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Estimating drinking water PFAS exposures associated with serum clinical action levels using a probabilistic toxicokinetic model

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Per- and polyfluoroalkyl substances, drinking water exposure, toxicokinetic modeling, regulatory decision-making, transgenerational transfer

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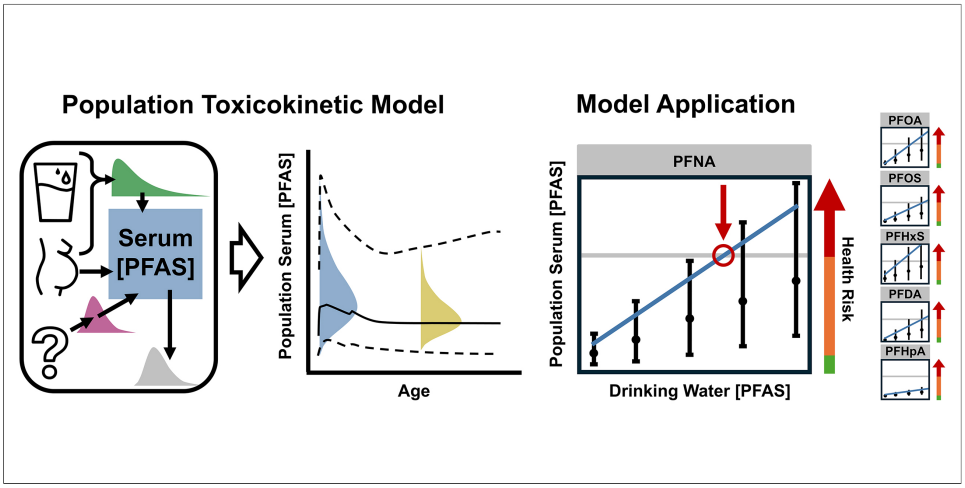

Abstract

Consumption of drinking water is a major and actionable human exposure pathway for per- and polyfluoroalkyl substances (PFAS), making it a focus of regulatory efforts. Population-based clinical guidelines recently established serum PFAS levels associated with health risk, but translating these into corresponding external drinking water exposure concentrations remains a data gap. Toxicokinetic (TK) models that relate drinking water PFAS concentrations to population serum levels provide a framework to link clinical guidelines with environmental exposure. In this study, we expand and evaluate a probabilistic, one-compartment TK model to estimate population serum PFAS concentrations for six regulated PFAS. We implement the model to estimate drinking water PFAS concentrations that maintain serum concentrations in sensitive populations below a clinical action level recommended by the National Academies of Sciences, Engineering and Medicine. Model revisions include incorporating population variability distributions for exposure factors, implementing continuous modeling from birth to account for transgenerational transfer, improving computational efficiency, and adding two additional

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PFAS. Distributions of modifiable parameters are combined using Monte Carlo simulations to predict the distribution of serum PFAS concentrations across defined populations. Model predictions reproduced empirical serum concentrations for populations of infants, children, and adults within two-fold error of observed central tendencies and upper percentiles for six PFAS. Drinking water concentrations that maintain serum concentrations below the National Academies of Sciences, Engineering, and Medicine clinical action level for breastfed infants range from 19 to 162 ng/L for the six PFAS, individually. These drinking water concentrations can inform regulatory, policy, and clinical decision-making, although they are not appropriate as health-protective drinking water standards.

INTRODUCTION

Per- and polyfluoroalkyl substances (PFAS) are a large class of fluorinated organic compounds that have been widely used in commercial and industrial products for decades. PFAS comprise a class of more than 10,000 compounds characterized by carbon-fluorine bonds that impart exceptional chemical stability and environmental persistence[1]. Only a subset of PFAS used in commercial applications have been routinely measured in the environment and in human populations. However, human epidemiological studies and large-scale population biomonitoring efforts indicate near universal exposure to some PFAS[2].

Ongoing human exposure remains a public health concern due to well-documented associations between PFAS exposure and high cholesterol[3,4], dyslipidemias[5,6], liver effects[7,8], low birth weight[9,10], immunosuppression and decreased response to vaccination in children[11], and certain types of cancers[12]. Animal toxicological studies provide supporting evidence for many of the outcomes observed in epidemiological studies[5,13-15]. Adverse developmental health effects, combined with early life exposure pathways such as placental[16,17] and lactational[18-21] transfer, make infants and young children particularly sensitive populations for PFAS exposure.

While PFAS exposure occurs through multiple pathways[22,23] including diet[24-26], household dust[27], and consumer products[28], the consumption of drinking water remains an important exposure source[29] and one that is uniquely actionable from a regulatory perspective. In the general population, drinking water is estimated to account for approximately 2%-34% of total PFAS exposure, depending on the specific compound[30]. Studies have shown that populations exposed to elevated PFAS concentrations in drinking water exhibit significantly higher PFAS blood levels than populations with lower drinking water exposure[31-38].

Drinking water regulations are one tool to reduce population PFAS exposure and communicate whether concentrations in drinking water present a health risk. Since 2016, multiple federal and state authorities have developed and promulgated drinking water guidelines and standards for PFAS[39]. These standards and guidelines, with few exceptions, have decreased over time, reflecting a better understanding of the behavior of PFAS in the environment, human exposure, and associated negative health impacts[39]. In 2020, Massachusetts (MA) promulgated drinking water standards based on protection of susceptible populations for perfluorooctanoic acid (PFOA), perfluorooctanesulfonic acid (PFOS), perfluorononanoic acid (PFNA), perfluorohexanesulfonic acid (PFHxS), perfluoroheptanoic acid (PFHpA), and perfluorodecanoic acid (PFDA)[13]. The U.S. Environmental Protection Agency (USEPA) finalized standards for six PFAS in 2024[40], then proposed rules to narrow the regulatory scope to only PFOA and PFOS in 2026[41], leaving a range of regulatory decisions to protect sensitive populations to state authorities. The evolving federal and state regulatory landscape reflects both increasing scientific clarity around PFAS health risks and continuing uncertainty about the appropriate scope of national standards.

While drinking water standards aim to reduce population health risk from this external exposure source, the National Academies of Sciences, Engineering, and Medicine (NASEM) developed complementary clinical guidelines to interpret health risks associated with measured PFAS concentrations in serum (or plasma)[42]. The NASEM clinical guidelines address an important need for communities affected by PFAS contamination by providing the only available quantitative guidelines for clinicians on interpreting individual serum PFAS testing results[42]. Following a review of available approaches that identify human serum PFAS concentrations associated with a spectrum of health risk levels, the NASEM Committee concluded that health risk increases with serum PFAS Concentrations above 20 ng/mL to a level that warrants additional clinical actions beyond the usual standard of care[42]. The NASEM clinical guidelines apply to the sum of seven PFAS routinely measured in the National Health and Nutrition Examination Survey (NHANES): PFOA, PFOS, PFHxS, PFNA, PFDA, perfluoroundecanoic acid, and methylperfluorooctane sulfonamidoacetic acid, with a note that this additive approach could be expanded to apply to additional PFAS[42].

Quantitative tools that relate drinking water concentrations to serum PFAS levels of clinical concern are helpful for integrating PFAS exposure, regulatory limits, and clinical guidance. To this end, we revised and expanded an existing probabilistic toxicokinetic (TK) model to predict population-level serum PFAS concentrations attributable to drinking water exposure. Although several TK models estimate individual serum PFAS levels, the revised model we developed generates population-level serum predictions across the life course under varying drinking water exposure scenarios and is easily modified to address a range of clinical, risk communication, and policy questions. We demonstrate a novel application to derive PFAS concentrations in drinking water that are associated with levels of clinical concern for the six PFAS that currently have promulgated MA drinking water standards.

