MODEL FOR PREDICTING PERFLUORINATED CARBOXYLATES IN AN ARCTIC FOOD CHAIN

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Introduction

Perfluorinated carboxylates (PFCAs), including perfluorooctanoate (PFO), perfluorononanoate (PFN), and perfluorodecanoate (PFD), have been identified in environmental samples and biological matrices in remote Arctic regions.[1-4] At present, however, mechanisms of PFCA bioaccumulation within Arctic food chains are not well defined. To help interpret the available monitoring data and explore existing uncertainties, a four-tier food chain model was developed to predict PFO, PFN, and PFD transfers from seawater through a pelagic food web comprised of zooplankton (Amphipod [*Themisto libellula*] and Copepod [*Calanus hyperboreus*]), Arctic cod, (*Boreogadus saida*), ringed seal (*Phoca hispida*), and polar bear (*Ursus marmitus*). The results of the model are compared to available field data at each tier of the Arctic food chain to assess the level of agreement between modeled and observed concentrations. The biomagnification potential of the various chain lengths of PFCAs is also evaluated by determining biomagnification factors (BMFs) for PFO, PFN and PFD for various species in an Arctic food chain. Based on previously published studies, the biomagnification potential of PFCAs is expected to be proportional to chain length with higher values for the higher molecular weight compounds. Key uncertainties and data gaps are highlighted in this paper to help guide future environmental monitoring of abiotic and biotic compartments.

Modeling Approach

Modeling was conducted to predict PFO, PFN and PFD concentrations in four biotic compartments: zooplankton (whole body), Arctic cod (whole body), ringed seal (blood, blubber and liver), and polar bear (blood and liver). For each of the four biotic compartments, a submodel was developed using laboratory-derived and field measurements of PFCAs entering the compartment via seawater and/or the diet combined with theoretical models describing the bioaccumulation of organic compounds within each compartment. Submodel predictions were compared to relevant environmental monitoring data to help determine the main route of PFCA exposure for each biotic compartment. Following the development of the four submodels, the various input parameters and equations were combined into a multi-compartment model to predict PFCA concentrations in polar bear blood and liver based on observed PFCA concentrations in Arctic seawater.

Initially, an attempt was made to develop a bioconcentration model from seawater into zooplankton based on estimates of the partitioning behavior of PFCAs to organic carbon. This approach resulted in predicted PFCA concentrations in zooplankton that were several orders of magnitude lower than measured concentrations. To provide more reasonable agreement with monitoring data, an apparent bioaccumulation factor (BAF) was calculated for zooplankton using data in Powley et al.[5] and concentrations of PFCAs in seawater and arctic ice cores:[6-8]

$$BAF_{Z,Apparent} = \frac{[PFCA]_{zooplankton}}{[PFCA]_{seawater}} \quad \text{(Equation 1)}$$

Both bioconcentration and bioaccumulation was considered in developing a submodel for Arctic cod:[9]

$$[PFCA]_{Arctic\,cod} = ([PFCA]_{seawater} \times BCF_{Trout}) + ([PFCA]_{zooplankton} \times BAF_{Trout}) \quad \text{(Equation 2)}$$

BCF_{Trout} and BAF_{Trout} values for each PFCA are based on values estimated for laboratory exposures of juvenile rainbow trout.[10,11]

Steady state concentrations of PFCAs in ringed seal and polar bear blood were estimated using a one-compartment pharmacokinetic model as described in Equation 3:[12]

$$Cs = \frac{D}{VOD \times k} \quad \text{(Equation 3)}$$

where Cs represents the steady-state blood serum concentration in the target organism, D represents the dose rate, which is determined by the ingestion rate (kg food/kg bw·d) times the PFCA concentration in the ingesta (ng PFCA/kg food), VOD represents the apparent volume of distribution (L/kg) and k represents the depuration rate constant for PFCA (d^{-1}).

