OCCURRENCE AND FATE OF SELECTED ORGANIC CONTAMINANTS IN SOILS, SEDIMENT AND ESTUARINE WATER FROM SOUTH- EAST QUEENSLAND

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Abstract

Organic contaminants are ubiquitous in the environment and can have health implications on humans and ecological life. Concerns on the presence of these contaminants in the environment, particularly sediments, waterways and soils around firefighting training grounds have featured in national and international news. While a lot of data on organic contaminants is now available in the literature, there is limited information on the contamination of estuarine sediments, water and firefighting training grounds in South-East Queensland. Therefore, there is an urgent need to understand their transport and fate in the estuarine sediment, water and soils from firefighting training grounds. It is also important to understand the physico-chemical factors impacting the distribution of the contaminants in the environment.

Based on their chemical persistence and potential toxicities, and the dearth of reports in the literature in relation to South-East Queensland, the organic contaminants: polybrominated diphenyl ethers (PBDEs), polychlorinated biphenyls (PCBs), hexabromocyclododecanes (HBCDDs), per-and polyfluoroalkyl substances (PFASs), pharmaceuticals and personal care products (PPCPs) and current-use pesticides (CUPs) in the Brisbane River estuary as well as PFASs contamination of core soils at a firefighting training ground (FTG) were investigated.

Samples were prepared by solid phase extraction (water samples for PPCPs, CUPs and PFASs), accelerated solvent extraction (sediment for PBDEs, PCBs and HBCDDs) and ultra-sonication (sediment and soils for PFASs) followed by analysis of the extracts using Gas Chromatography (for PBDEs and PCBs) and High performance Liquid Chromatography (for BDE-209, HBCDDs, PFASs, PPCPs and CUPs) in tandem with mass spectrometry performed in selected reaction monitoring mode. For the PPCPs and CUPs, the fate of the contaminants were assessed using a modified mixing plot model based on a salinity gradient while for the PFASs studies, residual PFASs at the FTG was estimated based on Theissen polygon concept.

Consistently, >90% of the observed $\sum_8$PBDE concentration in the sediment was attributed to BDE-209. Mean PBDE levels (ng/g dry wt.) were: 4.4 ± 3.2 ($\sum_8$PBDE) and 4.4 ± 3.0 (BDE-209) across 22 sampling sites along the estuary. The mean $\sum_7$PCB and

∑HBCDD were 5.4 ± 4.5 and 1.0 ± 1.5 ng/g dry wt. respectively. Contaminant levels were evenly distributed along the River and were generally low compared to similar studies around the world. Perfluoroalkyl sulphonate (PFOS) contamination in the sediment was the highest measuring up to 2.6 ± 0.8 ng/g dry wt. Similarly, PFOS was dominant in the water samples with a mean concentration of 13.7 ± 3.5 ng/L followed by perfluorooctanoic acid (PFOA) measuring 7.4 ± 0.9 ng/L. The concentrations of PFOS and PFOA in the water samples that were collected in 2017 for this study have increased by factors of 3 and 2 respectively when compared to a previous study in the Brisbane River following the flood events of 2011.

In the soil samples, PFOS concentration was the highest, measuring up to 2170 ng/g dry weight at a depth 0.5-1.0 m. The mass load of PFOS at the FTG was estimated to be ~6.5 kg within a 21000 m^3 volume of bulk soil. The PFASs plume along the depth profile also indicates transport from the top sleeve (0-0.5 m) into lower sleeves (0.5-2 m). Estimated average distances of PFASs migration over 10 years; 0.6 m (PFOS) and 2.6 m (both PFOA and PFHxS), suggest that PFASs released at the FTG (>20 years of first exposure at the site) would have been transported into lower depths; between 1 m and >2 m depth of soil. Salinity was observed to affect the transport and distribution of PFASs in the core soils among other soil physico-chemical factors such as organic carbon, pH and mineralogy.

The mean concentrations of major pharmaceuticals in the Brisbane River were: 46 ± 30 ng/L (carbamazepine), 42 ± 34 ng/L (gabapentin), 28 ± 25 ng/L (iopromide), 26 ± 20 ng/L (tramadol) and 24 ± 19 ng/L (venlafaxine). The pharmaceutical products (carbamazepine, temazepam and paraxanthine) and CUPs (tebuconazole, simazine and 2,4 D) showed conservative transport along the River estuary. The major source of the PPCPs was identified as a large wastewater treatment plant in the upper part of the estuary, while the major sources of most CUPs were agricultural and parkland areas upstream of the city core, presumably via surface water runoff drains discharging to the river.

This research provided new knowledge regarding: (a) PFASs and HBCDD contamination of sediments, for the first time, in the Brisbane River estuary, (b) the development and application of a modified mixing plot model in determining the conservative behaviour of PPCPs and CUPs in estuarine waters, (c) estimation of mass

load of residual PFASs as a useful input in the design of effective remediation strategies at the site and also understanding the soil physico-chemical factors impacting the transport of PFASs in soils along a depth profile at the FTG. The study also produced a publication in a peer reviewed journal which has been cited by other researchers around the world; thereby contributing to knowledge base on the contaminants.

Studies like this contribute to the much needed documentation of the global budget of persistent organic contaminants in environmental matrices. When compared to historic data, the observed increase in PFASs contamination of water samples from the Brisbane River is indicative of on-going PFASs inputs along the estuary; hence, the need to carry out source investigations in future studies. Estimation of residual PFASs mass load at the FTG can contribute to the development of effective containment strategies for AFFF impacted soils. The observed conservative contaminants reported in this thesis can be used to investigate the impacts of the chemicals on aquatic organisms since they are not readily degrading along the River.

List of Publications

Refereed Journal Papers

- Alfred K. Anim, Daniel S. Drage, Ashantha Goonetilleke, Jochen F. Mueller, Godwin A. Ayoko. (2017). Distribution of PBDEs, HBCDs and PCBs in the Brisbane River estuary sediment. Marine Pollution Bulletin, 120, 165-173.

Papers in Preparation for Submission

- Alfred K. Anim, Jennifer Braunig, John Corfield, Craig Barnes, Curtis Godlonton, Ashantha Goonetilleke, Godwin A. Ayoko, Jochen F. Mueller. Distribution of PFASs in core soils impacted by aqueous film-forming foams. [Proposed Journal: Chemosphere]
- Alfred K. Anim, Michael S. McLachlan, Kristie Thompson, Godfred O. Duodu, Gavin Birch, Ashantha Goonetilleke, Godwin A. Ayoko, Jochn F. Mueller. Occurrence and fate of target pharmaceuticals, personal care products and pesticides in the Brisbane River estuary. [Proposed Journal: Chemosphere]
- Alfred K. Anim, Jennifer Braunig, Gavin Birch, Ashantha Goonetilleke, Jochen F. Mueller, Godwin A. Ayoko. Contamination of PFASs in the Brisbane River estuary. [Proposed Journal: Marine Pollution Bulletin]

Oral Conference Presentations

- Anim A.K, Goonetilleke A., Mueller J.F., Ayoko G.A., Spatial assessment of some legacy POPs in estuarine sediment. Paper presented at the Queensland Annual Chemistry Symposium (November 27, 2017), Queensland University of Technology, Australia.
- Alfred K. Anim, Jennifer Braunig, Ashantha Goonetilleke, Jochen F. Mueller, Godwin A. Ayoko, Investigations of perfluoroalkyl substances in Brisbane River sediment. Poster presented at the Analytical and Environmental Chemistry Division Symposium (July 18-20, 2016), Adelaide, Australia.