EXPERIMENTAL

TK model overview

We revised a previously published probabilistic one-compartment TK model by Lynch *et al.* (2023)[43]. Model revisions predict the distribution of population serum PFAS concentrations attributable to drinking water exposure over the life course for six PFAS. The population PFAS TK model is designed as a tool to support clinical, policy, and other decision-making by providing a quantitative linkage between exposure to PFAS in drinking water and the distribution of resultant internal serum PFAS concentrations for populations, with explicit consideration of sensitive subgroups, including infants. Exposure pathways include ingestion of contaminated drinking water, PFAS transfer from maternal serum at birth and to breast milk, and non-drinking-water sources [Figure 1]. Chemical-specific TK parameters and age-specific exposure factors are represented with probabilistic parameter distributions, which are combined using Monte Carlo simulation to generate population serum PFAS distributions over time. All model parameters and inputs are listed in Supplementary Table 1 and described in the methods below.

We demonstrate the model capabilities by applying reverse dosimetry to estimate drinking water concentrations predicted to elevate serum PFAS concentrations in infants to the NASEM clinical action level. This approach provides a quantitative framework to evaluate the relationship between internal and external PFAS exposure and inform public health decision-making.

Model updates *vs.* ATSDR model

The model by Lynch *et al.* (2023) is intended for use by individuals and predicts age groups separately, with one model for infants/children less than 6 years old and one for children older than 6 and adults[43]. Model revisions predicting the distribution of population serum PFAS concentrations, fall into four main categories including: (1) modifications to exposure factor distributions and exposure scenario flexibility to allow population-level predictions; (2) changes to the modeling framework for infants and young children and to

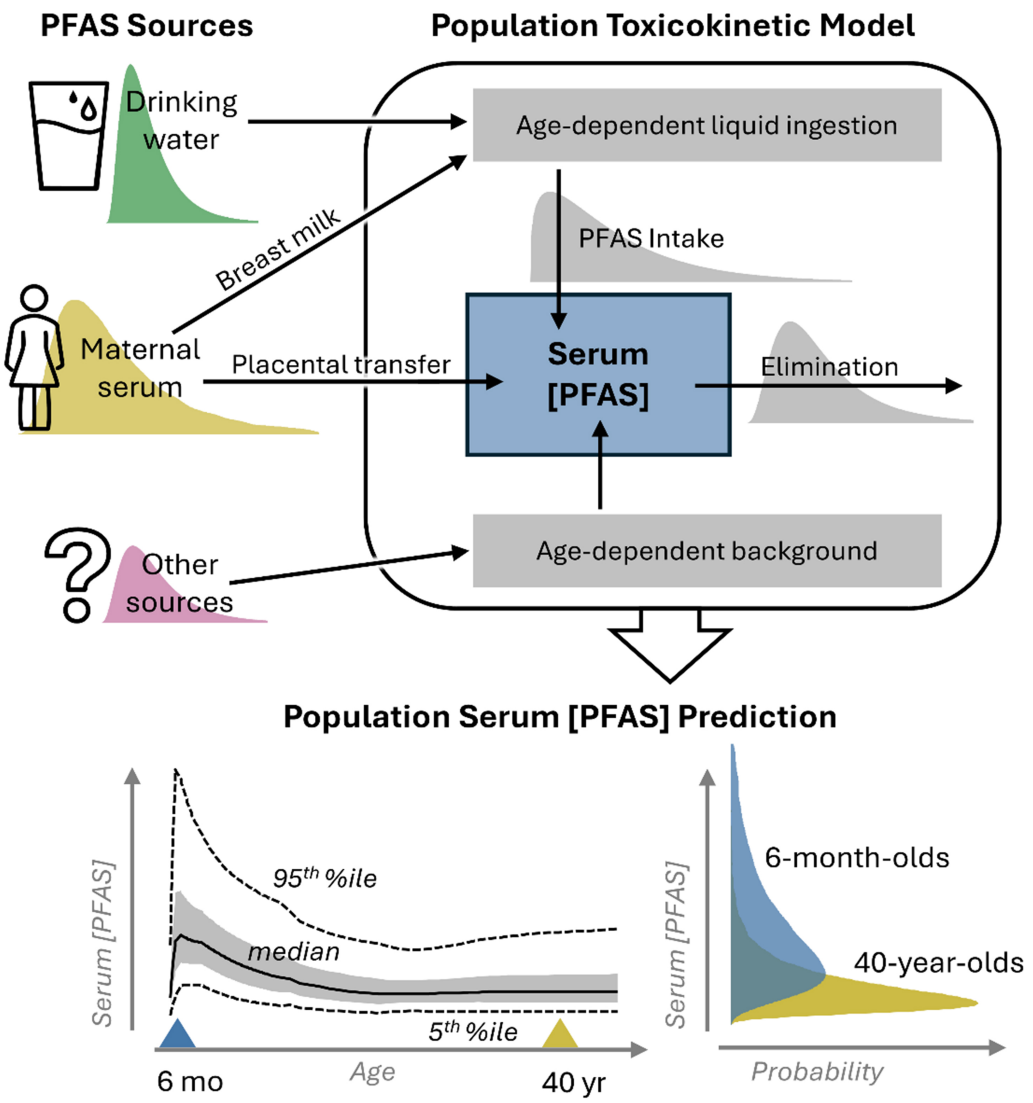

Figure 1. Overview of the PFAS TK model processes for predicting population serum PFAS concentrations ([PFAS]) from drinking water PFAS. Population distributions of chemical-specific parameters and exposure factors, some of which change with age, are combined using Monte Carlo simulation to predict the distribution of serum [PFAS] in a specified population. PFAS: Per- and polyfluoroalkyl substances; TK: toxicokinetic.

enable continuous prediction from birth; (3) model processing changes to increase computational efficiency, and (4) the addition of parameters and modeling capability for PFDA and PFHpA. Model revisions are listed and compared with the Lynch *et al.* (2023) model in Supplementary Table 2, and parameters for PFDA and PFHpA are described in Table 1[43].

Modeling population serum PFAS concentrations over time

An overview of the exposure sources, TK modeling, and model outputs is shown in Figure 1. Serum PFAS is calculated over time using the integrated rate law for the one-compartment system with zero-order intake and first-order elimination [Supplementary Figure 1][44]. Serum PFAS for a specified population is modeled across the entire life course, beginning with calculation of serum PFAS at birth (C_0) [Equation (1)][43]. For each date, the current serum PFAS and associated parameter values are used to calculate serum PFAS for subsequent dates, accounting for exposure from drinking water or breast milk, exposure from non-drinking water or "background" sources, and PFAS elimination [Equation (2)].

$$C_0 = C_M * PTF \quad (1)$$

Where:
C_M is the maternal serum PFAS concentration at birth (μg/L),
and PTF is the placental transfer factor (unitless ratio).

$$C_{i+1} = B_{i+1} + (C_i - B_i)e^{-k_{el}(t_{i+1}-t_i)} + \frac{LI_i * AF_i * LC_i}{k_{el} * V_d * 10^6}(1 - e^{-k_{el}(t_{i+1}-t_i)}) \quad (2)$$

Where:
B_{i+1} is the serum PFAS "background" on the subsequent $(i+1)^{th}$ date (μg/L),
C_i is the total serum PFAS on the i^{th} date (μg/L),
B_i is the serum PFAS "background" on the i^{th} date (μg/L),
k_{el} is the elimination rate constant (1/days), calculated from half-life $k_{el} = \ln(2)/(T_{1/2}*365.25)$,
t_i is the number of days after birth on the i^{th} date,
t_{i+1} is the number of days after birth on the subsequent $(i+1)^{th}$ date,
LI_i is the mean daily body weight-adjusted liquid intake starting on the i^{th} date [mL/(kg-day)],
AF_i is the adjustment factor for liquid intake starting on the i^{th} date (unitless ratio),
LC_i is the concentration of PFAS in the liquid consumed (ng/L),
V_d is the weight-adjusted volume of distribution (L/kg), and
the factor of 10^6 converts the third term's units to μg/L.