After completion of the four submodels, a single multi-compartment model was developed by linking the submodels to permit the calculation of concentrations of PFCAs in polar bear blood and liver as a function of the various abiotic and biotic transfers beginning with concentrations in Arctic seawater. Trophic level biomagnification factors (BMFs) were estimated using model results and empirical field data according to Equation 4:[13]

$$BMF = \frac{[PFCA]_{Predator} \div [PFCA]_{Prey}}{\text{Predator trophic level} \div \text{Prey trophic level}} \quad \text{(Equation 4)}$$

Results and Discussion

Overall, the model predictions agree reasonably well with observed concentrations of PFCAs in polar bear liver tissue and affirm that PFCA accumulation is directly related to fluorinated carbon chain length for the compounds. However, in contrast to this general relationship, field data suggest that trophic enrichment of PFN is higher than that of PFD, especially in mid-tropic levels (fish and seals). Summaries of the results of multi-compartment models for PFO, PFN, and PFD are provided in Table 1.

Table 1: Summary of results for a multi-compartment model of PFO, PFN, and PFCA in food chain leading to polar bear.

Multi-compartment Model	Compartment	[PFCA] (ng/g, ww)	
		Predicted Median (range)	Observed Range
PFO			
seawater → zooplankton	whole organism	1.7 (0.11 - 3.4)	< 0.2 - 3.4
zooplankton + seawater → Arctic cod	whole organism	0.091 (0.0062 - 0.18)	< 0.2 - 0.5
Arctic cod → ringed seal	liver	0.51 (0.015 - 4.6)	< 0.2 - 7.0
ringed seal → polar bear	liver	1.2 (0.016 - 24)	1.1 - 55.8
PFN			
seawater → zooplankton	whole organism	0.2 (0.2 - 0.2)	< 0.4
zooplankton + seawater → Arctic cod	whole organism	0.019 (0.019 - 0.019)	< 0.4
Arctic cod → ringed seal	liver	0.62 (0.25 - 1.1)	2.4 - 27
ringed seal → polar bear	liver	7.7 (1.5 - 49)	12.2 - 540
PFD			
seawater → zooplankton	whole organism	0.80 (0.5 - 1.1)	0.5 - 1.1
zooplankton + seawater → Arctic cod	whole organism	0.15 (0.085 - 0.21)	0.28 - 0.47
Arctic cod → ringed seal	liver	5.9 (1.2 - 16)	0.98 - 19
ringed seal → polar bear	liver	100 (14 - 800)	1.2 - 209

The final products of the multi-compartment models for PFO, PFN, and PFD overlap with the range of concentrations detected in the polar bear liver, with the best agreement for the PFD and PFO models. Via Monte Carlo analysis, approximately 50%, 30%, and 80% of the predicted PFO, PFN, and PFD concentrations in polar bear liver, respectively, are within the range of measured concentrations, indicating that diet is a significant or possibly dominant exposure pathway for higher trophic levels in the Arctic.