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List of Abbreviations

PFBA	Perfluorobutanoic acid
∑	Sum
µg/ml	microgram per millilitre
µL	micro litre
2,4 D	2.4-dichlorophenoxyacetic acid
2,4,5_T	2,4,5-trichlorophenoxyacetic acid
AFFF	Aqueous film-forming foams
BDE	Brominated diphenyl ether
CUPs	current use pesticides
DCPU	3,4-dichlorophenyl urea
DEET	N,N-diethyl-m-toluamide
FR-Ps	Flame retardant pollutants
FTG	Firefighting training ground
g	Gram
GC-MS/MS	Gas cromatography-Tandem Mass spectrometry
HBCDD	Hexabromocyclododecane
HPLC-MS/MS	High performance liquid chromatography-Tandem Mass Spectrometry
K_d	Partition coefficient
kg	Kilogram
km	Kilometre
K_{oc}	Organic carbon normalized K_d
L	Litre
L/kg	litre per kilogram
LOD	Limit of detection
LOQ	Limit of Quantitation
LRAT	Long range atmospheric transport
MCPA	2-methyl-4-chlorophenoxyacetic acid
MeOH	Methanol
ml	Millilitres
mM	Millimolar
NESS	Non-extracted side spike
ng/g	nanogram per gram
ng/L	nanogram per litre
ng/ml	nanogram per millilitre
NH_4OH	Ammonium hydroxide
PBDE	Polybrominated diphenyl ether
PCB	Polychlorinated biphenyl

PFAA	Perfluoroalkyl acids
PFASs	Per-and polyfluoroalkyl substances
PFBS	Perfluorobutane sulphonate
PFCA	Perfluoroalky carboxylic acids
PFCs	Per-and polyfluoroalkyl chemicals
PFDA	Perfluorodecanoic acid
PFDoDA	Perfluorododecanoic acid
PFHpA	Perfluroheptanoic acid
PFHxA	Perfluorohexanoic acid
PFHxS	Perfluorohexane sulphonate
PFNA	Perfluorononaoic acid
PFOA	Perfluorooctanoic acid
PFOS	Perfluorooctane sulphonate
PFPeA	Perfluoropentanoic acid
PFSA	Perfluoroalkyl sulphonic acids
PFUnDA	Perfluoroundecanoic acid
POPs	Persistent organic pollutants
PPCP	Pharmaceuticals and personal care products
QA/QC	quality assurance/quality control
SMRM	Scheduled multiple reaction monitoring
STPs	Sewage treatment plants
TOC	Total organic carbon
WWTPs	wastewater treatment plants
α	Alpha
β	Beta
ϒ	Gamma
PDMS	Polydimethylsiloxane
SPME	Solid phase microextraction
PSU	Practical salinity unit

Statement of Original Authorship

The work contained in this thesis has not been previously submitted to meet requirements for an award at this or any other higher education institution. To the best of my knowledge and belief, the thesis contains no material previously published or written by another person except where due reference is made.

Signature: QUT Verified Signature

Date: 16/10/2019

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Dedication

I dedicate this Thesis to my dear wife, Gloria Kumiwaa Anim and our beloved children, Edwin, Erwin and Gloria for their love and encouragement throughout the study.

Chapter 1: Introduction

This chapter outlines the background (Section 1.1) and research problem (Section 1.2), and a justification for the research (Section 1.3). Section 1.4 provides the research hypothesis whereas the aims and specific objectives of the research are outlined in Section 1.5. Finally, Section 1.5 includes an outline of the remaining chapters of the thesis.

1.1 Background

Organic contaminants are man-made chemicals which have the potential to cause environmental pollution due to their use and release into the wider environment. As the populations of societies keep increasing, the demand for and use of products containing organic contaminants are expected to increase and the associated release of these contaminants into environmental media including soils, sediment and water is expected to grow.

Organic contaminants that are of environmental concern include diverse groups of chemicals such as polybrominated diphenyl ethers (PBDEs), polychlorinated biphenyls (PCBs), hexabromocyclododecanes (HBCDDs), organochlorine pesticides (OCPs), polyaromatic hydrocarbons (PAHs), per-and polyfluoroalkyl substances (PFASs), pharmaceuticals and personal care products (PPCPs) and current-use pesticides (CUPs). This thesis focuses on PBDEs and PCBs as legacy contaminants whereas HBCDDs, PFASs, PPCPs and CUPs are classified as emerging contaminants.

Due to the widespread presence of PBDEs, PCBs and HBCDDs as ingredients in consumer products used to protect equipment and property from fire, they will be referred to as flame retardant pollutants (FR-Ps). Flame retardant pollutants are also incorporated into industrial products including electric/electronic equipment, paints, transformer oil and automobiles because of their fire inhibiting properties. However, FR-Ps can leach out from the consumer products into environmental compartments such as soil, sediment, air and water. While the presence of the strong carbon-halogen (halogen:Cl or Br) bonds renders the FR-Ps (C-Cl or C-Br bonds) persistent, their chemical structure is also characterised by both lipophilic and hydrophilic groups which defines their ubiquitous nature and hence their availability in

varying environmental matrices. Therefore, their environmental fate and mobility are of high concern due to their ubiquitous nature, persistence, potential for bioaccumulation, and consequent adverse effects on human and ecosystem health [1, 2].

Similarly, some PFASs (such as the perfluoroalkyl sulphonic acids and perfluoroalkyl carboxylic acids) are also persistent and ubiquitous. In addition, they have the potential to bioaccumulate and cause health risks to ecological life. Unlike the FR-Ps, however, PFASs are characterised by strong C-F bonds and are often referred to as emerging contaminants because investigations are still on-going to conclusively understand their toxicity. The widespread incorporation of PFAS into aqueous film-forming foams (AFFF) as surfactants used for firefighting as well as their applications as stain and oil repellents (e.g. food packaging materials, textiles and non-stick agents in cookware) highlight the need to monitor their presence in the environment.

Other emerging contaminants including PPCPs and CUPs are also ubiquitous and can alter the physiology of non-target organisms when they are released into the environment and this could result in potential health effects to both human and ecological organisms [3-5]. CUPs (e.g. herbicides, insectides) have mostly been applied to increase agricultural yield while PPCPs (e.g. antibiotics, analgesics) are used to improve human or veterinary health and well-being. These applications increase their potential to contaminate the environment.

FR-Ps, PFASs, PPCPs and CUPs can be mobilised into the environment from their primary products. For example, the formulations in FR-Ps and PFASs can leach out from the consumer products into the environment through varying routes including; the manufacturing stage due to poor processing protocols, household products due to temperature variations, poorly engineered waste disposal sites and or/ direct fallouts into soils and water during firefighting from the use of AFFF. Similarly, PPCPs, mostly released via excreta or bath water into waste streams of households and hospitals, are not fully removed when they are channelled through sewage and waste water treatment plants and can therefore contaminate environmental matrices while CUPs can contaminate soils directly during applications or indirectly contaminate water and sediments via surface wash-offs and stormwater drainage.

The presence of organic contaminants in environmental matrices can lead to the contamination of the food chain, resulting in adverse health effects to humans and animals. For example, some studies have linked cancer, endocrine disorders, diabetes and reproductive failures in some humans and animals to the potential exposure of PCBs and PBDEs [6, 7]. Although studies on the toxicity of PFASs in humans are inconclusive to date, preliminary studies have linked human exposure to potential immunotoxicity, reproductive damage and neurotoxicity [8-10]. Also, prolonged exposures of CUPs and PPCPs can alter the biological functioning of aquatic organisms even at low concentrations [11-13]. Accordingly, production and use of some organic contaminants have been restricted and/ or banned worldwide [14, 15].

Notwithstanding the legislations to control their production and usage, FR-Ps and PFASs are still present at measurable concentrations in various samples, including air, water, sediment, soils, blood serum, human milk and biota even in areas far from the regions where they have been produced or used [16-21]. Soils and sediment, in particular, can serve as sinks for these contaminants, therefore good matrices for assessing and evaluating FR-Ps and PFASs in the environment.

The fate and mobility of organic compounds in soils, water and sediment can be influenced by some key physicochemical properties (e.g. pH, salinity, organic carbon, cation/anion exchange ratio, mineralogy) of the matrices. Once in the environment, contaminants can also undergo degradation to produce compounds of lower molecular weight (congeners) which are more toxic [22]. For example, the PBDE congeners: penta-bromodiphenyl ethers (penta-BDE) and octa-BDE have recently been measured in sediment and soil samples from Sydney, Australia [18]. Similarly, PFASs precursors such as fluorotelomer alcohols (FTOHs) and perfluoroalkyl sulfonamides (PFOSA) can undergo biotransformation to produce more persistent and potentially toxic lower molecular weight congeners, namely perfluorooctanoic acid (PFOA) and perfluoroctane sulphonate (PFOS), respectively [23].

