ASSESSMENT OF THE PFOS AND PFOA INTAKE THROUGH DRINKING WATER AND FISH CONSUMPTION IN THE ITALIAN GENERAL POPULATION

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Introduction

Perfluoroalkylated substances (PFAS) is the general name for a vast group of fluorinated chemicals comprising hundreds of compounds.[1–5] Many PFAS are commercially produced due to their surfactant properties and used in a vast number of applications. Perfluorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA) are two members of the group of PFAS. Recently these highly persistent compounds have raised scientific interest due to their occurrence in the environment, ability to bioaccumulate and biomagnify, and recognized toxicity.[2,5] PFOS and PFOA can be formed by environmental and microbial degradation or, in higher organisms, by metabolism from related substances, i.e. molecules containing the PFOS- or PFOA-moiety. The relative importance of these precursors to the current environmental load of PFOS and PFOA is yet unknown.[6]

PFAS were identified in non-stick coatings and in contact materials, such as microwave popcorn bags. Fluorotelomers present in contact materials were shown to migrate into fatty food.[7] PFAS were often detected in biota, where PFOS was generally the predominant member of the family.[8] Relatively high levels of PFOS and PFOA were found in fish and shellfish; in particular, PFOS was often found to accumulate in specific organs or tissues as a consequence of binding to proteins (e.g. in liver and plasma).

In humans, PFOS and PFOA seem to be readily absorbed following oral exposure. Metabolic elimination may not play a relevant role for their clearance, that is slow and occurs mainly *via* the kidneys and to a lower extent with the faeces. However, there are major uncertainties in what PFOS and PFOA levels in human blood represent. Their half-lives in human serum were estimated to be in the range of 5.4 and 3.8 years, respectively.[9] Epidemiological studies have suggested a possible association between occupational exposure to PFOS and bladder cancer.[10] No risk management guideline (e.g. "tolerable daily intake") has as yet been derived.

As to the occurrence of PFOS, PFOA, and other PFAS in food, there are some data concerning US and Canada, whereas data of the European Union are very sparse and mostly lacking. No specific data on the occurrence of PFAS in feed seem to be available. Until now, in the European Union there is no legislation for PFAS in food or feed. In this preliminary investigation, the dietary intake of PFOS and PFOA in the Italian population has been studied combining data from a national survey on food consumption with average concentration data estimated by the authors.

Materials and methods

Food consumption data were provided by the second nation-wide food consumption survey undertaken during 1994–1996 by the Istituto Nazionale di Ricerca per gli Alimenti e la Nutrizione (INRAN).[11] Individual food consumption was recorded for 1978 subjects over three to seven consecutive days. Subjects were randomly selected to be representative of Italy's north-west, north-east, centre, and south plus islands geographical areas.

As no data from systematic investigations of the occurrence of PFAS in food were available for the European Union countries, the exposure assessment performed here was primarily based on occurrence data selected from the scientific literature. Such data concerned drinking and surface fresh water[1,12–19] — the latter arbitrarily taken as a precursor of drinking water — and wild and farmed fish and other aquatic species.[1,19–30] No data for other food components were found. To draw a statistical profile of occurrence data, the "medium bound" approach was used.[31] Table 1 exhibits a summary of results for the two food matrices examined.

Sufficiently sensitive analytical methods are available for a number of PFAS substances in different matrices.[32] However, large variabilities (e.g. RSD up to 200 %) resulted in a recent international inter-laboratory

exercise.[33] As the data used in this work were derived from various laboratories whose sampling designs and analytical methods were not harmonized, the present assessment is presumed to entail a considerable amount of uncertainty. The assessment, limited to drinking water and fish and fishery products, was carried out for the "consumers only" subjects common to both food groups and ranked in three different groups of age (toddlers, 0.5–6 years, breastfeeding excluded; children, 7–12 years; adults, 13–94 years). For each subject, the cumulative intake was divided by the paired individual body weight (bw), as available from the INRAN data base.

Statistics were carried out with SPSS Statistical Software 13.0 and Statistica 6.1 (StatSoft Italia). Data distributions for each group were skewed to the right (high values); due to the general lack of normality (Shapiro-Wilks' *W* and Kolmogorov-Smirnov tests, $P = 0.05$), occurrence and intake data sets were evaluated with non-parametric statistics and — where possible (non-detect frequency < 50 %) — characterized by several canonical descriptors.

Results and discussion

The results of PFOS and PFOA intakes *via* food (drinking water and fish and fishery products only), grouped in the three different age ranges, are shown in Table 2.

