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Dietary Accumulation of PFOS, PFOA and GenX in Fish

Dietary Uptake and Depuration Kinetics of PFOS, PFOA and GenX in a Benthic Fish

Kathryn L. Hassell^{1,2*}, Timothy L. Coggan³, Tom Cresswell⁴, Adam Kolobaric³, Kathryn Berry³, Nicholas D. Crosbie⁵, Judy Blackbeard⁵, Vincent J. Pettigrove^{1,2} and Bradley O. Clarke³.

¹ Centre for Aquatic Pollution Identification and Management (CAPIM), The University of Melbourne, Parkville, Victoria, Australia 3010

² Aquatic Environmental Stress Research Group (AQUEST), School of Science, RMIT University, GPO Box 2476, Melbourne, Victoria, Australia 3001.

³ Centre for Environmental Sustainability and Remediation (EnSuRe), School of Science, RMIT University, GPO Box 2476, Melbourne, Victoria 3001, Australia.

⁴ Institute for Environmental Research, ANSTO, Locked Bag 2001 Kirrawee, New South Wales 2232, Australia.

⁵ Melbourne Water Corporation, 990 La Trobe St, Docklands, Victoria 3008 Australia
(Submitted 13 June 2019, Returned for Revisions 21 October 2019, Accepted 19 November 2019)

Abstract: Per- and poly-fluoroalkyl substances (PFAS) are ubiquitously distributed throughout aquatic environments and can bioaccumulate in organisms. Here we examined dietary uptake and depuration of a mixture of three PFAS: *perfluorooctanoic acid* (PFOA; C₈HF₁₅O₂) *perfluorooctane sulfonate* (PFOS; C₈HF₁₇SO₃) and GenX (trade-name; HPFO-DA; C₆HF₁₁O₃). Benthic fish (blue spot gobies, *Pseudogobius sp.*) were fed contaminated food (nominal dose 500 ng g⁻¹) daily for a 21-day uptake, followed by a 42-day depuration period. PFOA, L-PFOS (linear PFOS) and total PFOS (sum of linear and branched PFOS) were detected in freeze-dried fish, whilst GenX was not detected in any, indicating either a lack of uptake, rapid elimination (<24 hours).. Depuration rates (d⁻¹) were 0.150 (PFOA), 0.045 (L-PFOS) and 0.042 (L+Br-PFOS) with corresponding biological half-lives of 5.9, 15 and 16 days, respectively. PFOS isomers were eliminated differently, resulting in enrichment of linear PFOS (70-90%) throughout the depuration period. This is the first reported study of GenX dietary bioaccumulation potential in fish,

This is the author manuscript accepted for publication and undergone full peer review but has not been through the copyediting, typesetting, pagination and proofreading process, which may lead to differences between this version and the [Version of Record](#). Please cite this article as [doi: 10.1002/etc.4640](https://doi.org/10.1002/etc.4640).

and the first dietary study to investigate uptake and depuration of multiple PFAS simultaneously, allowing us to determine that whilst PFOA and PFOS accumulated as expected, GenX, administered in the same way, did not appear to bioaccumulate.

Graphical Abstract

Individually-housed fish were exposed to a mixture of PFAS via contaminated food for a 21-day uptake period, followed by a 42-day depuration period. At regular intervals fish were removed and freeze-dried whole body tissue was analysed.

Keywords: PFAS, HPFO-DA, GenX, perfluoroalkyl substance, bioaccumulation, isomeric differences

This article includes online-only Supplemental Data.

*Address correspondence to kathryn.hassell@rmit.edu.au

Published online XXXX 2019 in Wiley Online Library (www.wileyonlinelibrary.com).

DOI: 10.1002/etc.xxxx

Introduction

Per- and poly-fluoroalkyl substances (PFASs) are ubiquitously distributed throughout aquatic environments and bioaccumulation in some aquatic biota is well-established (Sedlak et al. 2017). However, much less is known about the mechanisms of accumulation and depuration (Houde et al. 2006, Xiao 2017) and the relative contribution to body burden from dietary (digestive) and aqueous (gills, dermal) exposure pathways is still to be determined (Houde et al. 2006, Xiao 2017).

PFASs are a large group of over 4700 man-made chemicals that have been used for industrial and domestic purposes since the 1950s. The carbon-fluorine bond imparts unique chemical and physical properties, enabling excellent thermal and chemical stability, and PFASs have become an important chemical class for modern society (Xiao 2017). Perfluorooctane sulfonate (PFOS: $C_8HF_{17}SO_3$) and perfluorooctanoic acid (PFOA: $C_8HF_{15}O_2$) were the main PFASs produced and continue to be detected in environmental matrices, typically with greater frequency and higher concentrations than other PFASs (Xiao 2017). The ubiquitous presence of PFOS and PFOA in biological samples throughout the world, combined with their toxicity has resulted in recent regulatory efforts to restrict their manufacturer and use, and replace them with less toxic and less persistent alternatives (Giesy and Kannan 2001).