Modeled populations consume PFAS in either drinking water (adult) or breast milk (infant). Breast milk PFAS concentrations are determined by maternal serum PFAS concentrations at birth and decrease over time [Equation (3)].

$$BMC_i = C_M * LTF * e^{-k_{milk}*t_i} * 1000 \quad (3)$$

Where:
BMC_i is the breast milk PFAS concentration on the i^{th} date (ng/L),
C_M is the maternal serum PFAS concentration at birth (μg/L),
LTF is the lactational transfer factor (unitless ratio),
k_{milk} is the rate constant for breast milk decline over time (1/days),
t_i is the number of days after birth on the i^{th} date, and
the factor of 1000 converts units to ng/L.

TK model parameters

Half-life ($T_{1/2}$) and volume of distribution (V_d)

Parameter distributions for $T_{1/2}$ and V_d are shown in Table 1. The geometric mean (GM) and geometric standard deviation (GSD) for $T_{1/2}$ and V_d for PFOA, PFOS, PFHxS, and PFNA were calibrated from a Bayesian analysis of paired drinking water and serum concentrations in communities with PFAS exposure via drinking water[43,44]. The population data used in this analysis included males and females, resulting in distributions that capture sex-based differences in PFAS elimination for the study populations. Therefore, we used the same $T_{1/2}$ for modeling males and females. The same V_d distribution also applies to males and females. This model adds parameters and prediction capability for PFDA and PFHpA. Human TK parameter data are limited for PFDA and PFHpA. The PFDA $T_{1/2}$ was calculated using rodent-based V_d estimates and human clearance[45]. The $T_{1/2}$ value for PFHpA was derived from an analysis that used Monte Carlo simulation to aggregate available human PFHpA $T_{1/2}$ data separately for males and females[46]. The slightly

Table 1. Chemical-specific TK parameter distributions including shape, means and standard deviations, and sources of values

PFAS	Half-life (years)		Volume of distribution, weight-adjusted (L/kg)		Placental transfer factor (unitless)		Lactational transfer factor (unitless)		Breast milk elimination rate constant (1/day)[a]	
	GM (GSD)	Source	GM (GSD)	Source	Mean (2% SD)	Source	Mean (2% SD)	Source	Mean[b]	Source
PFOA	3.14 (1.57)	Chiu *et al.* (2022) [44]	0.434 (1.12)	Chiu *et al.* (2022)[44]	0.87	Lynch *et al.* (2023)[43]	0.052	Lynch *et al.* (2023)[43]	4.26×10^{-3}	Supplementary Table 3
PFOS	3.36 (1.57)		0.32 (1.10)		0.42		0.013		1.99×10^{-3}	
PFHxS	8.30 (1.57)		0.29 (1.11)		0.7		0.014		8.44×10^{-4}	
PFNA	2.35 (1.53)		0.19 (1.12)		0.53		0.01		9.11×10^{-4}	Supplementary Equation (3)
PFDA	4.72 (1.57)	USEPA (2024)[45]	0.365 (1.12)	USEPA (2024)[45]	0.32		0.023	Zheng *et al.* (2022)[50]	2.10×10^{-3}	
PFHpA	0.384 (1.57)	Dawson *et al.* (2023)[46]	0.21 (1.12)	Kabadi *et al.* (2018)[48], Ohmori *et al.* (2003)[47]	1.25	Appel *et al.* (2022)[16]	0.052	Set to PFOA	4.26×10^{-3}	Set to PFOA

[a]The breast milk elimination rate constant (k_{milk}) is calculated either from data quantifying the decrease of PFOA, PFOS, or PFHxS in breast milk over defined time periods [Supplementary Equation (2)], or using the proportionality between breast milk elimination and lactational transfer for other PFAS to estimate the elimination rate [Supplementary Equation (3)].
[b]For each iteration, the k_{milk} is adjusted relative to the LTF; k_{milk} does not have an independent distribution.
TK: Toxicokinetic; PFAS: per- and polyfluoroalkyl substances; GM: geometric mean; GSD: geometric standard deviation; SD: standard deviation; PFOA: perfluorooctanoic acid; PFOS: perfluorooctanesulfonic acid; PFHxS: perfluorohexanesulfonic acid; PFNA: perfluorononanoic acid; PFDA: perfluorodecanoic acid; USEPA: U.S. Environmental Protection Agency; PFHpA: perfluoroheptanoic acid.

longer mean female $T_{1/2}$ value from the resulting distribution is used for modeling[46]. The arithmetic mean of available V_d values from male and female rats is used for both PFDA[45] and PFHpA[47,48]. No distributional analyses for PFDA or PFHpA $T_{1/2}$ or V_d were found, so we used the PFOA GSD for $T_{1/2}$ and V_d.

Transgenerational transfer

This model requires a user-defined selection for maternal serum PFAS concentrations at birth (C_M) and includes two options: a steady-state distribution calculation from Lynch *et al.* (2023) and a new model feature to enter a C_M distribution either from TK model output or literature/known values[43]. Transgenerational transfer is then characterized by the placental transfer factor (PTF), the lactational transfer factor (LTF), and the elimination rate constant from breast milk (k_{milk}) [Table 1]. PTF and LTF are normal distributions with a 2% standard deviation[43]. k_{milk} is adjusted relative to LTF and does not have an independent distribution.

PTFs are calculated from the ratio of cord blood to maternal serum PFAS concentrations. Mean PTFs for PFOA, PFOS, PFHxS, and PFNA are adopted from Lynch *et al.* (2023)[43]. These are within 20% of the mean PTFs from a quantitative meta-analysis of 20 studies[16] and an updated PFOA PTF[49]. The PFDA and PFHpA PTFs are weighted mean estimates from meta-analyses of nine studies (total N = 1,179) and four studies (total N = 682), respectively[16].

Lactation transfer factors are the ratios of breast milk to maternal serum PFAS concentrations. The LTF determines the starting PFAS concentration in breast milk. Mean LTFs for PFOA, PFOS, PFHxS, and PFNA are adopted from Lynch *et al.* (2023)[43]. The LTF for PFDA is the mean of 23 paired serum and breast milk samples with PFDA detections[50]. The single study reporting an LTF for PFHpA had high variability, encompassing the LTF of PFOA[51]. Because of the high variability, we chose to use the PFOA LTF for PFHpA.

The breast milk PFAS concentration declines during the course of breastfeeding[52]. A breast milk elimination rate constant (k_{milk}) was determined for each PFAS. For PFOA, PFOS, and PFHxS, literature values for the monthly observed percent decrease of PFAS in breast milk were used to calculate k_{milk} [Supplementary Equation (2) and Supplementary Table 3]. For PFNA and PFDA, k_{milk} was projected from the theoretical proportionality between k_{milk} and LTF [Supplementary Equation (3)]. Because the PFHpA LTF is approximated using the LTF for PFOA, PFHpA uses the same k_{milk} as PFOA.

Liquid ingestion rates

Liquid ingestion (LI) rates apply to both drinking water and breast milk. Population variability in LI rates is incorporated in the model by multiplying a mean body weight-adjusted LI rate by an adjustment factor (AF) drawn from the AF distribution.

Age-specific mean LI rates are the combined direct and indirect water ingestion rates from the USEPA Exposure Factors Handbook (EFH) Tables 3-21 and 3-63 for ages > 1[53] or human milk intake rates from Table 15-1 for ages < 1[54] [Supplementary Table 4]. Mean LI changes each year and interpolates linearly between age-group-specific means for ages > 1. For ages < 1, mean LI updates to new mean values on the first day a new age group is achieved, without interpolation.

Population variability in LI rates for ages > 1 year was neither normally nor lognormally distributed. Variability for each age group/life stage was estimated using mean LI and associated percentiles of the population distributions supplied in the EFH. For each age group, LI at each reported percentile was divided by the corresponding mean LI to create an AF ratio. The R package rriskDistributions[55] was used to select and fit distributions to the observed percentiles for mean-adjusted LI, accounting for data with less statistical reliability [Supplementary Section 2]. Visual inspection and absolute error of fitted AF distributions for every age group showed that variability could be captured with three AF distributions: infant (0 - <1 year), child and adult (1 - <16 years and ≥ 21 years), and youth (16 - <21 years) [Supplementary Section 2]. Grouping population variability estimates across ages decreases computational complexity while retaining age group distribution characteristics.

