Reviewer’s report

Title: Can Profiles of Poly- and Perfluoroalkyl Substances (PFASs) in Human Serum Provide Information on Major Exposure Sources?

Version: 0 Date: 21 Nov 2017

Reviewer: Eleni Papadopoulou

Reviewer’s report:

The authors have accessed the determinants of exposure to several PFAS using a different approach. The manuscript is well-written, however there are some points throughout the manuscript that need revision.

Abstract: lines 28-30: I am not sure I agree with this argument. For example considering a woman's body burden of PFAS, I wouldn't think that her history of pregnancies or breastfeeding are «confounders» of her PFAS exposure, but rather than determinants.

Abstract: lines 30-33: including their precursors maybe, yes then this is true.

Introduction: lines 62-63: your references are focused on your populations you are aiming on studying (USA, Canada & North Norway). Then revise this sentence or revise the references.

Introduction: lines 76-88: this whole paragraph needs revision because it is written in the author's "convenience". For example, the large body of literature shows that contaminated food consumption is a major pathway of PFAS exposure as identified in several populations, while the major contributor is seafood consumption, along with meat and eggs in some populations. In addition, infants and toddlers are exposed via other pathways that are not listed here, such as trans-placental transfer, breastfeeding and consumption of contaminated food. Also, there are PBPK models that perform well and provide good agreement between the model-predicted and measured concentrations and obviously the one cited here was not a good example of a good PBPK model. The introduction section must be used to provide all the available evidence to support why the current study is needed and where are the knowledge gaps.

Methods: the Faroese population was chosen more on convenience rather than representativeness. Methods: lines 167-169: How many of the NHANES participants and the CHirP participants consume whale meat? Why the authors have not comment on the representativeness of the major dietary exposure pathway for the Faroese population.

Methods: lines 176: what was the age range of the 1,751 individuals for the period 2011-2012? The Faroese children were 13 years old, was this matching with the Americans?

Methods: lines 182-183: the CHirP women had PFAS analyzed in blood collected in the 1st trimester of pregnancy and the Faroese women had PFAS analyzed in blood sample collected 2
weeks postpartum? How can these sample be comparable, given that trans-placental transfer, blood loss and breastfeeding are major excretion pathways for PFAS?

Methods: lines 198-200: so for PFAS with detection frequency 2% you have imputed the 98%? How did you/can you check the validity of these imputed data? And why then the DF cut off is changed to <50% (lines 228-229)?

Methods: lines 204-206: why is this so important to present for your study? This is more of an effort to validate the questionnaire data.

Methods: lines 215-217: the Faroese women were 2 weeks postpartum. How is this comparable with NHANES women?

Figure 1: PFAS with DF<30-40% should be excluded from this comparison analyses as well as the PCA.

Results: lines 262-273: I do not think that is surprising that the mostly detected PFAS are loaded in the 1st component that can distinguish the whaler men, from post-partum women and 7 year old children. This is mainly related to half-lives, bioaccumulation and excretion pathways that are different in each population. And obviously whale meat in this population is a major source of exposure. Regarding the other two components if the PFAS with low detection frequencies are not removed from the analysis I would not trust the results.

Discussion: lines 325-326: how did you conclude that? Did you access the association between the 2nd component of the PCA and the use of PFAS-containing consumer products in the Faroese population? This was an assumption that became a conclusion. Regarding the seafood consumption, not the same congeners were found associated in the NHANES and Canadian populations.

Lines 334-336: I do not agree with this argument. It is not the whale consumption that differentiates the populations, but the age and the gender that modify the exposure and excretion pathways to PFAS. In addition, the correlation with mercury was only strong for PFUnDA and in the CHirP cohort shellfish consumption was associated with PFNA levels only.

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