Hexafluoropropylene oxide dimer acid (HPFO-DA: $C_6HF_{11}O_3$; trade name: GenX) has been in production since 2010, primarily as a replacement to PFOA (Sun et al. 2016). Concern over this chemical was raised when it was detected in drinking water at concentrations up to 4500 ng L^{-1} near a fluorochemical production facility in North Carolina, USA (Sun et al. 2016). Subsequently, GenX has been detected in surface waters from other industrialised areas such as the Xiaoqing river in China (3825 ng L^{-1}) and the Lower Rhine river in Germany (86.1 ng L^{-1}) and Netherlands (73.1 ng L^{-1}) (Heydebreck et al. 2015). To date there are only a few studies that have detected the compound in environmental samples and the extent of its contamination in the environment is largely unknown (Xiao 2017). The toxicity of GenX was recently tested in algae, invertebrates and fish, and in both the ammonium salt and acid form, acute ($>100\text{ mg L}^{-1}$) and chronic toxicity ($<33\text{ mg L}^{-1}$) was low (Hoke et al. 2015). In the same study, a 28-day bioconcentration test with common carp showed no bioconcentration following aqueous exposure to 0.198 mg L^{-1} . However, to date there is no information available regarding dietary bioaccumulation potential, including uptake and elimination behaviour in aquatic biota. In rats and mice, GenX was completely absorbed through orally administered doses, then rapidly eliminated in urine with no evidence of metabolism (Gannon et al. 2016). Given the potential for this chemical to be ubiquitously present in the environment, more information regarding uptake and elimination pathways in aquatic biota is needed.

PFAS are considered potentially harmful because they are hepatotoxic and capable of inducing liver tumours (Lau et al. 2007, Seacat et al. 2003), and have been shown to cause immunotoxicity (DeWitt et al. 2011) and changes in lipid metabolism (Colli-Dula et al. 2016, Hu et al. 2005, Oakes et al. 2005, Seacat et al. 2003). In fish, adverse effects to the reproductive axis and multi-generational effects including increased deformity and mortality in embryos, increased vitellogenin expression and histological alterations to liver, thyroid and gonads have been observed at PFOS concentrations of $10\text{--}300\text{ }\mu\text{g/L}$ (Du et al. 2009, Keiter et al. 2012). The specific mechanism(s) responsible for PFAS toxic action are not well characterised, and current consensus is that it is not due to effects on a single receptor (such as the aryl-hydrocarbon receptor for dioxin toxicity), but rather that multiple receptors such as the peroxisome proliferator-activated receptor- α (PPAR α) and constitutive androstane receptor (CAR), amongst others, may be involved (Carr et al. 2013, Peters and Gonzalez 2011). In fish, studies have reported alterations in gene expression predominantly related to steroidogenesis (Ankley et al. 2005, Colli-Dula et al. 2016). Thus, there appear to be multiple mechanisms of toxicity and potential species differences, and it is these complexities that contribute to the difficulties in understanding and managing PFAS contamination in environmental and human health settings. For these reasons, there is a need for more research effort worldwide.

The bioaccumulation of PFAS in fish has been reported in a range of marine and freshwater species from around the world (Giesy and Kannan 2001, Houde et al. 2006, Lau et al. 2007). PFAS differ from 'traditional' hydrophobic, persistent and bioaccumulative toxicants (PBTs) since they tend to associate with protein-rich tissues (liver and blood serum) (Jones et al. 2003) rather than lipid. Chain length is an important

determinant of bioaccumulative potential, and perfluoroalkyl sulfonic acids (PFASs) are more bioaccumulative than perfluoroalkyl carboxylic acids (PFCAs) of the same chain length (Hong et al. 2015). PFOS is usually the major PFAS detected in aquatic species, and the highest concentrations reported ($>3000 \text{ ng g}^{-1} \text{ ww}$) have been measured in predatory, bottom-dwelling species (Houde et al. 2006). Bioaccumulation in organisms is influenced by factors such as habitat, food source (prey), metabolism and dominant exposure pathway (Hong et al. 2015). PFAS compound profiles in benthic fish have been shown to be similar to the PFAS compound profiles of surrounding sediments (Lescord et al. 2015, Sedlak et al. 2017, Thompson et al. 2011) and it is likely that PFAS-contaminated sediments are an important source of exposure (relative to diet and dissolved phase), either directly, or indirectly through contamination of sediment-dwelling prey organisms (Lescord et al. 2015, Martin et al. 2004).

Laboratory studies with fish indicate species-specific (thought to be related to species-specific physiology and ecology) and sex-specific (thought to be related to sex-steroid dynamics) differences in uptake rates, total uptake (maximum tissue concentrations) and elimination rates of PFAS (Lee and Schultz 2010, Martin et al. 2003a). Furthermore, differences in tissue distribution, total body burden and elimination rates also occur depending on the predominant route of absorption into the bloodstream (i.e. via the gills (aqueous) or via the gut wall (dietary)) (Falk et al. 2015, Goeritz et al. 2013, Martin et al. 2003a, b). For example, PFOS biological half-lives (whole fish) in dietary studies are in the range of 11-17 days (Falk et al. 2015, Goeritz et al. 2013, Martin et al. 2003b) whilst in aqueous exposure studies the values are 29-35 days (Fang et al. 2016, Sakurai et al. 2013). Fish that are exposed via the dietary route tend to have highest PFAS concentrations in the liver, followed by blood and kidney (Falk et al. 2015, Goeritz et al. 2013), whereas fish that are exposed via the aqueous route tend to have highest concentrations in blood, then kidney and liver (Martin et al. 2003a).