Background serum PFAS

This model uses the same approach to incorporating exposure to non-drinking water "background" sources used by Lynch, *et al.* (2023)[43]. The general population PFAS exposure level is multiplied by 0.8, which apportions 80% of general population PFAS exposure to non-drinking water sources for incorporation in the model as "background". The apportionment is based on USEPA's default 20% relative source contribution from drinking water used when data are too limited to estimate contributions from different exposure sources[56,57]. These non-drinking water "background" sources of PFAS may include diet, dust, air, and consumer products, and these may change over time as PFAS use changes and may differ by location. General population exposure distributions for each PFAS come from from the sex-specific NHANES distribution of serum PFAS concentrations for individuals older than 12 (2017-2018 cycle) and for children ages 3-11 (2013-2014 cycle) [Supplementary Table 5]. Background at birth is set to zero and increases linearly to the child background level at age 3.

Model implementation

User-defined inputs are organized in Microsoft Excel and calculations are performed in R (Version 4.5.1 for this analysis[58]). Monte Carlo simulations combine exposure and TK parameter distributions to generate a population serum PFAS distribution. The user-specified exposure scenario includes selecting the modeling approach for maternal serum (C_M), population sex(es), age(s), duration of breastfeeding, exposure period(s) specified as dates, and PFAS drinking water concentration(s) during the exposure period(s). The population serum distribution is calculated monthly before age 1 and annually thereafter, as well as when water exposure changes.

Each iteration of the simulation draws a single value from chemical-specific parameter distributions that are constant across the life course, including $T_{1/2}$, V_d, PTF, LTF, and k_{milk}. The age-dependent parameter distributions, AF and B, are drawn independently for each age range [Supplementary Table 1].

We demonstrate the utility of the population PFAS TK model by: (1) showing predicted population serum PFAS concentrations over the life course at a constant drinking water exposure; (2) evaluating model performance using studies that report paired drinking water and serum PFAS concentrations; and (3) deriving drinking water concentrations associated with serum PFAS levels that reach the NASEM clinical action level of 20 ng/mL in sensitive populations. These three model applications are described below. We ran all modeling scenarios with 35,000 Monte Carlo iterations. This number of iterations is higher than the typical 500 to 1,000 iterations in other simulation studies and was selected to increase the reproducibility of model results across the distribution of predicted results[59]. Serum concentrations are presented in ng/mL or μg/L (1 ng/mL = 1 μg/L) and water concentrations are presented as ng/L.

Predicting lifetime population serum PFAS concentrations

To demonstrate the model's ability to capture population serum trajectories over time, we modeled a male population from birth to age 70 with constant drinking water exposure of 20 ng/L for each PFAS [Supplementary Table 6]. We used the model feature to enter a known maternal serum PFAS distribution. The maternal distribution is the predicted serum PFAS distribution of a modeled female population at age 40 with lifetime exposure to 20 ng/L PFAS in drinking water. Breastfeeding duration was set to 6 months for the male population. The only difference between modeling male and female populations is the contribution from "background", which is higher for males than females for most PFAS [Supplementary Table 5]; no other model parameters differ by sex.

We performed a local sensitivity analysis to assess how comparable shifts in select input parameters affect predicted 50th and 90th percentile serum concentrations using the output for six-month-old exclusively breast-fed male infants. For the sensitivity analysis, we used the 35,000 values of each parameter drawn for the baseline model run. A comparable shift in the mean for each variable was determined first by expressing all variables on a normal scale with lognormally distributed variables log-transformed to approximate normality; then shifting the mean value up or down by one standard deviation, yielding a new mean for the distribution. Lognormal parameters were back-transformed to their original scale. Identical positions drawn from the original variable distribution were selected from the shifted distribution, and the model was re-run with each parameter shifted up or down by one standard deviation on the normalized scale. The relative change in 50th and 90th percentile values per standard deviation change in the input parameter is compared. This approach allows for consistent comparison of parameter influence across normal and lognormal distributions and avoids Monte Carlo sampling error while maintaining the probabilistic structure of the model.

Model evaluation

To assess performance, the model was used to predict serum PFAS concentrations for published data from studies of paired drinking water and serum PFAS concentrations and studies reporting late pregnancy maternal serum PFAS levels and infant serum PFAS levels. Only studies reporting sufficient information about exposure duration, population demographics, drinking water PFAS concentration(s), and serum PFAS concentrations in the study population could be used to reconstruct a scenario for input into the model. Five suitable studies reporting results from nine populations of adults and older children were used in this evaluation[35,60-63]. The modeling scenarios for each cohort, including drinking water PFAS concentrations and exposure durations, are described in the Supplementary Table 7.

The second type of study used for model evaluation reported late pregnancy maternal serum PFAS levels and infant serum PFAS levels. Two suitable studies were identified[64,65]. The distribution of maternal late pregnancy PFAS levels was directly entered into the model, and measured infant PFAS concentrations at 6 months were compared to model-predicted results. Details on the infant populations along with the modeled scenario for each study are described in the Supplementary Table 8.

All evaluation scenarios were run with 35,000 Monte Carlo iterations to produce the model-predicted population distribution of serum PFAS levels. Summary statistics for serum PFAS levels reported for the study population were compared with the corresponding summary statistic or percentile from the modeled distribution of serum PFAS levels. For studies that reported a minimum or maximum measured concentration, these values were compared to the 5th and 95th percentiles of the modeled distribution, respectively. All studies reported at least one central tendency (median or GM) and upper percentile value (maximum, 90th, or 95th percentile).

Model performance was considered good when predicted serum concentrations fell within 2-fold of the observed values, a criterion that has been used to assess the performance of other PFAS TK models[44,66]. Root mean square error (RMSE) was calculated for the modeled *vs.* measured summary statistics for each study and was used to evaluate the relative performance of the model for different PFAS and at different percentiles, among other comparisons. A lower RMSE indicates a better fit between predicted and measured serum PFAS concentrations.

Estimation of Clinical Guidance-based Water Concentrations

The population PFAS TK model was implemented to estimate population lifetime drinking water concentrations corresponding to the NASEM clinical action level of 20 ng/mL, where additional health screening is recommended[42] for each of the six PFAS (PFOA, PFOS, PFHxS, PFNA, PFDA, PFHpA) regulated in MA. The drinking water concentration corresponding to the NASEM clinical action level is termed the Clinical Guidance-based Water Concentration (CGWC). The CGWC provides an indication of lifetime drinking water exposure levels where enhanced clinical action could be recommended. Consistent with public health approaches, we made choices in the modeling scenario (described below) to calculate health-protective CGWCs, including for sensitive populations, based on an upper-end estimate of the relationship between drinking water and serum levels; the modeling scenario was not designed to estimate the median CGWC for the US population.

We calculated CGWC for a sensitive population of breastfed infants and also calculated adult values for comparison. Infants are a sensitive population because (1) we expect infants to have higher internal serum concentrations than adults at the same drinking water exposure due to high body weight-adjusted liquid intake relative to adults [Supplementary Table 4]; and (2) infants have increased susceptibility to the adverse effects of PFAS at this life stage[45,67-70]. The higher likelihood of both exposure and adverse health effects

makes the CGWC particularly relevant for infants. Calculating CGWC for adult populations as well enables a comparison of differences between general population estimates and estimates for sensitive populations.

The infant population we selected for modeling is breastfed male infants. Due to efficient transfer from breast milk for some PFAS, breastfed infants have higher exposure than formula-fed infants; thus, using modeled breastfed infants is also protective of formula-fed infants. While breastfed infants are predicted to have higher serum PFAS levels than bottle-fed infants, breastfeeding remains important for the development of the child. We modeled male infants because the mean and GSD "background" for male children is higher than corresponding female values for most modeled PFAS [Supplementary Table 5]. As a result, drinking water concentrations corresponding to clinical action levels for breastfed male infants produce an estimate that is protective of female and formula-fed infants.