In Brisbane, soils and sediment contamination resulting from PBDEs, PCBs, HBCDDs, and PFAS have not been extensively investigated. Limited data exist on the levels of PCBs [24] and PBDEs [25] measured in River sediments from Australia. While studies on HBCDD contamination in sediments from the Brisbane River, for example, have not been frequently

cited in the literature until this research, concentrations of HBCDD in estuarine core sediments from Sydney in Australia, shows about 100% increase from the late 1990s (0.12-2.9 ng/g) to 2014 (1.8-5.3 ng/g) [18]. This gives an indication that river systems in other Australian states such as the Brisbane River could be contaminated too. Similarly, there is no data representing PFASs contamination of sediment from the Brisbane River. Recent studies, however, only measured PFASs levels in water samples collected from the Brisbane River [26] and in sediment collected from the Sydney River estuary [27]. Elsewhere, soils from AFFF impacted fire-fighting training grounds (FTG) have been found to contain PFASs and the potential to contaminate ground/surface water and aquatic organisms noted [18, 19, 20, 21, 29, 30]. Notwithstanding, the distribution of PFASs in most AFFF impacted FTGs in Queensland have not been investigated, particularly, along a soil depth profile. The consistent inputs of CUPs and PPCPs into aquatic environments due to the solubility properties which makes it possible for measurable concentrations of these contaminants to remain unremoved by wastewater treatment plants (WWTPs) and sewage treatment plants (STPs) can threaten aquatic life due to chronic exposures [5]. Apart from the lack of investigations to understand the transport mechanisms of these compounds in water, soils/sediment from South-East Queensland, data on PCBs, HBCDDs and PFASs contamination in sediments is not cited in the literature in the past decade,.

These persistent organic contaminants are likely to impact water, sediments and soils in South-East Queensland. For example, two major floods which occurred in 2011 and 2013 were reported to have washed-off household materials and automobiles into the Brisbane River estuary [28]. These floods also compromised the integrity of some landfill sites along the Brisbane River catchment [26]. The Brisbane River is an economical water way in South-East Queensland which serves many purposes, including transportation and recreational activities. Nonetheless, the River is also susceptible to stormwater via drains from residential, commercial and agricultural sources. Such occurrences can contribute to the introduction of organic contaminants from consumer products as well as from waste disposal sites with engineering defects into River estuaries and soils. Notably, leachates from landfill sites, agricultural fields and industrial waste streams are potential sources of POPs contamination in Rivers and soils [29-33]. In addition, the historic usage of fluorinated aqueous film-forming foams (AFFF) such as 3M Lightwater and Ansulite, at least over the last two decades at most FTGs in South-East Queensland can serve as a liable source of PFASs contamination

in the soils. These incidences coupled with the potential transportation of contaminants across borders and their characteristic persistence makes it prudent to investigate their fate in water, sediment and soils, including their detection and measurement at low levels using Gas chromatography (GC) and High-Performance Liquid Chromatography (HPLC) in tandem with triple quadruple mass spectrometry (MS/MS).

This research, therefore, primarily investigated the occurrences and transport mechanisms of some FR-Ps, PFASs, CUPs and PPCPs in estuarine waters and sediment as well as soils from a fire-fighting training ground (FTG) where PFASs, incorporated as surfactant in aqueous film-forming foams (AFFF), was previously used. Potential contaminant hotspots were assessed, while also providing understanding for the behaviour of contaminants and the physicochemical factors impacting their mobility in water, sediment and soils. In addition, results from this research provide background information on the status of the contaminants in water, sediment and soils in South-East Queensland and thus supporting future environmental legislations.

1.2 Research problem

The research questions that inspired this thesis are as follows:

- What are the current levels of organic contaminants (PBDEs, PCBs, HBCDDs, PFASs, PPCPs and CUPs) in environmental samples (soils, sediment, water) from Brisbane, South-East Queensland?
- How does land-use influence spatial distribution of these contaminants along the Brisbane River?
- How are the organic contaminants (PPCPs and CUPs) behaving in estuarine waters along a salinity gradient and what are their potential environmental impacts?
- How much of PFASs mass load arising from the past use (>20 years) of AFFF at a FTG is present in the bulk soils up to a depth of 2 m?
- What are the migration patterns of PFASs in soil cores at a FTG along a depth profile and what physico-chemical factors could be influencing transport of PFASs in the soils?

1.3 Justification for the research

Australia has adopted international restrictions like the Stockholm Convention to reduce the impact of PFASs contamination on the environment and health. For example, the risk reduction approaches of PFASs by OECD/UNEP [34, 35]. Nonetheless, a survey across 13 landfill sites in Australia shows the presence of PFASs, PBDEs and HBCDDs in leachates [29], suggesting that these compounds are still present in the environment. Currently, there is no data in the literature reflecting baseline concentrations of PFASs and HBCDD contamination in sediment from the Brisbane River. Uncontrolled release of organic contaminants even at low concentrations can lead to their accumulation particularly in soils and sediment. Consequently, soils and sediment can become secondary sources as hydrodynamics and anthropogenic activities coupled with physicochemical conditions can subsequently remobilise these compounds into water and hence pose health hazards to aquatic fauna and humans [3, 36]. The contaminated soils can also impact ground/surface waters [37-40]. Elsewhere, concentrations of CUPs and PPCPs have been reported in both fresh and saline waters due to their continuous uncontrolled inputs [3-5, 41], indicating the need to investigate the fate of these contaminants in rivers and estuaries such as the Brisbane River estuary.

The mobility of these contaminants can be influenced by partitioning properties of the compounds as well as soil and sediment physicochemical properties. However, previous studies in Queensland [18, 26, 42, 43] did not investigate the impact of these properties on the distribution of the organic contaminants. Similarly, the few studies [40, 44-46] reported on AFFF- impacted FTGs in Queensland did not account for the role of soil physicochemical properties (e.g. organic carbon, salinity, pH, and mineralogy) on the transport and fate of PFASs in soils, along a depth gradient. Also, investigations have not been carried out to understand the behavior of CUPs and PPCPs in estuarine waters in Australia, although these contaminants can be released through effluents and surface run-off into the estuarine waters. It is therefore important to investigate the mobility mechanisms of the organic contaminants as these mechanisms can differ depending on the characteristics of the soils, sediment or water at specific sites.

1.4 Research hypothesis

Soils and sediment have been identified as major matrices for organic contaminants in the environment. These contaminants are usually released primarily from consumer products into soils/sediment through varying release pathways: pesticides from agriculture lands, household products from residential/commercial catchments, industrial waste discharges or leachate from waste disposal sites. Also the increasing population and increased usage of CUPs and PPCPs coupled with the ability of some of these compounds to remain unremoved in effluents after wastewater treatment could potentially contribute to their continuous release into aquatic environments. While transects of the Brisbane River in South-East Queensland are susceptible to influences from stormwater discharges and land-use catchments (agricultural, commercial and industrial), there are also firefighting training grounds which have previously used aqueous film-forming foams containing some PFASs for firefighting training exercises as well as quenching accidental fires. The persistent and pseudo-persistent nature of these compounds allows them to stay in soils/sediment over a long period even at very low concentrations. Thus physicochemical properties of the compounds such as partitioning coefficient as well as soil/sediment physicochemical properties (e.g. organic carbon, pH, salinity, and mineralogy) can play a significant role in the partitioning and distribution of these compounds.

Hypothesis 1 (H1): There are measurable concentrations of PBDEs, PCBs, HBCDDs, and PFASs in sediments along transects of the Brisbane River estuary.

Hypothesis 2 (H2): PPCPs and CUPs can be detected and measured in surface water samples from Australia regardless of the channelling of domestic and hospital waste streams through treatment plants prior to environmental release or re-use of wastewater.

Hypothesis 3 (H3): Salinity potential of the Brisbane river estuary could impact the behaviour and fate of PPCPs and CUPs in the water.

Hypothesis 4 (H4): Regardless of the cessation of the usage of fluorinated AFFF at FTGs in South-East Queensland for at least two decades, PFASs due to past AFFF usage are still available in soil cores up to 2 m depth at these FTGs.