Whilst PFAS do bioaccumulate in fish (Akerblom et al. 2017, Falk et al. 2015, Martin et al. 2003b), biomagnification through food webs to higher trophic levels is not always observed (Lescord et al. 2015, Loi et al. 2011) and, unlike other PBTs, there are no strong correlations between age or size with total body burdens (Akerblom et al. 2017, Lescord et al. 2015). When food webs include groups such as birds and marine mammals, PFAS biomagnification is observed (Kelly et al. 2009, Loi et al. 2011), whereas in food webs that only include invertebrates and fish, PFAS biomagnification is not always observed (Lescord et al. 2015, Martin et al. 2004). This is thought to be due to reduced capacity of air breathing organisms to eliminate PFAS via the lungs as compared to aquatic organisms that eliminate PFAS via the gills (Kelly et al. 2009). The partitioning and sorption characteristics of PFOS and PFOA have also been shown to be affected by organic content and salinity, indicating that benthic food webs in estuarine environments may be at increased risk of exposure (relative to freshwater environments) (Hong et al. 2015). Yet, most laboratory-derived data on PFAS accumulation kinetics in fish is based on freshwater model species such as zebrafish, carp and rainbow trout (Fang et al. 2016, Keiter et al. 2012, Martin et al. 2003b). To address this knowledge gap, we have focused

on examining PFAS bioaccumulation in a benthic, sediment-associated estuarine fish species.

We hypothesise that benthic fishes that are associated with sediments in estuarine environments may be at increased risk of exposure due to the above-mentioned differences in partitioning and sorption characteristics of PFASs in these environments. Hence, the aims of the present study were to determine the uptake and depuration rates of a mixture of PFAS compounds (PFOS, PFOA and for the first time GenX) in a small benthic fish (*Pseudogobius sp.*) following dietary exposure.

MATERIALS AND METHODS

Experimental Design

Adult blue spot gobies (*Pseudogobius sp.*) were captured from the Werribee River (37° 56' 31.6752" S; 144° 40' 13.638" E) between 29th Dec 2016 and 17th Jan 2017 and transferred to the marine laboratory at the Victorian Marine Science Consortium (VMSC), Queenscliff. There, fish were housed individually in purpose-built cages (37 x 10 x 9.6 cm³) contained within 80 L tanks (Figure S1). Housing fish individually enabled accurate measurements of growth and daily feeding rates, which also enabled accurate calculations of the PFAS dose in each fish. Forty-eight fish were allocated to each of the control and PFAS treatment (exposed) groups, and the numbers of each sex were based on what was caught during the sampling. Male and female fish were distributed evenly between control and treatment groups, resulting in each group containing 40 female and 8 male fish. Each tank received a continuous flow of filtered (0.45 µm) seawater that was sourced from Port Phillip Bay (salinity 36 parts per thousand; pH 8.02; dissolved oxygen >80% S; temperature 16-19°C).

The experiment lasted a total of 11 weeks, which included a 14 day acclimation period, 21 day uptake period and 42 day depuration period. Fish were fed a control or PFAS-contaminated diet for 21 days (uptake period), then fed the control diet for a further 42 days (depuration period). This exposure regime was based on other PFAS dietary accumulation studies (Falk et al. 2015, Martin et al. 2003b) and relevant guidance documents (OECD Guidelines #305, Bioaccumulation in Fish: Aqueous and Dietary Exposure) (OECD 2012).

Four fish were sampled from each treatment group (prior to feeding) on Day 0, 4, 7, 14, 21, 28, 35, 42, 49, 56 and 63 for chemical analysis. The fish were killed prior to feeding to ensure 24 h digestion of previously consumed food. Each fish was weighed (wet weight; ww) and measured, killed, rinsed with seawater, blotted dry and then the digestive tract was removed. Due to the small size of this fish species, individual organs could not be dissected so whole body samples were used for analysis instead. Frozen fish (whole body) were freeze dried using a VirTis Benchtop SLC freeze dryer and stored frozen (-20°C) prior to chemical analysis.