Using these two defined populations - breastfed male infants and male adults - we determined the relationship between drinking water and serum PFAS levels by modeling the population distribution of serum PFAS levels for each PFAS over a series of constant drinking water concentrations between 4 and 90 ng/L [Supplementary Table 6]. While the breastfed infant population does not consume drinking water, drinking water is used to model maternal serum concentrations which then set the initial infant serum PFAS concentration and the breastmilk PFAS concentration. For this modeling scenario, maternal serum PFAS concentrations are from a population of 40-year-old women who were breastfed for three months as infants; the infant population used to calculate CGWC had a breastfeeding duration of six months to estimate an upper-end exposure scenario [Supplementary Table 6]. This population of infants was modeled to age 40 with constant exposure at each drinking water concentration [Supplementary Table 6]. For each population, this step produced a distribution of serum PFAS concentrations for each lifetime drinking water exposure level.

We then fit a linear regression model to the 50th and 90th percentiles of the output population serum PFAS distributions with drinking water as the predictor variable. This produced two linear regression models for each population: one describing the relationship between drinking water and 50th percentile serum PFAS concentrations and the second describing the relationship between drinking water and 90th percentile serum PFAS concentrations. We used these regression models to calculate the drinking water serum concentration predicted to result in a serum level of 20 ng/mL at the 50th percentile of the distribution and at the 90th percentile of the distribution.

We defined CGWCs as the PFAS drinking water concentration predicted to result in the 90th percentile of a modeled population's serum reaching the NASEM 20 ng/mL clinical action level. We selected the 90th percentile to align with standard USEPA practice for calculating enforceable drinking water standards using 90th percentile drinking water ingestion rates[40,71].

CGWCs were calculated separately for PFOA, PFOS, PFHxS, PFNA, PFDA, and PFHpA. The NASEM clinical guidelines apply to the sum of seven PFAS regularly monitored in NHANES which does not include PFHpA (though PFHpA has been monitored previously in NHANES). We chose to extend the NASEM guidelines to PFHpA because the NASEM report indicates that the additive approach could be expanded to apply to additional PFAS[42] and PFHpA health outcomes overlap with outcomes observed for some of the NASEM PFAS, which is the justification for its inclusion in state-specific drinking water regulations[13].

The NASEM clinical guidelines apply to the sum of seven PFAS measured in serum. Cumulative risk could be evaluated using the chemical-specific CGWC by applying mixture assessment methods based on chemical additivity such as the hazard index (HI) approach[72,73]. A HI can be calculated using Equation (4), by dividing

the measured water concentration of each PFAS (DWC_{PFAS}) by the CGWC for each PFAS ($CGWC_{PFAS}$)[72]. A HI equal to or greater than one (1) indicates that the sum of the predicted serum PFAS concentrations exceeds the NASEM clinical action level of 20 ng/mL.

$$HI = \frac{DWC_{PFHxS}}{CGWC_{PFHxS}} + \frac{DWC_{PFOA}}{CGWC_{PFOA}} + \frac{DWC_{PFDA}}{CGWC_{PFDA}} + \frac{DWC_{PFNA}}{CGWC_{PFNA}} + \frac{DWC_{PFOS}}{CGWC_{PFOS}} + \frac{DWC_{PFHpA}}{CGWC_{PFHpA}} \quad (4)$$

Where:
HI is the hazard index,
DWC_{PFAS} is the measured concentration for each PFAS, and
$CGWC_{PFAS}$ is the PFAS-specific Clinical Guidance-based Water Concentration.

RESULTS AND DISCUSSION

Predicting key changes in serum PFAS concentrations over time

Model-predicted population serum PFAS concentrations change over time based on chemical-specific parameters and age-specific exposure factors [Figure 2]. For all six PFAS, model predictions show serum concentrations rise sharply by a factor of 2-5 between birth and 6 months due to high PFAS levels in breast milk and high liquid intake relative to body weight [Supplementary Table 9 and Supplementary Figure 2]. With lifetime drinking water exposure at 20 ng/L, breast milk concentrations are 2-6 times higher than the drinking water concentration for PFOA, PFOS, PFHxS and PFDA, and similar to the drinking water concentration for PFNA and PFHpA at the median of the distribution [Supplementary Table 9]. As a result, the median modeled infant serum PFAS concentrations after 6 months of exclusive breastfeeding are 1.2-3 times higher than the median modeled maternal serum PFAS concentrations [Supplementary Table 9]. Sensitivity analysis results indicate that infant serum concentrations are most influenced by maternal serum levels, breast milk intake rate, volume of distribution, and lactational transfer. Similar patterns are observed for all six PFAS at both the median and 90th percentile of the distribution [Supplementary Figure 3].

Peak serum concentrations for all six PFAS occur within the first 3 years of life [Figure 2]. After this peak, serum PFAS concentrations decline as the declining body weight-adjusted drinking water intake reduces exposure [Supplementary Figure 2]. Key trends in modeled lifetime serum PFAS concentrations, including peak concentrations in early life, have been observed in both epidemiological and TK modeling studies. Longitudinal epidemiological studies measuring early life serum concentrations show trajectories similar to the modeled lifetime trajectory, with PFOA, PFOS, PFNA, and PFDA generally increasing during the first 6-11 months followed by decreases at later timepoints[64,74]. For PFHxS, findings differ across studies: one reported increasing concentrations between birth and 6 months, followed by decreases[64], while another observed declining concentrations after birth[74].

Additional evidence indicates that early life serum concentrations of PFOA, PFOS, PFHxS, and PFNA are significantly higher than those measured in mid- and late childhood[75]. The early life increase observed for most PFAS corresponds to the nursing period[64,74], indicating that breastfeeding is an important exposure pathway in early life. Consistent with this interpretation, multiple studies have reported positive associations between breastfeeding duration and PFAS concentrations in infants and children, especially for PFOS and PFOA[64,74-78]. TK models similarly predict peak serum concentrations of PFOA, PFOS, and PFHxS within the first 1-2 years of life for both breastfed and formula-fed infants, with the latter peak resulting from high body weight-adjusted drinking water intake for young children[18,49]. In addition to these temporal trends, some studies report changes in population variability similar to the lifetime trajectory modeling. One longitudinal epidemiological study measuring serum PFOA, PFOS, PFHxS, and PFNA concentrations between ages 1-10.5 observed decreasing variability in the distribution of measured concentrations over time along with decreasing median concentrations[75]. Together, these findings support the ability of the population PFAS TK