Hypothesis 5 (H5): PFAS residual concentrations at the FTGs will decrease at the top 0.5 m level of soil cores and increase at lower depths (0.5-2 m).

Hypothesis 6 (H6): The physicochemical parameters such as organic carbon, pH, salinity, and mineralogy of soils and sediment will significantly contribute to the partitioning and distribution of organic contaminants at the study sites.

1.5 Research Objectives

The overarching aim of this thesis is to investigate the fate of some organic contaminants and understand the key factors that influence their mobility in soils/sediment and water from South-East Queensland.

This will be achieved specifically as follows:

- Assess the spatial distribution of PBDEs, PCBs, HBCDDs, PFASs, PPCPs and CUPs in sediment and water from the Brisbane River along transects of different land-use influences and provide baseline concentration data (HBCDDs and PFASs) in the sediments;
- Investigate and understand the influence of salinity on the behaviour of PPCPs and CUPs in estuarine waters as well as assess the contaminant sources;
- Understand PFASs migration in soil cores over 0-2 m depth at 0.5 m intervals at a FTG with a focus on the influences of soil physicochemical factors (organic carbon, pH, salinity and mineralogy) on the transport of PFASs in the soil cores;
- Estimate PFAS mass load in the bulk soil at a FTG.

1.6 Organisation of the thesis

The organisation of this thesis is presented on the schematic in Fig. 1.1. Also, the organisation is presented in bullet form as shown below, giving further detail for each chapter:

- **Chapter 1** presents background information on the thesis and outlines the research hypothesis, aims and objectives.
- **Chapter 2** presents a review of existing literature on PBDEs, PCBs, HBCDDs, PFASs, PPCPs and CUPs. It discusses the status of previous research work carried out in South-East Queensland as well as distribution and fate of these contaminants in soils/sediment from other locations around the world and the influences of physicochemical factors on soil-water and sediment-water partitioning.

- **Chapter 3** describes the study areas and experimental methods used during the work.
- **Chapter 4** presents data and discusses the distribution of PBDEs, PCBs and HBCDDs in sediment from the Brisbane River.
- **Chapter 5** presents data and discusses the occurrence and spatial distribution of PFASs in sediment and water from the Brisbane River.
- **Chapter 6** presents data on PPCPs and CUPs in surface waters from the Brisbane River estuary and discusses the spatial distribution and the influence of salinity and land-use applications on the distribution.
- **Chapter 7** presents data on PFASs transport in soil cores along a depth profile and also the dependence of soil-water partitioning on organic carbon, salinity, pH, and mineralogy to understand PFASs mobility along the soil depths.
- **Chapter 8** presents the overall summary of the key findings of this research and including recommendations for future studies.

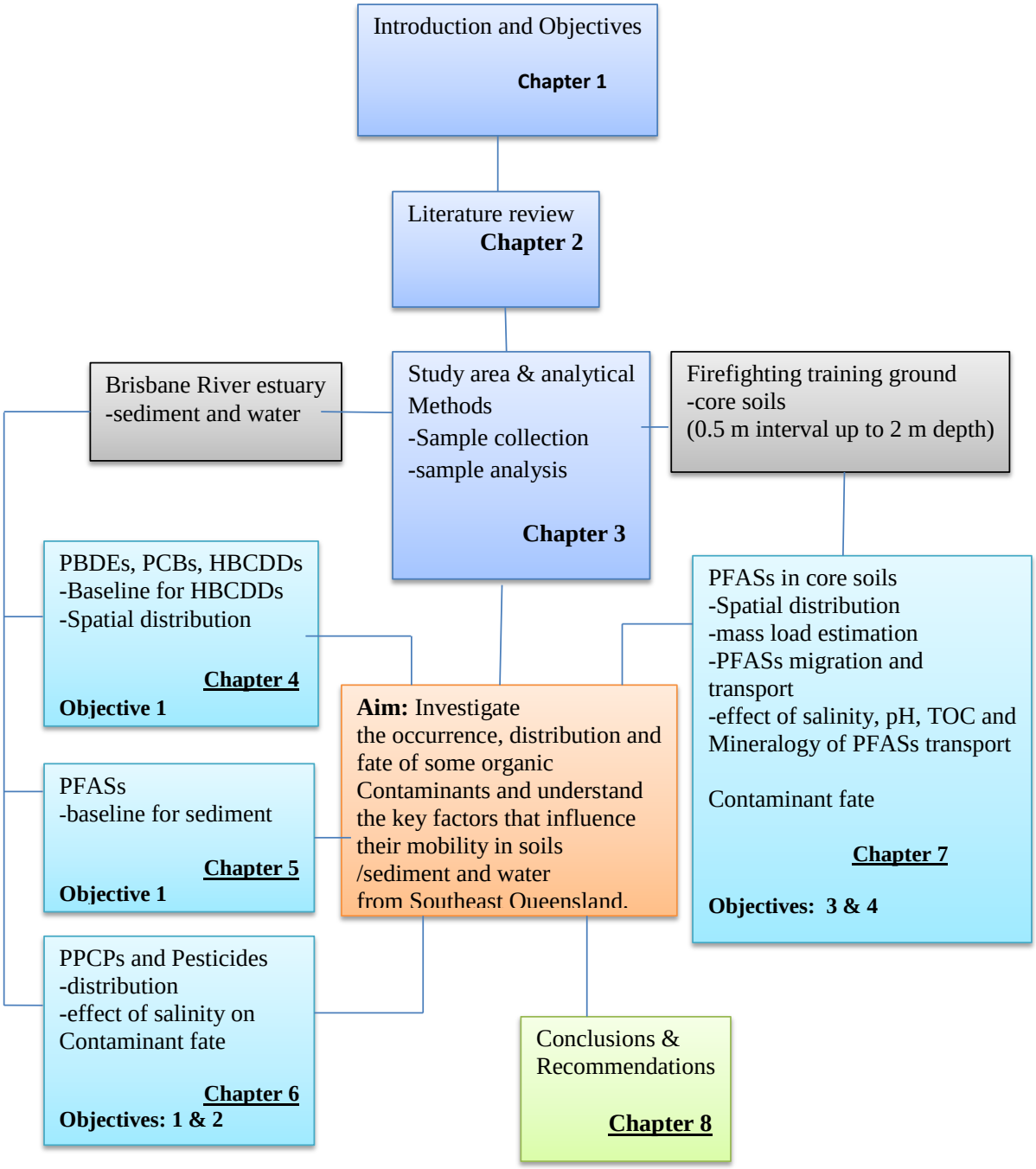


Fig. 1.1: A schematic showing the organisational structure of the thesis

Chapter 2: Literature Review

Overview

This chapter begins with general information on organic contaminants (Section 2.1) and reviews literature regarding the following specific organic contaminants: flame retardant pollutants (FR-Ps) and PFASs (Section 2.2). (The section discusses the structure, properties, uses and environmental relevance of each of PBDEs, PCBs, HBCDDs, and PFASs); Section 2.3 discusses the application of pharmaceuticals and personal care products (PPCPs) and their fate in receiving waters. Section 2.4 discusses the applications, environmental contamination pathways and the fate of current use pesticides (CUPs) in receiving waters while the transport and exposure routes of FR-Ps and PFASs were discussed in Section 2.5. Human health risks associated with FR-Ps and PFASs exposure, including some potential health risks associated with the exposure to PBDEs, PCBs, HBCDDs and PFASs were reviewed in Section 2.6. This section also highlights the concerns that underpin the investigation of these contaminants and the quest for understanding their fate in order to safeguard the environment. The influence of some physico-chemical properties of soil and sediment on the transport of organic contaminants were reviewed in Section 2.7 while the fate of organic contaminants in soils and sediments were also discussed under this section. Section 2.8 contains an up-to date review of the fate of FR-Ps and PFASs in Australia and other parts of the world. This section also identifies the research gaps and sets the basis for the investigation of the fate of the organic contaminants in water and sediments from the Brisbane River estuary as well as soils from a firefighting training ground in Brisbane that was previously impacted by PFASs laden AFFF. The analytical techniques used for monitoring organic contaminants were reviewed in Section 2.9. Finally, Section 2.10 highlights the research problems based on the information derived from literature and provides justification for carrying out this research work.