Chemical Standards

Technical mixtures of branched plus linear PFOS (97% purity, as potassium salt, ~ 70% linear PFOS; Sigma-Aldrich, Australia), PFOA (99% purity, linear only; Sigma-Aldrich, Australia) and GenX were dissolved in methanol to create 100 $\mu\text{g mL}^{-1}$ stock solutions. These were used to generate a 25 $\mu\text{g mL}^{-1}$ PFAS-stock solution (L+Br PFOS, PFOA and GenX) for food spiking that was prepared by combining PFOS, PFOA and GenX stock solutions at a ratio of 1:1:2, respectively. Analytical standards and isotopically labelled analogues of linear PFOS, PFOA, GenX and 14 other PFAS were purchased from Wellington Laboratories (Ontario, Canada) as solutions of 50 $\mu\text{g mL}^{-1}$ (>98% purity) in methanol. Methanol (LC-MS grade, Honeywell, USA), ultrapure water (>18 M $\Omega\cdot\text{cm}$, Merck Millipore, Australia), ammonium hydroxide solution (28% in H₂O, $\geq 99.99\%$), sodium acetate, glacial acetic acid and ammonium acetate ($\geq 99.99\%$; Sigma-Aldrich, Australia) were used for analysis and sample processing.

Food Preparation

PFAS-spiked fish food was prepared by mixing 10 g brine shrimp (purchased from Aqua One, Australia) with molten agar (Agar, bacteriological grade, Ajax) and 200 μL of 25 $\mu\text{g mL}^{-1}$ PFAS-stock solution (methanol carrier solvent) to yield a target concentration of 500 ng g^{-1} for each compound (PFOA, L+Br PFOS, GenX). This target concentration was selected to enable comparison with other PFAS dietary accumulation studies [27, 28]. Fresh batches of food were prepared each week to minimise the risk of microbial contamination and spoilage. Control food was spiked with 200 μL of carrier solvent (methanol) only. Preliminary testing indicated that PFAS did leach from the food into the surrounding water if left for prolonged periods, and therefore food was offered daily for 5 min only, then all uneaten food and faeces was removed and a complete (100%) water change was performed. Water was tested at each sampling point, and all measurements of PFOS, PFOA and GenX were below the limit of quantification (LOQ) (PFOS, PFOA: 4 ng L^{-1} ; GenX: 8 ng L^{-1}). The amount of food removed from each exposure cage was recorded (by measurement of wet mass) to enable accurate estimations of daily food ration and calculations of the total dose eaten by each fish throughout the course of the experiment.

Chemical Analysis

Extraction from fish and fish food samples.

All samples were extracted using the same method. Freeze dried whole fish or fish food samples were spiked with 250 μL of a mixture of isotopically labelled PFAS standards (100 ng mL^{-1} PFOS and PFOA, 200 ng mL^{-1} GenX in methanol), and homogenized in a 50 mL polypropylene centrifuge tube. A 4.9 mL aliquot of 10 mM sodium hydroxide in methanol was added to each tube then sonicated and shaken for 12 hrs. Extracts were then neutralised with 100 μL of glacial acetic acid, cooled on ice then subjected to

dispersive solid phase extraction (d-SPE) with 50 mg of primary secondary amine (PSA) and 100 mg C18 followed by centrifugation (10,000 rpm, 10°C, 10 min). Centrifuged extracts were then filtered with a 0.45 µm PES syringe filter (pre-rinsed with methanol) into a polypropylene chromatography vial. The fish extraction method was validated using homogenised whole bait fish spiked at two levels (low (n=6) and high (n=6)) for PFOA, PFOS and GenX (absolute recoveries of 80-97%, 76-107% and 60-113%, respectively). The limit of reporting (LOR) was set as the LOQ for the fish samples, which were: PFOA = 0.3 ng g⁻¹, L-PFOS = 0.6 ng/g, L+Br-PFOS = 0.6 ng g⁻¹ and GenX 1.0 ng g⁻¹. For data analysis, values below the LOQ were substituted with the half LOR value for each compound, except for GenX, which was excluded from analysis since all fish samples were below detection (Shoemaker and Tettenhorst 2018).

Extraction from water samples.

Quantification of PFAS in water samples was performed for 17 PFASs: PFPeA, PFBS, PFHxA, PFPeS, PFHpA, PFHxS, 6:2 FTS, PFOA, PFHpS, PFNA, PFOS, 8:2 FTS, PFDA, PFDS, PFUDA, PFDoA and GenX (Table S1) using a modified version of US EPA method 537.1 (Shoemaker and Tettenhorst 2018). 250 mL water samples were filtered using glass fibre filters (1 µm, Whatman, England), spiked with 5 ng of isotopically-labelled ¹³C PFAS standards before extraction using weak anion exchange cartridges (Oasis WAX, 6 CC, 150 mg, 30 µm, Waters, Australia) that had been pre-conditioned with 4 mL 0.1% (v/v) ammonium hydroxide in methanol, 4 mL methanol, and 4 mL ultrapure water. Following sample loading, the cartridges were washed with 4 mL of a pH 4 buffer (sodium acetate/acetic acid), then eluted with 2 mL methanol that had been used to rinse the corresponding sample bottle, then 4 mL of 0.1% (v/v) ammonium hydroxide in methanol. Eluents were evaporated to 500 µL under a gentle stream of nitrogen (TurboVap LV, Biotage, Sweden) at 30°C and reconstituted to 1000 µL in 50/50 MeOH/ultra-pure water before analysis. The limit of reporting in water samples was set as the lowest validated water method extraction (MQL): 4 ng L⁻¹ for all analytes except GenX 8 ng L⁻¹.