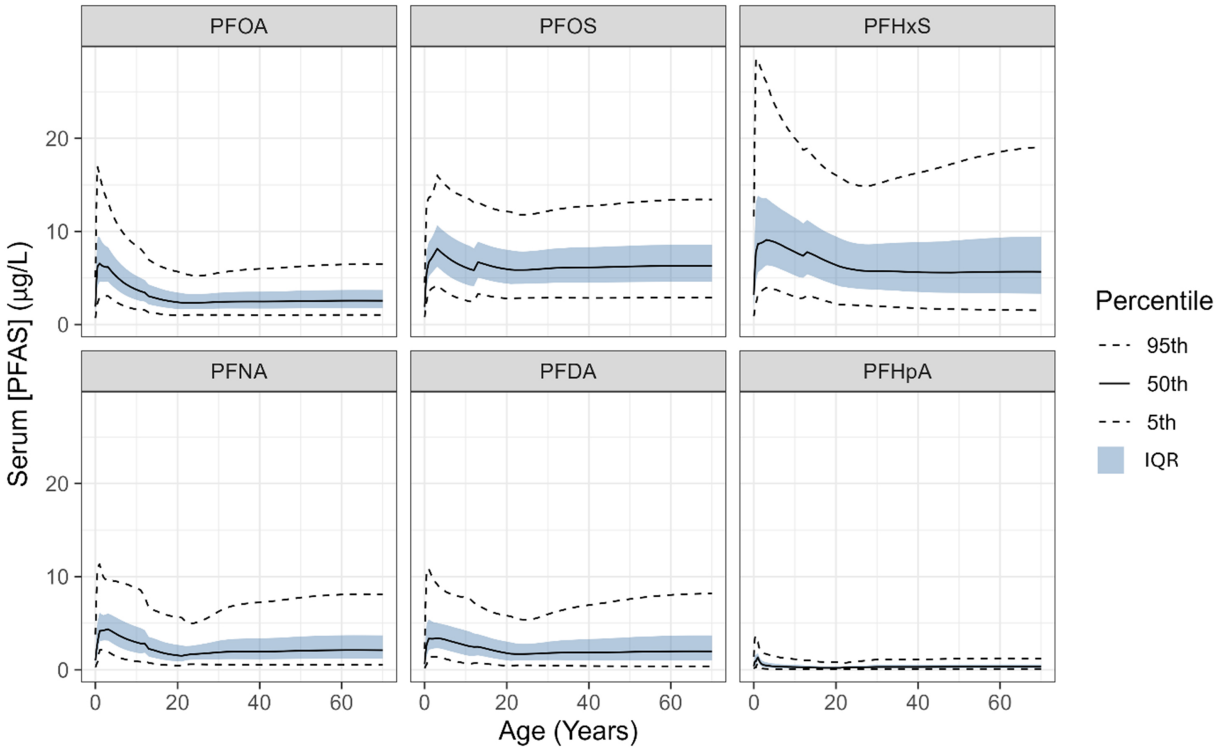

Figure 2. Serum PFAS concentrations modeled over time with constant exposure to 20 ng/L PFAS in drinking water. This modeled male population was breastfed for six months; maternal exposure is from a modeled population of 40-year-old women [Supplementary Table 6]. Lifetime drinking water exposure for both maternal and breastfed male populations was set at 20 ng/L PFAS in drinking water. PFAS: Per- and polyfluoroalkyl substances; PFOA: perfluorooctanoic acid; PFOS: perfluorooctanesulfonic acid; PFHxS: perfluorohexanesulfonic acid; PFNA: perfluorononanoic acid; PFDA: perfluorodecanoic acid; PFHpA: perfluoroheptanoic acid; IQR: interquartile range.

model to capture both temporal trends in serum PFAS concentrations over time and changes in population-level exposure factors during sensitive life stages.

Evaluating model performance

The population PFAS TK model performed well in predicting the distribution of population serum concentrations reported in empirical studies with paired drinking water and serum data for the six modeled PFAS, across exposure levels spanning 3-4 orders of magnitude [Figure 3]. Modeled central tendency and upper percentile serum PFAS concentrations mostly fell within two-fold error of the observed concentrations reported in each study. When results were combined for all PFAS and modeled study populations, RMSE values were similar for predictions of both central tendency and upper percentile values from the observed serum PFAS distributions [Figure 3A and B]. The comparable predictive performance at the center and upper percentiles of the distribution indicates the model captures population variability in TK and exposure factor parameters.

Less human TK data are available for PFNA, PFDA, and PFHpA, and data gaps for these chemicals were filled with a combination of data from animal studies, read-across from other PFAS, and imputation in the case of breastmilk elimination rate constants for PFNA and PFDA, as described in the methods. Overall, model error was not consistently higher for PFAS with more limited human TK data compared with data-rich PFAS. For example, imputed breast milk elimination rate constants for PFNA and PFDA, combined with less available data on breast milk transfer factors, did not result in consistently higher model prediction error for these PFAS. PFHpA, which has the least human TK information among the six modeled PFAS,

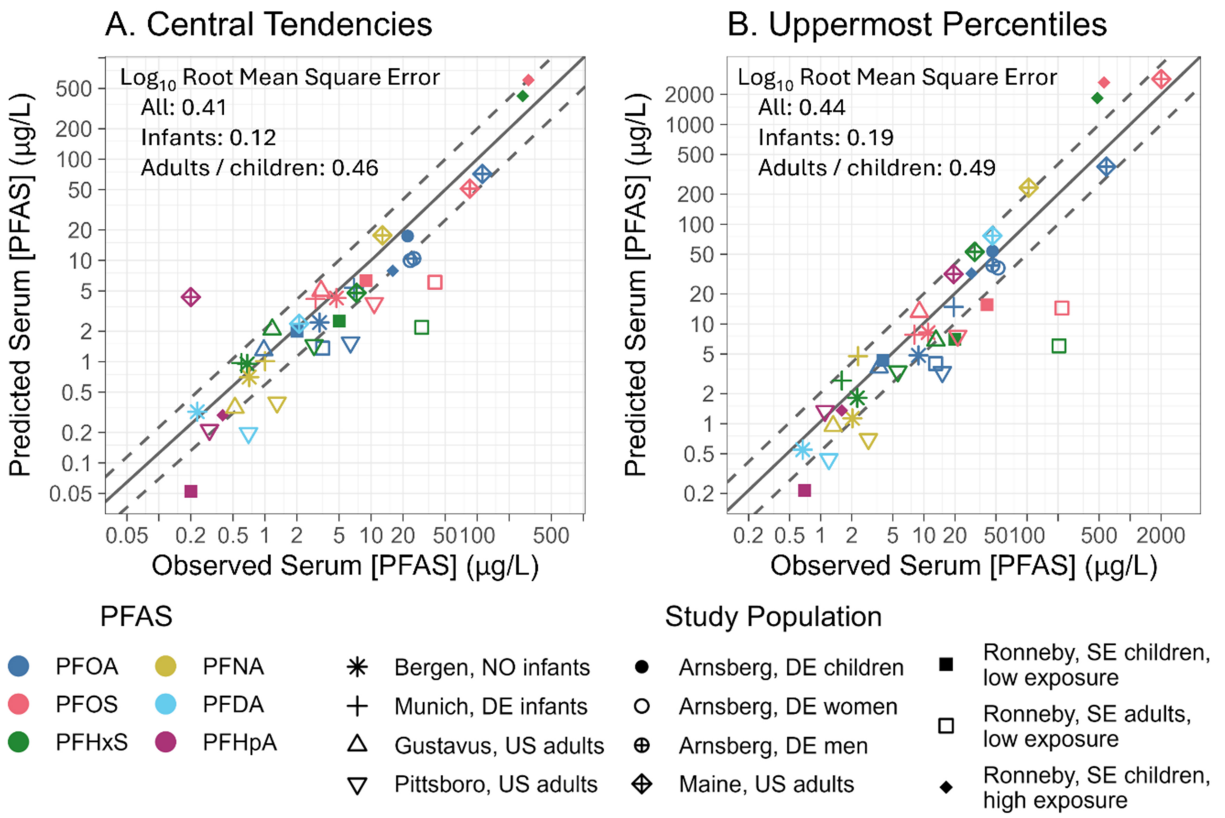

Figure 3. Goodness of fit model evaluation results for (A) central tendency values and (B) upper percentile values on log scale axes. Central tendency values are medians or GMs. Upper percentile values are 90th or 95th percentiles or study maxima. Modeling exposure scenarios are described in Supplementary Tables 7 and 8. The dashed line shows a two-fold difference from the solid line of equality. For points above the line of equality, the model overpredicts the empirical data. For points below the line of equality, the model underpredicts the empirical data. For each data point, the color corresponds to the PFAS and the shape corresponds to the Study Population. GMs: Geometric means; PFAS: per- and polyfluoroalkyl substances; PFOA: perfluorooctanoic acid; PFNA: perfluorononanoic acid; PFOS: perfluorooctanesulfonic acid; PFDA: perfluorodecanoic acid; PFHxS: perfluorohexanesulfonic acid; PFHpA: perfluoroheptanoic acid; NO: Norway; DE: Germany; US: United States of America; SE: Sweden.

showed the highest model error at central tendency values but comparable error to the data-rich PFAS at the upper percentile values [Supplementary Table 10]. These results suggest that the methods used to address PFAS-specific TK data gaps, especially for modeling infant exposure, can capture population-level TK for comparatively data-poor chemicals. However, serum PFAS distributions for compounds with less certain TK parameters are associated with greater modeling uncertainty because both the central estimates and variability of the input parameters are less well-characterized. In contrast, for PFAS with TK parameter distributions informed by human data, the resulting serum distribution is more representative of human variability and therefore associated with less uncertainty. Additional sources of uncertainty intrinsic to the model include the application of a single $T_{1/2}$ distribution for modeling all populations, including the single-sex populations from Arnsberg, DE, and uncertainty in exposure factors used for modeling.