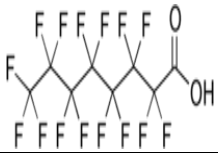
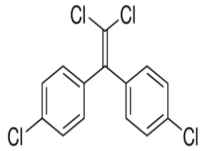
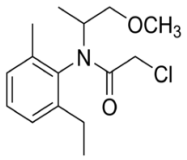
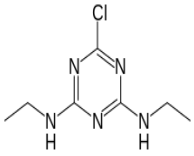
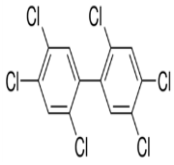
2.1 Organic contaminants

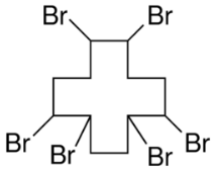
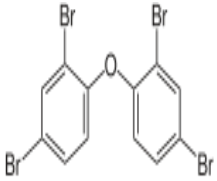
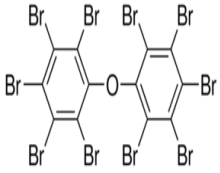
Organic contaminants include a wide range of organic compounds that can be harmful or potentially harmful to the environment and human health [14]. Bioaccumulation of some organic contaminants have been linked to cancer, reproductive defects, diabetes, learning disabilities and neurological disorders in humans and some animals [6, 7, 47]. The

"Stockholm Convention" [14], a global treaty, therefore, became effective on 12th May, 2004 to protect the environment and humans from the production and use of some contaminants (e.g. some PBDEs, PCBs, PFASs, pesticides) that are persistent and have detrimental effects on ecological life.

Initially, twelve organic compounds were identified by the Stockholm Convention and listed as persistent organic pollutants (POPs) for ban/restriction of production and usage. These were classified as the "dirty dozen" and are: nine pesticides (Aldrin, chlordane, DDT, dieldrin, endrin, heptachlor, hexachlorobenzene, mirex and toxaphene); two industrial chemicals (hexachlorobenzene and polychlorinated biphenyls); and by-products (polychlorinated dibenzo-p-dioxins and polychlorinated dibenzofurans). To date, the listing by the Stockholm Convention includes hexabromocyclododecanes (HBCDDs), polychlorinated biphenyls (PCBs), polybrominated diphenyl ethers (PBDEs), per-and polyfluoroalkyl substances (PFASs) and polyaromatic hydrocarbons (PAHs). Currently, other emerging contaminants, pharmaceuticals and personal care products (PPCPs) and current use pesticides (CUPs) are also been investigated to establish their health effects. These compounds have properties that enable them to enter environmental matrices (e.g. soils, sediment, water) by leaching out directly from their primary consumer products or from secondary sources such as waste streams with consequent implications on both humans and ecological life. Table 2.1 lists some of these groups of organic contaminants and includes some specific examples. Each of the groups of compounds has congeners, depending on the number of halogenated substitution, carbon-carbon chain and or functional group orientation.

Table 2.1: Examples of some organic contaminants and their potential health effects on ecological life.

Group of organic contaminant	Common name/abbreviation	IUPAC name	Chemical Structure	Uses and Effects
PFASs	PFOS	perfluorooctane sulphonic acid		Stain repellents in fabric, paint, paper and leather. Also an ingredient in AFFF. Risk of chronic kidney diseases
PFASs	PFOA	Perfluorooctanoic acid		Teflon liners in non-stick pans and cookware and AFFF. A potential carcinogen to wildlife
Organochlorine pesticide	DDT	1,1-Dichloro-2,2-bis(4-chlorophenyl)ethene		Used as pesticide. Even though it is banned, its use is restricted for special programmes such as mosquito control. It is highly toxic and a carcinogen
CUPs	metolachlor	(RS)-2-Chloro-N-(2-ethyl-6-methyl-phenyl)-N-(1-methoxypropan-2-yl)acetamide		Herbicide used to control broadleaf weeds and grass in corn, soybean, peanuts etc. Potential genotoxic effects in tadpoles as well as human lymphocytes.
CUPs	simazine	6-Chloro-*N*,*N*′-diethyl-1,3,5-triazine-2,4-diamine		Herbicide used to control broadleaf weeds and annual grass in berry crops, vegetables, algae in aquarium. Potential dermatitis upon occupational contact, muscular tremor in sheep
PCBs	PCB-153	2,2′,4,4′,5,5′-Hexachlorobiphenyl		This is one congener of 209 PCBs. Used as insulating fluids in transformers and capacitors, flame retardants, pesticides,

				paints, wooden floor furnishes etc. It can also be produced as a by-product during combustion. Causes liver diseases and lessoned immune response.
HBCDDs	HBCDD	1,2,5,6,9,10-Hexabromocyclododecane		Used as a flame retardant particularly in polystyrene foam as thermal insulation in buildings. Also used as upholstered furniture, automobile interior textiles, electric and electronic equipment. Causes potential reproductive toxicity.
PBDEs	BDE-47	2,2′,4,4′-tetra-bromodiphenyl ether		A component sof penta-BDE mixture used in polyurethane foams (upholstery furniture, mattresses, bedding and carpet underlay). Potential effects on thyroid, liver and neurobehavioral development..
PBDEs	Deca-BDE or BDE-209	2,3,4,5,6-Pentabromo-1-(2,3,4,5,6-pentabromophenoxy)benzene		The heaviest PBDE of 209 congeners. Used as flame retardants in plastics, foam and textiles, computers, televisions, carpets, furniture etc. Can accumulate in human blood and breastmilk. Effects on thyroid function reduced male fertility and damaged ovarian development.

2.2 Flame retardant pollutants (FR-Ps) and PFASs

PBDEs, HBCDDs, PCBs and PFASs have become the contaminants of high interest due to environmental and human safety concerns [48]. Apart from the application of PFASs compounds as stain and oil repellents in consumer products such as food packaging materials and textiles, PFASs have also been applied in aqueous film-forming foams (AFFF) for firefighting. PBDEs and HBCDDs have gained wide applications in household appliances and automobiles [49-51] while PCBs have previously been used as floor furnishers, transformer oils and coolants in capacitors. The United Nations Environmental Programme (UNEP), under the Stockholm Convention has listed HBCDD and some congeners of PBDEs (octa-BDE and penta-BDE) as Persistent Organic Pollutants (POPs) [52]. Flame retardants exhibit quenching properties that inhibit the combustion processes in flames and as a result slows down the process of burning or even prevent fires [53, 54]. These properties have promoted their use in many household materials (e.g. television sets, computers, carpets). Flame retardants such as PBDE and HBCDD are not covalently bonded to the materials to which they are added and therefore can easily be released into the environment [55]. Once in the environment, these flame retardants are environmentally persistent and bio-accumulative [56-58]; lypophilic, labile and undergo long range transport [59-61]. PBDEs for example, have been detected in human samples in Ghana where there is no historic production of flame retardants [62]. Across the world, and including some parts of Australia, these contaminants have been detected and measured in water [26, 63], sediment [18, 51], soil [64, 65], air [16, 66], and human milk [67, 68].

2.2.1 Polybrominated diphenyl ethers (PBDEs)

The PBDEs constitute a wide range of congeners which were incorporated as reactive or additive flame retardant compounds in polymers for applications in a range of commercial products such as furniture, upholstery, electrical/electronic devices, plastics, textiles etc. [69]. The presence of PBDEs in environmental matrices have been reported to disrupt oestrogen and thyroid hormones [70-72] as well as reduced male fertility and ovarian development [73] in some biological organisms. Humans can be exposed to PBDEs via ingestion and inhalation. The world's production of PBDEs was estimated at 67,400 metric tons/year with America contributing about 50% plus and Europe, 12% [69]. This is of human and ecological concern as these PBDE congeners can leach out from the primary products and

contaminate the environment. Chemically, the PBDEs are characterized by 2 to 10 bromine atoms attached to diphenyl ether as shown in Fig. 2.1

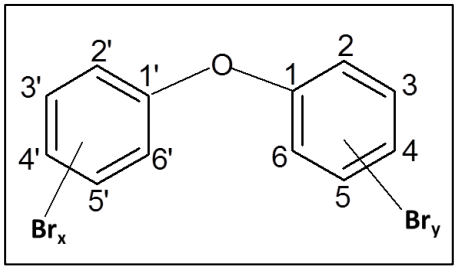


Fig. 2.1: A general chemical structure of PBDEs showing the 10 possible homolog sites on the rings where Br can attach.