Instrumental Analysis.

Analysis was performed on an Agilent Technologies 1290 infinity II liquid chromatograph (LC) coupled with an Agilent technologies 6495B tandem mass spectrometer (MS/MS) in negative electrospray ionisation mode (ESI) (MS/MS parameters (Table S2). Separation was achieved on a Zorbax eclipse plus RRHD C18 column (3.0 x 50 mm, 1.8 µm, Agilent Technologies, USA) with an injection volume of 2 µL. Gradient elution was performed using 5 mM ammonium acetate in ultrapure water (A) and methanol (B) at 400 µL min⁻¹ and the first 1.5 min was diverted to waste (t₀ = 10% B; t_{0.5} = 10% B; t_{2.5} = 55% B; t₉ = 90% B; t_{9.5} = 100% B; t_{11.5} = 100% B; t_{11.6} = 10% B; t₁₄ = 10% B). Due to low recoveries at high sheath gas temperatures, analysis of GenX was performed using a second method with separate MS parameters, a 10 µL injection volume, the same column for separation, a shorted elution gradient and no delay column

at solvent mixer (MS/MS parameters listed in Table S3). Elution gradient was performed using 5 mM ammonium acetate in ultrapure water (A) and methanol (B) at $400 \mu\text{L min}^{-1}$ and the first minute was diverted to waste ($t_0 = 5\% \text{ B}$; $t_{0.5} = 5\% \text{ B}$; $t_2 = 55\% \text{ B}$; $t_{2.25} = 100\%$; $t_{3.25} = 100\% \text{ B}$; $t_{3.5} = 5\% \text{ B}$; $t_{4.5} = 5\% \text{ B}$).

A 1000 ng mL^{-1} stock of linear native PFAS compounds was prepared. Dilutions of this stock were prepared for each batch to create a linear calibration curve to 8 levels ($r^2 > 0.99$) in 50/50 methanol/Ultra-pure water and containing 5 ng mL^{-1} of surrogate PFAS to match sample extracts. For PFAS with branched and linear isomers the combined peak area was quantified using linear analogues and reported as sum branched and linear of that compound. For PFOS the linear only peak area was also quantified and reported for data analysis.

Quality Control/Quality Assurance (QA/QC).

Each batch of 24 water samples consisted of two method blanks (ultrapure water), a matrix blank (sterilised seawater) and a lab control sample (LCS, spiked with 1 ng ($n=3$) or 5 ng ($n=2$) of native PFAS mixture). Blanks were free of contamination and LCS recoveries were generally within 70-120% (Table S1). For fish extraction each batch of 20 samples included a method blank (using acid washed sand), an LCS (acid washed sand spiked with PFOA, PFOS and GenX) and a matrix spike (homogenised whole baitfish spiked with PFOA, PFOS and GenX).

Data Analysis.

Data were analysed as per methods outlined in the Organisation for Economic Co-operation and Development (OECD) Guidelines document #305 (Bioaccumulation in Fish: Aqueous and Dietary Exposure) (OECD 2012). All statistical analyses were performed using JMP 12.0.1 (SAS, 2017), and where necessary, data were log-transformed to improve homogeneity of variance and normality.

Growth rates (g/day) were calculated based on the initial (t_1) and final (t_2) weights of each individual fish, as per the following equation:

$$\text{Growth rate: } [\ln(Wt_2) - \ln(Wt_1)] / (t_2 - t_1) \times 100 \quad (\text{Equation 1})$$

Linear regression was used to determine depuration (loss) rate constants (k_d), following the first-order elimination equation:

$$\text{Depuration rate constant: } \ln(C_{(\text{fish})}) = k_d t + a \quad (\text{Equation 2})$$

where $C_{(\text{fish})}$ is the PFAS concentration in fish ($\text{ng g}^{-1} \text{ ww}$), k_d is the depuration rate constant, t is time (days) and a is a constant. The depuration rate constant was then used to calculate the biological half-life for each PFAS compound, as per the following equation:

$$\text{Biological half-life: } t_{1/2} = \ln(2) / k_d \quad (\text{Equation 3})$$

The assimilation efficiency (α), as a measure of the absorption of PFAS across the gut, was calculated using the following equation:

$$\text{Assimilation efficiency: } \alpha = [(C_{0,d} \times k_d) / I \times C_{(\text{food})}] \times (1 / (1 - e^{(-k_d t)})) \quad (\text{Equation 4})$$

where $C_{0,d}$ is the concentration in fish at the beginning of the depuration phase (ng g^{-1}), k_d is the depuration rate constant, I is the food ingestion rate constant (g food/g fish/day), $C_{(\text{food})}$ is the concentration of PFAS in food (ng g^{-1}) and t is time (days). Assimilation efficiencies were calculated at the end of the uptake period (day 21).