Model error was lower for infant populations than for adults and older children across PFAS and at both the central tendency and upper percentiles of the distribution [Figure 3A and B, Supplementary Table 10]. For infant modeling scenarios, maternal serum PFAS concentrations are the exposure input that dictates the starting infant serum PFAS concentrations and the exposure via breast milk for exclusively breastfed infants. The availability of more complete exposure information and shorter modeled time periods for infant populations likely contributed to reduced model error.

Model accuracy depends on both an accurate reconstruction of the exposure scenario used for evaluation and the accuracy of the TK parameters and exposure factors used in the model. In addition to intrinsic model uncertainties discussed above, incomplete or inaccurately captured exposure information from sources including drinking water concentrations, exposure duration, and background exposure can contribute to discrepancies between predicted and observed concentrations.

For example, the model underpredicts empirical data from two study populations - the Ronneby, SE low exposure cohort and the Pittsboro, US cohort - at both the central tendencies and upper percentiles from the observed serum PFAS distributions. Uncertainty in reconstructing the exposure scenarios likely contributed to these discrepancies. For the Pittsboro, US cohort, lifetime drinking water exposure was modeled using constant PFAS concentrations reported in the study. However, PFAS concentrations in the Haw River, which serves as the drinking water source for the Pittsboro, US cohort, have varied substantially over time[62,79].

Similarly, for the adult population modeled from Ronneby, SE, the potential underestimation of exposure combined with the long modeling timeframe may have contributed to model underprediction. In Ronneby, one drinking water source was highly contaminated with PFAS (up to 8,000 ng/L for PFOS) while another source had exposure levels several orders of magnitude lower[35]. Although the evaluation cohort resided in areas served by the lower-exposure source, their proximity to areas supplied by the highly contaminated water source raises the possibility of unaccounted exposure through consumption of contaminated water or other exposure pathways not captured in the modeling scenario. Beyond the scenario-specific uncertainties addressed above, the use of 2017-2018 and 2013-2014 NHANES data for adults and children, respectively, for all modeling scenarios could contribute additional uncertainty, especially when modeling historical exposures. Importantly, the flexible model inputs that accommodate temporal and concentration variability over time in drinking water and background serum PFAS levels, among other inputs, allow for the reconstruction of complex historical exposures.

Relationship between modeled drinking water and serum concentrations

The population PFAS TK model was applied to estimate CGWC for the sensitive population of breastfed infants. Adult values were also calculated for comparison.

Figure 4 shows the relationship between increasing drinking water PFAS concentrations and the 90th percentile serum PFAS concentrations for six-month-old, exclusively breastfed infants. For this population, drinking water concentrations determine the distribution of maternal serum PFAS concentrations, which in turn define both the starting point for infant serum PFAS levels and PFAS concentrations in breast milk. For all six PFAS, increasing drinking water concentrations produced a linear increase in modeled serum concentrations.

The slope of the regression line at the 50th and 90th percentiles of the population serum distribution for infants and adults derived using this model compared with literature values are shown in Table 2. These slopes are the change in serum PFAS concentration (μg/L) per one ng/L increase in drinking water PFAS concentration. Comparing model-derived slopes for the six modeled PFAS, differences in slope across chemicals highlight the importance of elimination half-life for the relationship between drinking water exposure and serum levels in breastfed infants: PFHxS with the longest half-life has a large regression coefficient while PFHpA with the shortest half-life has the smallest.

The relationship between drinking water and serum PFAS concentrations has been investigated using a variety of methods in empirical studies. Regression analyses using either measured data[33,80,81] or modeled data[82] to relate drinking water and serum PFAS concentrations can control for potentially confounding

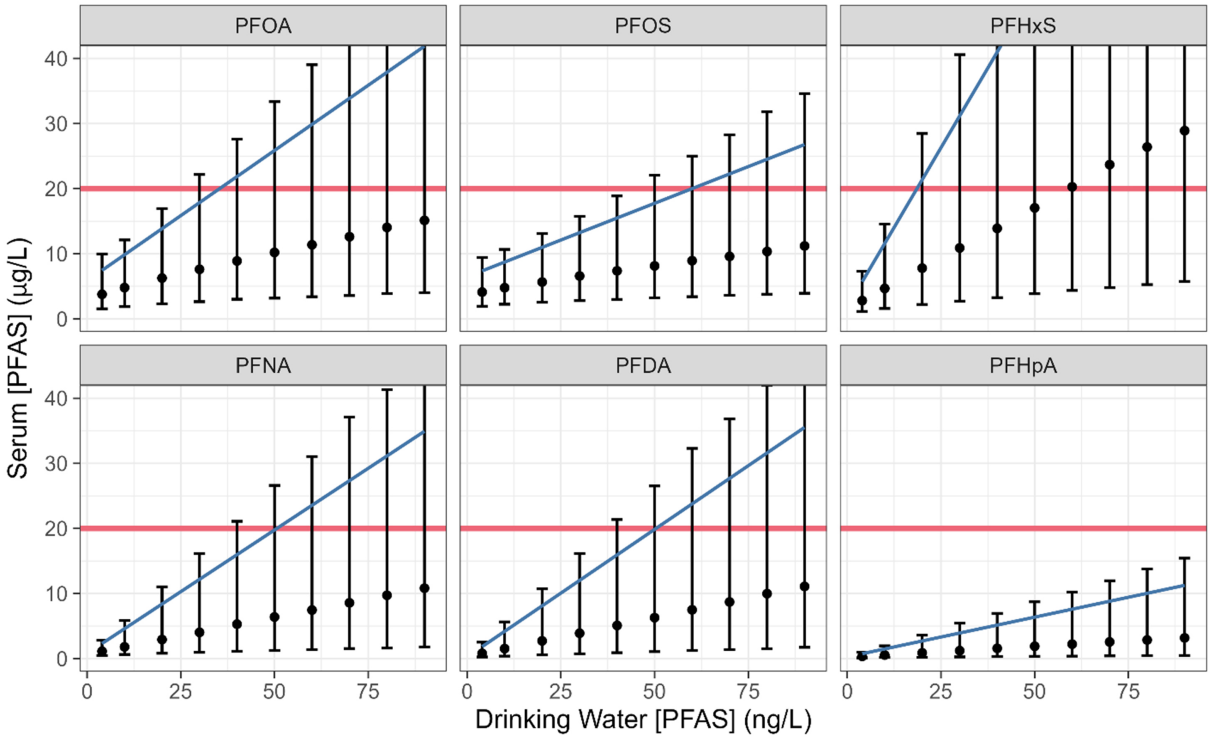

Figure 4. Model-predicted serum concentrations for 6-month-old, exclusively breastfed infants for a suite of PFAS at varying maternal drinking water exposure levels. The stated drinking water concentration determines maternal serum PFAS concentrations and breast milk concentrations. Vertical black lines are the modeled distribution of serum PFAS concentrations at each drinking water exposure level, with the median shown as a point and the horizontal bars showing the 5th and 95th percentiles. The blue regression line depicts the linear relationship at the 90th percentile of predicted serum values. The red horizontal line shows the NASEM 20 ng/mL clinical action level. PFAS: Per- and polyfluoroalkyl substances; NASEM: National Academies of Sciences, Engineering, and Medicine; PFOA: perfluorooctanoic acid; PFOS: perfluorooctanesulfonic acid; PFHxS: perfluorohexanesulfonic acid; PFNA: perfluorononanoic acid; PFDA: perfluorodecanoic acid; PFHpA: perfluoroheptanoic acid.