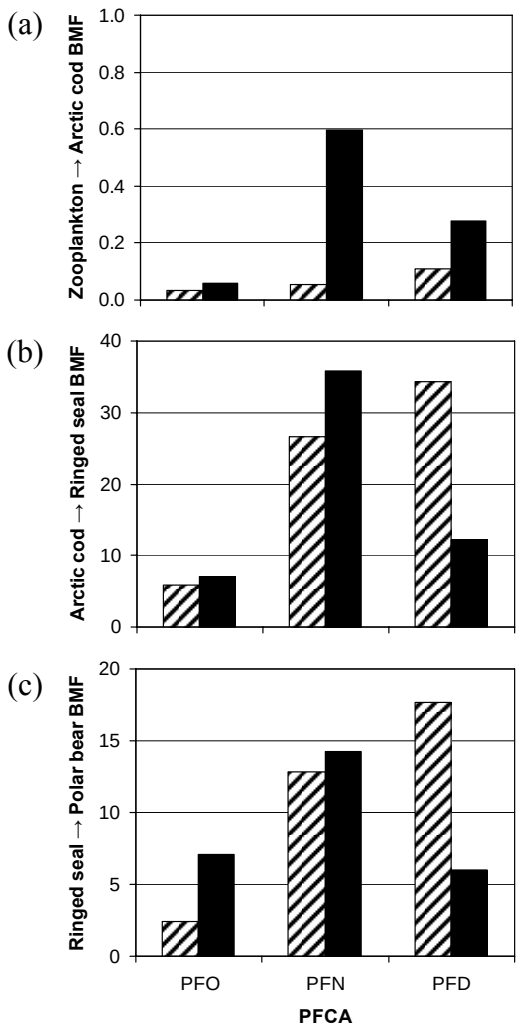

Figure 1. Trophic level biomagnification factors (BMFs) for zooplankton to Arctic cod (a), Arctic cod to ringed seal (b), and ringed seal to polar bear (c) trophic transfers of PFCAs. Striped columns represent mean BMFs based on multi-compartment model predictions; Solid columns represent mean BMFs based on empirical field data.

The pelagic food-chain model developed in this study is useful for evaluating the observed concentrations of PFCAs in various environmental compartments in the Arctic and identifying uncertainties in model predictions as well as areas for additional research or monitoring efforts. In general, the model suggests that the relative bioaccumulation potential for PFCAs in polar bear liver are expected to be PFD > PFN > PFO. This pattern emerges because the compound-specific variables in the model (BCFs, BAFs, and depuration half-lives) are directly related to fluorocarbon chain length, suggesting increased bioaccumulation potential with longer chains.[10,11,14] The biomagnification potential of PFCAs decreases with chain length in juvenile fish and this trend is likely to occur in higher trophic levels as well.[10,11] However, in liver tissues of mid-level trophic biota and in benthic fish, measured PFN, not PFD, becomes predominant, with the pattern of accumulation shifting to PFN>PFD>PFO.[1,4,15] As BMF values are independent of PFCA concentrations in seawater and other abiotic compartments, data suggest that PFN may be enriched much more efficiently than other PFCAs within the polar bear food chain (Figure 1). Assuming similar sea water concentrations and BAFs in zooplankton among PFO, PFN, and PFD, PFN will be found at the highest concentrations in polar bears simply due to biomagnification within the food chain. Thus, the explanation for higher levels of PFN in Arctic biota could be strictly related to PFN-specific pharmacokinetics and bioavailability, not higher concentrations in abiotic compartments such as seawater.

Since metabolism of telomer-based PFCAs and PFCA precursors is unlikely to lead to significant levels of PFN and exposure routes in trophic levels higher than zooplankton are primarily dietary, there is no clear biological explanation for higher biomagnification of PFN. It is possible that the pharmacokinetics of PFN in ringed seals and polar bears are not directly related to fluorocarbon chain length, despite results of laboratory studies in rodents and primates. Additional investigation on the biomagnification of PFCAs could help to improve the theoretical model, especially for middle trophic levels such as fish and seals.

The temporal and spatial pattern of PFCA accumulation in Arctic biota is also a major uncertainty and may be a source of error in compiling the results of monitoring programs. Monitoring must be carefully targeted in time

and space, and consider multiple trophic levels. For example, in lower trophic levels, PFCA accumulation may be highly dependent on the seasonal nature of Arctic ecosystems and environmental and biological processes that affect the fate and transport of persistent organic compounds.[16,17] Temporal variation in PFCA concentrations could be dominated by chemicals released when ice melts during the spring thaw, as occurs with atmospherically-deposited persistent organic compounds.[18] There is also little evidence of spatial trends of PFCA presence in the Arctic. With the possible exception of polar bear liver tissues, Arctic monitoring data for PFCAs are not yet sufficient to allow the robust spatial analysis that is necessary to better understand fate and transport mechanisms of these compounds.

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