The mean PFOS intake values estimated for toddlers, children, and adults are 81, 43, and 35 ng kg-bw^{-1} day^{-1}, respectively. The parallel PFOA intake estimates are 2.0, 1.1, and 0.88 ng kg-bw^{-1} day^{-1}. As expected, intakes decrease with age; in particular, toddlers have intakes two–three times higher than adults. This is due to the higher amount of food for body weight unit (kg-bw) consumed by toddlers in comparison to adults. In the scenario of Table 2, the high (95th percentiles) consumers in the subgroups of toddlers, children, and adults have PFOS intakes estimated at 230, 100, and 90 ng kg-bw^{-1} day^{-1}, respectively. The parallel PFOA intake values appear to be 5.6, 2.5, and 2.2 ng kg-bw^{-1} day^{-1}. In spite of the relatively lower descriptive power of PFOA data sets (Table 1), the overall picture is consistent with an average PFOA intake throughout ages of some 40-fold lower than that of PFOS.

The lack of appropriate occurrence reference data for most foodstuffs has already been highlighted. Therefore, this evaluation must be regarded as provisional while waiting for the necessary *ad hoc* food monitoring results to be gathered. At any rate, fish and fishery products seem to be an important source of human exposure.

In general, humans ingest some PFOS and PFOA with drinking water. Based on the national consumption model utilized — where the amounts of the "drinking water" ingested varies remarkably being integrated by ingestion of other drinks — drinking water appears to contribute to intake negligibly (<0.2 %) in the case of PFOS and less than 4 % for PFOA. Whether these are underestimates as contributions to intake from other drinks were not considered, is an issue to be clarified. Prolonged intake of PFAS through drinking water has been suggested to affect PFAS body burden to some extent.[13,17,34]

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Table 1. Statistical descriptors of PFOS and PFOA occurrence in drinking and surface fresh water and in fish and fishery products.[a] Values rounded off to a maximum of three figures.

PFC	N	N_{ND}	X_{MIN}	$Q_{.10}$	$Q_{.25}$	$Q_{.50}$	‹X›	SD	$Q_{.75}$	$Q_{.90}$	$Q_{.95}$	X_{MAX}
Drinking and surface fresh water (ng L^{-1})[b]												
PFOS	61	0.0	0.100	0.600	1.07	2.33	6.84	12.5	5.73	19.6	32.0	68.0
PFOA	14	14	*1.0*[c]	1.02	1.57	2.67	5.17	5.95	6.21	12.7	17.0	21.2
Fish and related items, muscle or whole body (ng g^{-1} wet weight)												
PFOS	211	26	0.114	2.00	*5.0*	14.9	43.4	60.7	69.9	93.0	127	410
PFOA	85	78	*0.10*	*0.10*	0.170	*0.85*	—	—	1.00	*1.2*	2.28	3.20

(a) Descriptors: N, number of selected original data (X); N_{ND}, fraction (%) of non-detects; X_{MIN}, lowest value; Q, percentile ($Q_{.50}$, median); ‹X›, arithmetic mean; SD, standard deviation; X_{MAX}, highest value.
(b) Many values entered in data sets for statistics as time and/or district subset averages.
(c) In italics the medium bound values ("0.5 × LD") derived from limits of determination.

Table 2. Statistical descriptors of estimated PFOS and PFOA dietary intakes (ng kg-bw^{-1} day^{-1}) for toddlers (0.5–6 years, breastfeeding excluded), children (7–12 years), and adults (13–94 years) in the Italian general population. Values rounded off to a maximum of three figures.

Population subgroup	N[a]	X_{MIN}	$Q_{.10}$[b]	X_{MEAN}	LCL[c]	UCL[c]	$Q_{.90}$	$Q_{.95}$	X_{MAX}
PFOS									
Toddlers	57	4.48	22.8	81.1	62.9	99.3	156	226	391
Children	84	2.54	5.76	42.7	35.2	50.2	84.9	100	190
Adults	1383	0.11	8.01	35.1	33.6	36.6	70.3	89.7	213
PFOA									
Toddlers	57	0.21	0.59	2.04	1.60	2.48	3.86	5.62	9.58
Children	84	0.08	0.17	1.07	0.89	1.26	2.09	2.49	4.63
Adults	1383	0.03	0.21	0.88	0.84	0.91	1.73	2.20	5.16

(a) Magnitudes of "consumers only" subgroups.
(b) Q, percentile.
(c) Lower (LCL) and upper (UCL) confidence limits (P = 95 %).