The dietary biomagnification factor (BMF) was then determined based on the following equation:

Dietary (lab-derived) biomagnification factor: $\text{BMF} = (I \times \alpha)/k_d$ (Equation 5)

Results and Discussion

Uptake and Depuration of PFOA, L-PFOS, L+Br-PFOS and GenX.

PFOA, L-PFOS and total PFOS were detected in exposed fish at all sampling points throughout the experiment, yet no GenX was detected in the tissue of any fish sampled during the uptake (0-21 days) or depuration (22-63 days) phases of the study. The performance of the analytical method was determined to be both accurate (%RSD 15-17%) and precise (68-113% recovery at 100 ng/g dw) and was within the recommended USEPA 537.1 performance guidelines (accuracy RSD <20%; precision $\pm 50\%$ for low level spikes, $\pm 30\%$ for low level spikes). GenX was also confirmed in experimental food (mean of $463\text{-}690 \text{ ng g}^{-1}$ over three weeks; Table 1). Carbon-labelled GenX was detected in all sample extracts indicating the extraction protocol worked and that the instrumental conditions were appropriate. Rapid elimination (hours) of orally administered GenX has been shown in other animals (Gannon et al. 2016) and thus even if GenX uptake did occur in the fish in this study, it may not have been bioavailable via ingestion or could have been rapidly eliminated between feeding and sampling cycles (<24 hr).

Rapid uptake of PFOA, L-PFOS and L+Br-PFOS was observed, and steady state, whole body concentrations for each compound were achieved by 14 days (Figure 1). In juvenile rainbow trout an estimated steady state time of 10 days for PFOA and 43 days for PFOS was reported following a 34 day dietary exposure to a similar dose ($\sim 500 \text{ ng g}^{-1}$) (Martin et al. 2003b).

All fish had low yet detectable concentrations of PFOA ($0.11\text{-}0.12 \text{ ng g}^{-1}$) and PFOS (L+Br) ($0.20\text{-}14 \text{ ng g}^{-1}$) at the beginning of the experiment (Table 2). These represent background levels and are considered low relative to other measured values of PFAS in Australian biota (Taylor and Johnson 2016, Thompson et al. 2011). Since fish were exposed individually, it was possible to adjust specific food portions (based on fish weight) to ensure the same dose was offered to each fish. In addition, the quantity of food eaten by each fish was recorded, enabling a calculation of an individual ingestion rate (grams day^{-1}) for each fish. The variation in ingestion rates between fish was subsequently low, with values ranging between $0.0192 \pm 0.0007 \text{ g day}^{-1}$ (Control) and $0.0185 \pm 0.0007 \text{ g day}^{-1}$ (PFAS). These differences were not statistically significant. The highest whole body concentrations observed in gobies in the present study (PFOA; $38.9 \text{ ng g}^{-1} \text{ ww}$; PFOS; $123 \text{ ng g}^{-1} \text{ ww}$) were much higher than reported in whole body rainbow trout samples from a similar dietary study (PFOA: $15.4 \text{ ng g}^{-1} \text{ ww}$; PFOS: $49.0 \text{ ng g}^{-1} \text{ ww}$).

(Goeritz et al. 2013), yet the depuration rates and biological half-lives were similar (PFOA: 5.9 days; PFOS: 15.4-16.7 days).

The depuration rates for PFOA and PFOS varied, with PFOA exhibiting much faster depuration than PFOS (L or L+Br PFOS) (Figure 1; Table 2). After 7 days, only 15% of the maximum concentration (24 ng g^{-1}) of PFOA remained in fish whole body, whereas for L-PFOS, 81% of the maximum concentration (100 ng g^{-1}) and for L+Br PFOS, 65% of the maximum concentration (123 ng g^{-1}) remained. The corresponding biological half-lives were calculated to be 5.9 days for PFOA, 16.7 days for total PFOS (L+Br) and 15.4 days for L-PFOS (Table 2). The depuration rates and biological half-lives calculated for PFOA and PFOS in the present study were in general agreement with values published for other fish species (pelagic, freshwater), despite differences in the specific tissues that have been used for analysis (i.e. blood, liver, muscle, carcass or whole body) (Falk et al. 2015, Goeritz et al. 2013, Martin et al. 2003b).

The assimilation efficiency of PFOA was low (22%) and there was a correspondingly low biomagnification factor (BMF) of 0.021. Linear PFOS showed a much higher assimilation efficiency of 60% and BMF of 0.346, and total PFOS (L+Br) was lower, with an assimilation efficiency of 42% and corresponding BMF of 0.261 (Table 2). GenX did not appear to accumulate at all in fish during the 21 day dietary exposure, which is in general agreement with other studies that have reported rapid elimination of GenX in both oral and intravenous infusions for rats, mice and cynomolgus monkeys (Gannon et al. 2016). It is unclear whether the lack of detection of GenX in any fish is due to a complete lack of uptake or rapid elimination. Further testing, in both aqueous and dietary exposure scenarios would assist in determining if any uptake of GenX does occur in fish. (Kelly et al. 2009)(Strynar et al. 2015, Sun et al. 2016)

Growth and Feeding Rates.