Where x + y = 1 to10 (x + y =1; defines monobrominated diphenyl ether). There are therefore 10 PBDE homologs: mono-, di-, tri-, tetra-, penta-, hexa-, hepta-, octa-, nona-, and deca- which have 3, 12, 24, 42, 46, 42, 24, 12, 3, and 1 congeners, respectively. For example, the penta-brominated diphenyl ether homolog has 46 congeners (structural isomers). There are therefore 209 theoretical congeners of PBDEs. However, fewer than 209 exist due to instability and consequent debromination of some congeners [69] under favourable environmental conditions.

Commercial PBDEs are classified under three main homologs, namely: commercial pentabromodiphenyl ether (penta-BDE), commercial octabromodiphenyl ether (octa-BDE) and commercial decabromodiphenyl ether (deca-BDE) mixtures. The commercial homologs are not pure substances containing the specified number of bromine atoms, rather mixtures of congeners -with congeners of the specified number of bromine atoms contributing the highest percentage in the mixture. The commercial mixture penta-BDE means the mixture contains penta-BDE as the major component. For example, commercial penta-BDE contains; tetra-BDE (24-38%), penta-BDE (50-62%), hexa-BDE (4-8%) and tri-BDE (0-1%). The commercial octa-BDE mixture contains hexa-BDE, hepta-BDE, octa-BDE and nano-BDE homologs with traces of deca-BDE. Commercial deca-BDE is composed of deca-BDE (97%) and traces of nano-BDE and octa-BDE homologs.

The environmental fate of PBDEs can be influenced by their physical and chemical properties (Table 2.2) as well as the type and properties of a particular environmental matrix. More volatile PBDEs (less brominated) dominate in the vapour phase while heavier (eg. BDE-209) predominates on particulates [74]. The higher PBDE (more bromine atoms) are thus less mobile in the environment due to their low volatility and water solubility [51]. They therefore strongly adapt to environmental compartments such as sediment and soils.

Table 2.2: Physical and Chemical properties of commercial PBDEs [75]. Data extracted from ATSDR (2015)

Property	**Penta-BDE**	**Octa-BDE**	**Deca-BDE**
IUPAC name	1,2,4-tribromo-5-(2,4-dibromophenoxy) benzene	2,2',3,4,4',5,5',6-octabromodiphenyl ether)	2,3,4,5,6-Pentabromo-1-(2,3,4,5,6 pentabromo-phenoxy) benzene
Trade names	DE-60F,DE-61,DE-62	DE-79	DE 83R, Saytex 102E
Molecular weight (g/mol)	564.69	801.47	959.22
CAS no.	32534-81-9	32536-52-0	1163-19-5
Physical description	Pale yellow liquid	Off-white powder	Off-white powder
Melting point (^{o}C)	-7 to -3	85 to 89	290 to 306
Boiling pt. (^{o}C)	>300	>330	>330
Density (g/mol)	2.28 at 25 ^{o}C	2.76	3.0
Water solubility (ug/L)	13.3 (commercial)	1.98(heptabromodiphenylether component)	<0.1[76]
logK_{OW}	6.64 to 6.97	6.29 (commercial)	6.265
logK_{OC}	4.89 to 5.10	5.92 to 6.22	6.80
Vapour pressure,25^{o}C (mmHg)	2.2 x 10^{-7}- 5.5 x 10^{-7}	9.0 x 10^{-10} to 1.7 x 10^{-9}	3.2 x 10^{-8}
Henry's const. at 25^{o}C(atm.m^{3}/mol)	1.2 x 10^{-5}	7.5 x 10-8	1.62 x 10^{-6}

PBDE compounds are ubiquitous and have been traced in both environmental and human matrices. A food basket survey in the USA has detected various PBDE congeners in varieties

of fish, meat and dairy products [77] with BDE-47, 99, 100, 153,154 showing percentage detections of 95.8, 100, 95.8 and 95.8 % respectively in the fish samples.

2.2.2 Polychlorinated biphenyls (PCBs)

PCBs were identified as persistent organic pollutants and therefore banned in 1979 by the US congress under the Toxic Substances Control Act [78, 79]. Subsequently, PCBs was banned by the Stockholm convention in 2004. Earlier, they were produced for commercial use between 1930s and late 1970s due to their low flammability and insulating properties. PCBs have high flash points (170-380^{o}C) and therefore used as fire resistant compounds [80]. Accordingly, PCBs have been used in industrial products such as sealants in buildings, ink and paint additives, coolant and insulating fluids (transformers and capacitors), wood floor furnishes and adhesives as flame retardants.

Similar to the PBDEs, there are 209 potential congeners in ten homologous groups. The general formula is $C_{12}H_{10-n}Cl_n$ (n=chlorine atoms) and chemical structure is given as shown in Fig. 2.2.

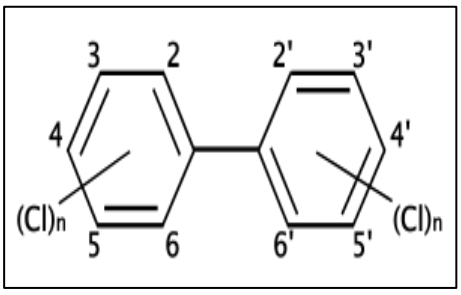


Fig. 2.2: A general chemical structure of PCBs showing the 10 possible homolog sites on the rings where Cl can attach.

PCBs have been produced for commercial applications in the US under the trademark Aroclor [81]. Aroclor is assigned a four-digit identification number; the number of carbon atoms represented by the first two digits and the last two digits representing the degree (percentage) of chlorination. For example, Aroclor 1260 simply means that there are 12 carbon atoms with a 60% degree of chlorination. Particularly, aroclor 1254 has been used extensively in many products, including capacitors, transformers, hydraulic fluids, pesticide extenders, and inks. In other countries such as Japan, Italy, France and Germany, PCBs were manufactured under the trademarks Kaneclor, Fenclor, Pyralene and Clophen, respectively

[82]. Thus there is a potential environmental exposure due to historic products containing PCBs such as aroclor 1254. PCBs can resist physical and chemical breakdown and are therefore persistent in environmental matrices such as air, water, soil and sediment. Humans are exposed through direct or indirect inhalation, ingestion and dermal contact with PCB containing products. The classification of PCBs as human carcinogens in 2013 by the International Agency for Research on Cancer [83] is alarming and therefore warrants total monitoring of all environmental compartments, including sediments. Toxic effects, includingimmune system disorders, behavioural alterations and reproduction defects have also been observed [78].

The extent of partitioning and environmental fate of PCBs also depend on the physical and chemical properties of each congener [84]. The physical and chemical properties of PCBs are presented in Table 2.3. For example, the heavy decachlorobiphenyl adsorbs onto atmospheric particles whilst the lighter congeners occur in the atmosphere as gaseous components [84]. If these PCB laden atmospheric particles settle on hard surfaces, they can be washed-off into river systems during precipitation and subsequently adsorbed onto sediments. Also, melting point and lipophilicity increases with increasing chlorine atoms whilst vapour pressure and water solubility decreases (Table 2.3). These properties, which favour the adsorption of PCBs onto particles make sediment a good matrix for environmental monitoring of PCB contaminants.

