

Response to “Comment on ‘Determinants of PFOA Serum Half-Life after End of Exposure: A Longitudinal Study on Highly Exposed Subjects in the Veneto Region’”

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We thank Olivieri et al. for their interest in our paper. We acknowledged in our paper¹ that the population investigated in the current study comprised only individuals adhering to the screening program offered to all residents of the contaminated area. We have no reason to consider them unrepresentative in ways that might affect half-life estimation. Importantly, we noted that the investigated population consisted only of subjects residing within Red Area A, including only municipalities served by the contaminated waterworks located on the groundwater contamination plume.

For many years, the predominant source of exposure to perfluorooctanoic acid (PFOA) among the study population was contaminated drinking water, which since 2013 had been largely mitigated through the implementation of charcoal filters. However, we recognized the importance of considering ongoing exposure from other sources. To address this concern, we subtracted published background concentrations reflecting general PFOA exposure to adjust for ongoing intake from other sources.² Olivieri et al. rightly highlight the possible additional local exposure in Red Area A from private wells and food irrigated with contaminated groundwater. Although we acknowledged this possibility in the discussion of our paper, the available information from the questionnaire answered by study participants pertains to the period prior to charcoal filter implementation (2013). As we did not have information on well use and local food consumption between the screening rounds, we could not address their impact on half-lives, so we did not include this in our analysis.

An estimated intake of 0.6 ng/kg/day from non–drinking water sources, cited³ by Olivieri et al., would correspond to about 6% of the average baseline water intake, based on published ratios of serum-to-water concentration.⁴ If such intake had persisted throughout the study period of our repeated observations, then adjusting the prediction model for this intake would have led to slightly shorter estimates of half-life. However, we considered an

appropriate correction to be somewhat <6%, given that industrial emissions of per- and polyfluoroalkyl substances had stopped some years before our study, and people were increasingly aware of the risk of exposure from local sources.

Regarding perfluorooctane sulfonate (and perfluorohexane sulfonate), we mentioned in the paper that the serum concentrations in the study population were comparable to those in a population outside the contaminated area. Therefore, it is evident that the additional contribution from factory emissions was quite a low proportion of total exposure, and ongoing exposure from other routes likely persisted during the follow-up period. Thus, the apparent half-life was not a good measure of the underlying half-life because exposure had evidently not stopped, as we noted in the discussion.

References

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