Growth rates between control and exposed fish showed no significant differences (ANOVA, $F_{(1,86)} = 1.2547$, $p = 0.2658$) and throughout the experiment, the mortality rates were very low (3%), with just 2 control and 1 treatment fish dying. Similar, low mortality rates have been reported in other PFAS bioaccumulation studies (Lee et al. 2012, Martin et al. 2003b). Fish were maintained in separate enclosures during feeding times, enabling individual feeding rates to be calculated for each fish. Outside of feeding times, two fish were housed in each plastic enclosure, and were paired together such that it was easy to keep track of each individual (i.e. by pairing a male with a female, or a larger fish with a smaller fish). The mean ($\pm$ SEM) feeding rates (percentage of body weight fed per day) ($\text{g food g}^{-1} \text{ fish d}^{-1}$) for control fish were $2.94 \pm 0.19\%$ and for PFAS-treated fish were $2.68 \pm 0.15\%$, and there were no statistical differences between the two groups (ANOVA, $F_{(1,95)} = 1.1162$, $p = 0.2934$). These values are in the same range as reported in other studies (Falk et al. 2015, Martin et al. 2003b) and are considered an appropriate reflection of what feeding rates (and therefore dose rates) might be in the wild.

Linear PFOS Enrichment.

One of the major production methods used to create PFOS is electrochemical fluorination, which results in the formation of a mixture of PFOS isomers, that usually has a fairly consistent ratio of linear (70%) and branched (30%) isomers (Fang et al. 2016, Sharpe et al. 2010). However, measured PFOS isomer ratios in terrestrial and aquatic wildlife sometimes deviate from this, to exhibit either enriched (>70%) or reduced (<70%) linear PFOS ratios (Greaves and Letcher 2013, Sharpe et al. 2010). The ratio of linear to branched PFOS in fish changed throughout the course of the experiment, from 77-81% during the uptake phase (with a consistent food ratio of 71-73%), to 90% in the depuration phase, indicating differences in the depuration kinetics of linear and branched PFOS (Figure 2; Table 2). Other laboratory studies have reported similar findings (Chen et al. 2015, Sharpe et al. 2010), whilst field studies with polar bears (Greaves and Letcher 2013) and fish and invertebrates (Asher et al. 2012) have observed differences in percent composition of linear to branched PFOS within specific tissues. The reasons for these discrepancies are not well described, but thought to be due to isomer-specific differences in pharmacokinetics, biotransformation, elimination capacity between species as well as potential differences in the sources of dietary PFOS to each group (Fang et al. 2016, Sharpe et al. 2010). Organ-specific differences in PFOS isomer elimination rates have also been observed in rainbow trout, with the gills and kidneys identified to preferentially eliminate branched PFOS (Sharpe et al. 2010). If excretory organs such as gills and kidneys preferentially eliminate branched PFOS in blue spot gobies, this could explain the enrichment in the percent of linear PFOS within our whole animal samples. A better understanding of the factors that influence isomer-specific elimination rates of PFOS in aquatic biota would be valuable for improving our understanding of how PFOS moves through the environment and may assist in determining both the origin and the timing of the PFOS bioaccumulation.

Conclusions

The present study has demonstrated that PFOA and PFOS (linear and branched) bioaccumulate in small benthic fish following dietary exposure, and the depuration rates and biological half-life ($t_{1/2}$) values are similar to values reported elsewhere in the literature for pelagic species. PFOA is depurated three times more quickly than PFOS, and we believe this is the first time a shift in the composition (between linear and branched PFOS) within whole fish over time has been reported under controlled conditions. Further research is required to understand the isomer-specific regulation of PFOS by different organisms at an organ-specific level to better understand the implications for toxicity.

The PFOA-replacement substance, GenX did not appear to bioaccumulate in fish following ingestion of spiked food at environmentally-relevant concentrations. We cannot confirm whether the lack of detection of this substance in any fish samples is due to (1) a lack of uptake or (2) very rapid elimination (<24 hr). However, since other studies

have reported rapid elimination without any metabolism, toxicity or bioconcentration in fish, we can hypothesise that GenX may also be eliminated rapidly in fish.

Future research should concentrate on the organ-specific accumulation of PFAS compounds, especially with regard to differences between branched and linear PFOS in each tissue to better understand the metabolism and regulation of these compounds.

There is an obvious need for more dietary and aqueous bioaccumulation studies in fish with different PFAS, especially newer, replacement substances (such as GenX and others), in order to establish reliable toxicokinetic information which is essential for effective risk assessment and environmental protection. In particular, studies should concentrate on generating whole body and organ-specific biological half-life data for a range of commercially important fish and aquatic species to understand the implications for human consumers.

Supplemental Data—The Supplemental Data are available on the Wiley Online Library at DOI: 10.1002/etc.xxxx.