Table 2.3: Physical and Chemical properties of some PCBs. Data extracted from Li et al., (2003) [84]

Property	PCB Congeners							
	PCB-28	PCB-52	PCB-101	PCB-118	PCB-138	PCB-153	PCB-155	PCB-180
IUPAC name	2,4,4'-trichlorobiphenyl	2,5,2',5'-tetrachlorobiphenyl	2,4,5,2',5'-pentachlorobiphenyl	2,4,5,3',4'-pentachlorobiphenyl	2,3,4,2',4',5'-hexachlorobiphenyl	2,4,5,2',4',5'-hexachlorobiphenyl	2,2',4,4',6,6'-hexachlorobiphenyl	2,3,4,5,2',4',5'-heptachlorobiphenyl
CAS number	7012-37-5	35693-99-3	37680-73-2	31508-00-6	35065-28-2	35065-27-1	33979-03-2	35065-29-3
Melting pt. ^{0}C	58	86.5	77	110	79	103	113	112
$\Delta_{fus}S/J.K^{-1}.mol^{-1}$	56	46.1	53.6	56	56	56	45.3	56
Aqueous solubility, S_{wL} in mol/m^3	[a] 1.01E-3 (2) [b] 8.85E-4 (-13)	[a] 6.82E-4 (4) [b] 4.78E-4 (-30)	[a] 1.05E-4 (3) [b] 1.02E-4 (-3)	[a] 8.88E-5 (5) [b] 6.83E-5 (-23)	[a] 2.08E-5 (5) [b] 1.87E-5 (-10)	[a] 3.77E-5 (3) [b] 3.07E-5 (-19)	[a] 3.93E-5 (1) [b] 3.82E-5 (-3)	[a] 8.01E-6 (5) [b] 1.32E-5 (65)
Vapour pressure, P_L in Pa	[a] 2.36E-2 (2) [b] 2.70E-2 (15)	[a] 1.06E-2 (2) [b] 1.20E-2 (13)	[a] 2.41E-3 (2) [b] 2.46E-3 (2)	[a] 8.93E-4 (2) [b] 9.91E-4 (11)	[a] 5.39E-4 (2) [b] 5.63E-4 (4)	[a] 5.29E-4 (2) [b] 6.06E-4 (15)	[a] 3.31E-3 (2) [b] 3.49E-3 (6)	[a] 1.32E-4 (2) [b] 1.08E-4 (-18)
Octanol-water partition coefficient, K_{OW}	[a] 3.58E+5 (3) [b] 4.61E+5 (19)	[a] 1.05E+6 (4) [b] 8.10E+5 (-18)	[a] 1.42E+6 (4) [b] 2.16E+6 (52)	[a] 3.09E+6 (2) [b] 4.87E+6 (58)	[a] 9.98E+6 (5) [b] 1.64E+7 (64)	[a] 5.11E+6 (5) [b] 7.44E+6 (46)	[a] 2.29E+7 (4) [b] 1.53E+7 (-33)	[a] 1.93E+7 (5) [b] 1.45E+7 (-25)
Henry's constant, H (Pa.m^3/mol)	[a] 33.1 (1) [b] 30.5 (-8)	[a] 28.2 (1) [b] 25.1 (-11)	[a] 31.4 (4) [b] 24.1 (-23)	[a] 32 (5) [b] 14.5 (-55)	[a] 39.5 (4) [b] 30.1 (-24)	[a] 25.0 (3) [b] 19.8 (-21)	[a] 76.5 (4) [b] 91.4 (19)	[a] 5.84 (4) [b] 8.13 (39)
Octanol-air partition coeffiecient, K_{OA}	[a] 8.58E+7 (2) [b] 7.05E+7 (-18)	[a] 1.65E+8 (2) [b] 1.65E+8 (0)	[a] 7.90E+8 (1) [b] 5.38E+8 (-11)	[a] 6.61E+9 (4) [b] 2.30E+9 (-65)	[a] 5.72E+9 (2) [b] 4.54E+9 (-21)	[a] 3.28E+9 (2) [b] 2.76E+9 (-16)	[a] 7.71E+8 (5) [b] 1.38E+9 (79)	[a] 1.37E+10 (1) [b] 1.46E+10 (7)
Solubility in octanol, S_{OL} in mol/m^3	- [b] 7.7E+2 (-)	[a] 735.3 (3) [b] 8.0E+2 (9)	- [b]5.3E+2 (-)	- [b] 9.2E+2 (-)	- [b] 1.0E+3 (-)	- [b] 6.7E+2 (-)	- [b] 1.9E+3 (-)	- [b] 6.3E+2 (-)

[a] Literature derived value at 25^{o}C (uncertainty) [84]; [b] Final adjustable Value at 25^{o}C (percentage of adjustment) [84].

2.2.3 Hexabromocyclododecanes (HBCDDs)

The IUPAC name is 1,2,5,6,9,10-Hexabromocyclododecane, with a structural formula given as shown in Fig. 2.3.

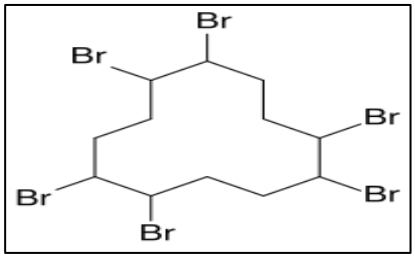


Fig. 2.3: A general chemical structure of HBCDDs showing the sites on the ring where Br can attach

HBCDDs have sixteen potential stereoisomers [85]. The commercial/technical HBCDDs however, consist mainly of three diastereomers; α-HBCDD (10-13%), β-HBCDD (1-12%) and γ-HBCDD (75-89%) [86]. The γ-HBCDD component is usually present in sediment at levels >90% of the total (α+β+ γ) HBCDD [69, 86]. At high temperatures above 160 ^{0}C, the technical HBCDD mixture undergoes tautomerization and hence affects the percentage composition of the diastereomers [87]. Therefore, only total HBCDD levels can be measured at higher temperatures. As a result of this chemical orientation, most previous studies have only measured total HBCDD thus making it difficult to assess environmental contributions arising from each diastereomer.

The HBCDDs are ubiquitous environmental contaminants that are also persistent due to their physical and chemical properties. The physical and chemical properties of HBCDDs which influence their environmental partitioning are summarised as: melting point (185-195 ^{0}C), vapour pressure (4.7 x 10^{-7} mmHg), density (2.24 g/cm^{3}), water solubility (0.0034 mg/L) and octanol-water partition coefficient, logK_{ow} (5.6) according to Lam *et al.,* (2009) [88] and the references therein. The low water solubility thus allows them to adsorb onto sediment when they are released into water bodies. The HBCDDs have widely been used as additive flame retardants in commercial products such as upholstery textiles, thermal insulators in building materials and electronics [86, 89]. As a result, they can leach out from these products into the environment.

The world's production of HBCDD in 2001 was 16700 metric tons, representing 8% out of 203,790 metric tons of all brominated flame retardants [69, 90]. In Australia, the importation of commercial HBCDD as powder granules for use in expandable polystyrene foams and polypropylene resins showed a decrease from about 90 tonnes (2006-2007) to about 60 tonnes between 2009-2010 prior to the cessation of imports in 2010 [89]. Regardless of the reduction in importations, it is expected that the concentrations of HBCDDs in environmental samples will continue to increase as the ban by the Stockholm Convention exempt the application of HBCDDs for wall insulation in the building industry until 2024 [48].
The environmental distribution of HBCDD according to literature does not follow a particular pattern. Lam *et al.,* (2009) identified inconsistencies in data patterns of HBCDDs based on the matrix type and the geographical study area. Time-series studies have therefore been proposed to fully understand the fate of HBCDDs in environmental matrices [91].

2.2.4 Per-and polyfluoroalkyl substances (PFASs)

The per-and polyfluoroalkyl substances (PFASs), previously referred to as per-and polyfluorinated chemicals (PFCs), are synthetic organic compounds produced since the 1950s and have been used as firefighting agents as well as protection against abrasion and stains in industrial and consumer products[92]. Other uses include stain protectors (water and oil repellents) in fabric, furniture, carpets and food packaging materials as well as widespread application as surfactants in aqueous film-forming foams (AFFF) for firefighting [10, 93]. PFASs incorporated in these consumer products can leach out and contaminate the wider environment due to their persistent and ubiquitous chemical characteristics.

