. Please feel free to contact me if you have questions about the issue raised in this letter.

Sincerely,

***Steve Risotto***

Stephen P. Risotto  
Senior Director