Acknowledgement— This project was funded by Melbourne Water Corporation with additional resources provided by the Centre for Aquatic Pollution Identification and Management (CAPIM), University of Melbourne, RMIT University and the Victorian Marine Sciences Consortium. We acknowledge the support of Agilent Technologies for technical assistance with instrument setup and operation. We thank T. Mehler, J. Myers, M. Hoak, J. French, M. Sole and F. Alexander for assistance with fish collections, and R. Watson, S. Krueger, N. Williamson, S. Nie for assistance with laboratory procedures. All fish collection and experimentation was done following methods approved by the University of Melbourne Animal Ethics Committee (Approved ID: 1613970.1).

Data availability—Data, associated metadata, and calculation tools are available from the corresponding author (kathryn.hassell@rmit.edu.au).

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Figures

Figure 1. Fish whole body concentrations (ng g^{-1} wet weight) of PFOA, linear PFOS (L PFOS) and linear + branched PFOS (L+Br PFOS) in blue spot gobies throughout the uptake (21 days) and depuration (42 days) phases of the study. Each data point represents the mean ($n=4$) $\pm$ standard error of the mean except day 63 where $n=2$. Whole body concentrations that were $<\text{LOR}$ (PFOA 0.3 ng/g ; PFOS 0.6 ng/g) were substituted with a $\frac{1}{2}$ LOR value.

Figure 2. Temporal change in ratio of linear to branched PFOS (ng g⁻¹ ww) in blue spot goby whole body samples throughout the course of the experiment. Prepared food samples contained 71-73% Linear PFOS. Each data point represents the mean (n=4) ± standard error of the mean. On day 63 n=1.

Table 1. Concentrations of PFAS compounds in food (brine shrimp-agar) throughout uptake and depuration phases of a fish dietary accumulation study. Control food samples contained an equivalent volume of carrier solvent (methanol) as the spiked food samples.

Treatment	Concentration (ng g ⁻¹ ww)				% linear PFOS
	PFOA	L+Br PFOS	L-PFOS	GenX	
PFAS-spiked food					
uptake phase (week 1)	417 ± 96.39	557 ± 110.4	407 ± 98.24	690 ± 158.9	71.8
uptake phase (week 2)	265 ± 21.36	437 ± 27.54	310 ± 21.27	463 ± 7.12	72.3
uptake phase (week 3)	282 ± 6.53	468 ± 10.87	343 ± 8.03	516 ± 60.33	72.4
Control food					
uptake phase (week 1-3)	<10	<10	<10	<20	-
depuration phase (week 4-9)	<10	<10	<10	<20	-

Table 2. Whole body concentrations (ng g⁻¹ ww) and kinetics information of PFOS, PFOA and GenX in blue spot gobies (*Pseudogobius sp.*), following dietary exposure up to 21 days and up to 42 days depuration.

PFAS Compound	Structure	Mean whole body concentration (ng/g wet weight)			Depuration rate constant (k _d) (d ⁻¹)	t _{1/2} (days)	BMF	t _{ss} (days)*	Assimilation efficiency^ (%)
		beginning of uptake period (0 days)	end of uptake period (21 days)	end of depuration period (63 days)					
PFOA	C ₈ HF ₁₅ O ₂	0.11 ± 0.002	38.8 ± 15.1	0.12 ± 0.00	0.1473	5.9	0.021 ± 0.001	14	22.43 ± 1.54
L-PFOS	C ₈ HF ₁₇ SO ₃	2.66 ± 1.54	99.6 ± 17.4	7.00	0.0449	15.4	0.346 ± 0.015	14	60.64 ± 3.84
L+Br-PFOS	C ₈ HF ₁₇ SO ₃	3.07 ± 1.80	123.1 ± 8.84	14.6 ± 7.56	0.0416	16.7	0.261 ± 0.011	14	42.17 ± 2.66
GenX	(HPFO-DA:C ₆ HF ₁₁ O ₃)	<LOQ	<LOQ	<LOQ	NA	NA	NA	NA	NA

*steady state (tss) was defined as the time after which there was no statistically significant increase in whole body concentration during the uptake phase.

^assimilation efficiency (a measure of the absorption of PFASs across the gut) was calculated at the end of the uptake period (21 days).

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Author/s:

Hassell, KL;Coggan, TL;Cresswell, T;Kolobaric, A;Berry, K;Crosbie, ND;Blackbeard, J;Pettigrove, VJ;Clarke, BO

Title:

Dietary Uptake and Depuration Kinetics of Perfluorooctane Sulfonate, Perfluorooctanoic Acid, and Hexafluoropropylene Oxide Dimer Acid (GenX) in a Benthic Fish

Date:

2020-03

Citation:

Hassell, K. L., Coggan, T. L., Cresswell, T., Kolobaric, A., Berry, K., Crosbie, N. D., Blackbeard, J., Pettigrove, V. J. & Clarke, B. O. (2020). Dietary Uptake and Depuration Kinetics of Perfluorooctane Sulfonate, Perfluorooctanoic Acid, and Hexafluoropropylene Oxide Dimer Acid (GenX) in a Benthic Fish. *Environmental Toxicology and Chemistry*, 39 (3), pp.595-603. <https://doi.org/10.1002/etc.4640>.

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