Table 2. Modeled slope for the relationship between drinking water and population serum PFAS levels compared with literature values

PFAS	Modeled regression coefficients (slope)				Literature regression coefficients or serum:water ratios[a]			
	Median (infant)	Median (adult male)	90th Percentile (infant)	90th Percentile (adult male)	Minimum	Median	Maximum	Number of studies (number of values)[b]
PFOA	0.13	0.048	0.40	0.14	0.03	0.10	0.25	10 (22)
PFOS	0.081	0.073	0.23	0.19	0.03	0.10	0.35	3 (9)
PFHxS	0.31	0.20	0.98	0.53	0.05	0.12	0.52	3 (8)
PFHpA	0.033	0.011	0.12	0.039	0.001	0.005	0.035	3 (8)
PFNA[c]	0.11	0.077	0.38	0.25	-	-	-	1
PFDA[c]	0.12	0.081	0.39	0.25	-	-	-	1

[a]Literature sources: Hoffman *et al.* (2011)[33], Post (2021)[39], Johanson *et al.* (2023)[80], Zhang *et al.* (2019)[81], Bogdan *et al.* (2023)[82], McDonough *et al.* (2021)[83], Lewis-Michl *et al.* (2025)[84], Xu *et al.* (2020)[85], Emmett *et al.* (2006)[86], Post *et al.* (2009)[87].
[b]Some studies report more than one regression coefficient or serum: water ratio for each PFAS.
[c]Single literature regression coefficients for PFNA and PFDA (0.038 and 0.005, respectively) are available but are uncertain due to differences in study methodology[80].
PFAS: Per- and polyfluoroalkyl substances; PFOA: perfluorooctanoic acid; PFOS: perfluorooctanesulfonic acid; PFHxS: perfluorohexanesulfonic acid; PFHpA: perfluoroheptanoic acid; PFNA: perfluorononanoic acid; PFDA: perfluorodecanoic acid.

variables such as age and sex. Another approach is to calculate the serum-to-water ratio from empirical data[83-86], or similar relationships predicted with TK modeling[39,87] enabling examination of the distribution of

Table 3. Clinical Guidance-based Water Concentrations (CGWCs) for six PFAS

PFAS	CGWC (ng/L, equivalent to ppt)[a]	
	Age 6 months[b]	Age 40 years[b]
PFOA	35	125
PFOS	60	67
PFHxS	19	33
PFNA	51	77
PFDA	50	78
PFHpA	162	515

[a]CGWC is the drinking water PFAS concentration predicted to result in serum PFAS concentrations that reach 20 ng/mL at the 90th percentile of the modeled population distribution. This is a concentration associated with increased risk of adverse health effects sufficient to warrant enhanced clinical action, not a health-protective drinking water value.
[b]PFAS contribution from maternal serum is from the distribution of modeled 40-year-old women. CGWC values are predicted for male populations.
CGWCs: Clinical Guidance-based Water Concentrations; PFAS: per- and polyfluoroalkyl substances; PFOA: perfluorooctanoic acid; PFOS: perfluorooctanesulfonic acid; PFHxS: perfluorohexanesulfonic acid; PFNA: perfluorononanoic acid; PFDA: perfluorodecanoic acid; PFHpA: perfluoroheptanoic acid.

serum to water ratios across a study population. Both regression analysis and serum-to-water ratios yielded similar interpretations, showing an estimated increase in serum PFAS concentrations for each unit increase in drinking water PFAS concentration, providing some comparability to the slope derived from the regression analysis from the TK modeling in this work.

The median and maximum of available literature regression coefficients for PFOA, PFOS, PFHxS, and PFHpA were within 2-fold of the regression coefficients at the median and 90th percentile modeled for adult male populations in this study [Table 2]. For the regression coefficients in infant populations, regression coefficients at the median and 90th percentile were similarly within 2-fold of literature-reported values for PFOA and PFOS, and were larger than literature-reported values for PFHxS and PFHpA. The single available literature regression coefficients for PFNA and PFDA may be biased by methods to impute data points below limits of detection[80]; regression coefficients at the median of the adult male population modeled for this study were higher for both chemicals. Despite the methodological variability in literature-based values, modeled regression coefficients from this analysis were overall similar to those reported in the literature for multiple PFAS. While literature approaches provide useful empirical summaries of the relationship between drinking water and serum concentrations, they do not explicitly represent the combined effects of life-stage-specific exposure pathways and interindividual variability in TK parameters. By contrast, the population PFAS TK model framework used here integrates these factors to estimate how drinking water exposure translates into population distributions of serum PFAS concentrations over time.

Predicting CGWCs for PFAS

The linear regression equation from the relationship between drinking water and serum PFAS concentrations at the 90th percentile of the distribution was used to calculate the CGWC for breastfed infants and adults. The CGWC is defined as the drinking water concentration for each PFAS predicted to result in 90th percentile serum concentrations reaching the NASEM clinical action level of 20 ng/mL.

Table 3 shows the PFAS CGWCs calculated for each PFAS for breastfed infants and adults. CGWCs are lower for breastfed infants than adults for all PFAS. For both populations, PFHxS has the lowest CGWC and PFHpA has the highest CGWC, underscoring the importance of chemical half-life in determining the relationship between drinking water exposure and serum PFAS levels.

For comparison, we entered the PFOA CGWC into a recently revised, transgenerational PFOA TK model that takes deterministic TK parameters and exposure factors to calculate a reasonable maximum exposure scenario with a combination of central tendency and upper percentile values[49]. The projected serum concentrations at the CGWC under parallel exposure scenarios for infants and adults were 22.5 and 19.5 ng/mL, respectively, showing good agreement with our model results. The slightly higher infant serum concentration likely stems from the use of a higher LTF and lower volume of distribution used by Greene *et al.* (2024)[49].

The CGWC for each PFAS in Table 3 could be used in Equation (4) to evaluate a mixture of PFAS concentrations in drinking water compared to the NASEM clinical action level of 20 ng/mL. CGWCs are not health-protective drinking water values but rather concentrations associated with elevated health risk sufficient to prompt clinical follow-up. The CGWC could be used as a screening tool for clinicians and regulatory agencies to screen for exposure scenarios where enhanced clinical action, health monitoring, and/or exposure interventions are warranted.

CONCLUSION

This population PFAS TK model provides a flexible tool to predict the relationship between drinking water and population serum levels for six PFAS. The model performed well in predicting the distribution of population serum concentrations reported in empirical studies with paired drinking water and serum data for the six modeled PFAS across 3-4 orders of magnitude of exposure. Potential model applications include reconstructing historical exposures, estimating the impact of drinking water regulations on human exposure and clinical metrics, assessing exposure interventions, and as a tool to support policy, regulatory, and clinical decision-making. By integrating population TK modeling with clinical guidance levels for PFAS in serum, this framework can help identify population exposure scenarios in which enhanced clinical action may be warranted. More broadly, this approach provides a quantitative link between drinking water - an important source of PFAS exposure - and clinical guidance, supporting more integrated evaluations of PFAS risks across environmental, regulatory, and public health contexts.

DECLARATIONS

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Authors' contributions

Made substantial contributions to conception and design of the revised model, model implementation, and interpretation: Nielsen, G.; Spady, E. S.; Moody, N. S.; Baird, S. J. S.; Heiger-Bernays, W.; Smith, C. M.
Drafted manuscript: Nielsen, G.
Developed model code: Spady, E. S.
All authors approved the final manuscript. Opinions expressed herein are those of the authors and do not represent the official position of the Massachusetts Department of Environmental Protection.

Availability of data and materials

Supporting information is available in the Supplementary Materials. The full model R code, ReadMe documentation, and model demo files are available as a zip file. User-defined inputs and model results underlying the results and conclusions in this paper are available upon reasonable request from the corresponding author.

AI and AI-assisted tools statement

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Conflicts of interest

All authors declared that there are no conflicts of interest.

Ethical approval and consent to participate

Not applicable.

Consent for publication

Not applicable.

Supplementary Materials

Supplementary Materials

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