Chemically, PFASs are made up of strong covalent C-F bonds with lipophilic (aliphatic chain group) and hydrophilic (functional group) end-groups, a property which makes them good surfactants as well as defines their ubiquitousness in the environment. When all hydrogen atoms attached to the C-C bond apart from the functional group carbon in a PFAS are replaced with fluorine atoms it is called a perfluoroalkyl substance (PFAA) whereas when the C-C chain has at least one hydrogen atom remaining in the PFAS structure then it is called a polyfluoroalkyl substance [94, 95]. Thus, while polyfluoroalkyl substances can undergo degradation, the highly electronegative characteristics caused by the fluorine atoms make PFAAs highly resistant to thermal, chemical and biological degradation and hence more persistent in the environment. Some PFAAs can bioacummulate and biomagnify in the

environment [40]. Depending on the type of functional group attached to the C-C chain, PFAAs can be classified as: perfluoroalkyl sulphonic acid (PFSA, formula: $F(CF_2)_nSO_3H$), perfluoroalkyl carboxylic acid (PFCA, formula: $F(CF_2)_nCO_2H$), perfluoroalkyl phosphonic acid (PFPA, formula: $F(CF_2)_nP(=O)(OH)_2$) or perfluoroalkyl phosphinic acid (PFPIA, formula: $F(CF_2)_nP(=O)(OH)$) [96]. The most environmentally relevant PFAAs are; however, the PFSA (e.g. perfluoroctane sulphonate (PFOS), perfluorohexane sulphonate (PFHxS)) and the PFCA (e.g. perfluorooctanoic acid (PFOA), perfluorohexanoic acid (PFHxA)) [15, 40, 97-99]. Fluorotelomer precursors such as 6:2 or 8:2 fluorotelomer alcohols [$F(CF_2)_nCH_2CH_2OH$; n= 6 or 8], 6:2 fluorotelomer thioether amido sulphonate (FtTAoS) and 8:2 fluorotelomer sulfonate (8:2 Fts) are polyfluoroalkyl substances that can break down upon their release into the environment to form PFCAs and PFSAs [100-102].

Thus, apart from direct releases of PFAAs from consumer products into the environment, the polyfluoroalkyl precursor compounds can undergo further breakdown to increase PFAAs concentrations in environmental matrices. It is worth-noting that even though Ti/SnO_2-Sb/PbO_2 anodes have been found to decompose PFOA in water electrochemically, no natural degradation pathways have been established for PFAAs [10]. This recalcitrant breakdown characteristics coupled with the production of about 3.3×10^6 kg of PFAAs such as PFOS in the USA (Minnesota) and Europe by 3M Company in 2000 [103] suggest that these compounds will be ubiquitous in consumer products and in turn, the environment. PFASs have since been detected globally in the environment, including biota, food and human fluids [10, 27, 99, 104]. There is also evidence of levels of PFOS and PFOA in the ranges of <LOQ-4.9 ng/L and 1.2-1.4 ng/L respectively in water samples from the Brisbane River in Australia [26]. Recent studies, although not exhaustive, have considered some PFASs as human carcinogenic candidates [8].

In order to ensure environmental and human safety, a global PFASs group, the organisation for economic co-operation and development (OECD) in collaboration with the united nations environmental programme (UNEP) under the framework of the strategic approach to international chemicals management (SAICM) have classified some PFASs as persistent, bio accumulative and toxic to humans and the environment [34]. Similarly, the production and use of PFOS and its precursors have been restricted as indicated by its listing as an Annex B compound in May 2009 under the Stockholm Convention [48]. In June 2015, Australia also

adopted the risk reduction approaches of OECD/UNEP to reduce the global impact of PFASs on the environment and human health [34]. Accordingly, Australia seeks to reduce the global impact of PFASs and embrace safer alternatives under four themes, namely "Regulatory Approach, Policy Approach, Voluntary Initiatives and Monitoring". Nonetheless, there is currently no data reflecting the status of PFASs contamination of sediment from several Australian rivers, including the Brisbane River.

2.3 Pharmaceuticals and personal care products (PPCPs)

Pharmaceuticals are therapeutic drugs used on humans or veterinary to treat diseases while personal care products (PCPs) are used for cosmetic purposes to improve quality of daily life [105]. PPCPs as they are collectively called mainly enter environmental matrices through wastewater treatment plants (WWTPs) via effluents or leakages from pipes carrying waste water for treatment. Pharmaceutical products (e.g. antibiotics, analgesics, blood lipid regulators, antiepileptic, cardiovascular, non-prescription antihistamines) are not often fully metabolized in the body and hence end up in the waste streams as unchanged compounds or metabolites through human and or/ veterinary excretion products [3, 4]. Similarly, PCPs (eg. insect repellents, UV-filters, anti-microbial agents) also enters the wastewater streams as they are mostly washed down during showers or directly into river bodies during recreational swimming. Unfortunately, most WWTPs are unable to efficiently remove all these contaminants and hence end up in water courses such as rivers to contaminate the aquatic environment as effluents are discharged directly into water ways [106]. Leachates from landfills can also serve as routes of PPCPs contamination in the environment since some expired or unused PPCPs are disposed as solid waste. When these contaminants get released into the aquatic environment, they can accumulate in sediment and soils, thus becoming secondary sources as the contaminants can be remobilized into water depending on the hydrodynamics, physico-chemical conditions or biological activity [3].

These emerging contaminants have recently gained global attention due to their presence in water bodies and their potential threat to aquatic life and humans [4, 106]. When PPCPs become available in receiving waters even at low concentrations, non-target aquatic fauna can be exposed leading to potential adverse effects to the aquatic organisms as well as humans via the food chain [107, 108]. It is worth noting that with the ever increasing population, globally, and the associated usage of PPCPs to treat diseases as well as improve

aesthetic quality of life, the use and subsequent release of these contaminants into the environment will potentially be on the increase. It is, therefore, important to investigate the fate of these contaminants as there is evidence of persistence of some of them in the aquatic environment although it is conceivable that some of them will breakdown through routes such as biodegradation, photo-degradation or adsorption to particles and subsequent sedimentation [109, 110]. For example, the pharmaceutical product carbamazepine is conserved in both fresh and saline waters; hence does not significantly breakdown [4, 108] along the water course. For estuaries such as the Brisbane River, the intrusion of seawater into fresh water creates a mixing interface between the river and the sea for some chemicals, where salinity can be used as a tracer to assess the conservative behaviour of contaminants [111]. The linear dependency of salinity gradient and contaminant concentration was used by Bester *et al*., (1998) to determine the behaviour of contaminants in a river estuary [112]. This engenders the understanding the fate of a contaminant as it travels along a salinity gradient and can be used to investigate ecological safety due to potential exposure to the contaminant.

2.4 Current use pesticides (CUPs)

In the quest to increase agricultural yield to meet the ever growing population demands, the application of CUPs to crops and animal farms has resulted in the contamination of environmental matrices including soils, sediment and water with its associated potential effects on ecological life, even in remote environments [113-116]. Regardless of the less persistence of CUPs compared to the legacy organochlorine pesticides which were earlier banned and or/ restricted by the Stockholm convention, the continuous inputs into receiving waters even at relatively low concentrations can affect the lives of aquatic fauna [5]. Release of CUPs (herbicides, insecticides, fungicides) into aquatic environments have been identified to be mainly via stormwater drains, surface run-off from parks and agricultural fields as well as effluents from WWTPs [106, 117]. Once released into receiving water bodies, these compounds, can be transported across a wide salinity gradient making them available in both fresh and marine environments. Although some CUPs can undergo biological or photolytic degradations, some are recalcitrant and can pose pseudo-persistent behaviour thus becoming conservative over a wider area and affecting aquatic life [118]. In South-East Queensland and other Australian states, stormwater is discharged into the river systems and is thus a potential route of pesticide contamination from agricultural fields, parklands and households with backyard lawns. The salinity gradient of the Brisbane River estuary can serve as a benchmark

to investigate the fate and behaviour of these contaminants as they will potentially experience mixing when they are released and travel along the river course

2.5 Transport and exposure routes of FR-Ps and PFASs

FR-Ps and PFASs which have been incorporated in domestic and commercial products such as electrical equipment, paints, transformer oils, polystyrene foams and carpets [119-121] are semi-volatile and can therefore be released into the environment from these products. They can be released into the environment through leachate from landfills, direct release from home/office consumer products or atmospheric transport and deposition from remote sources during precipitation. Fig. 4 shows a schematic of varying routes of environmental exposure to these contaminants.

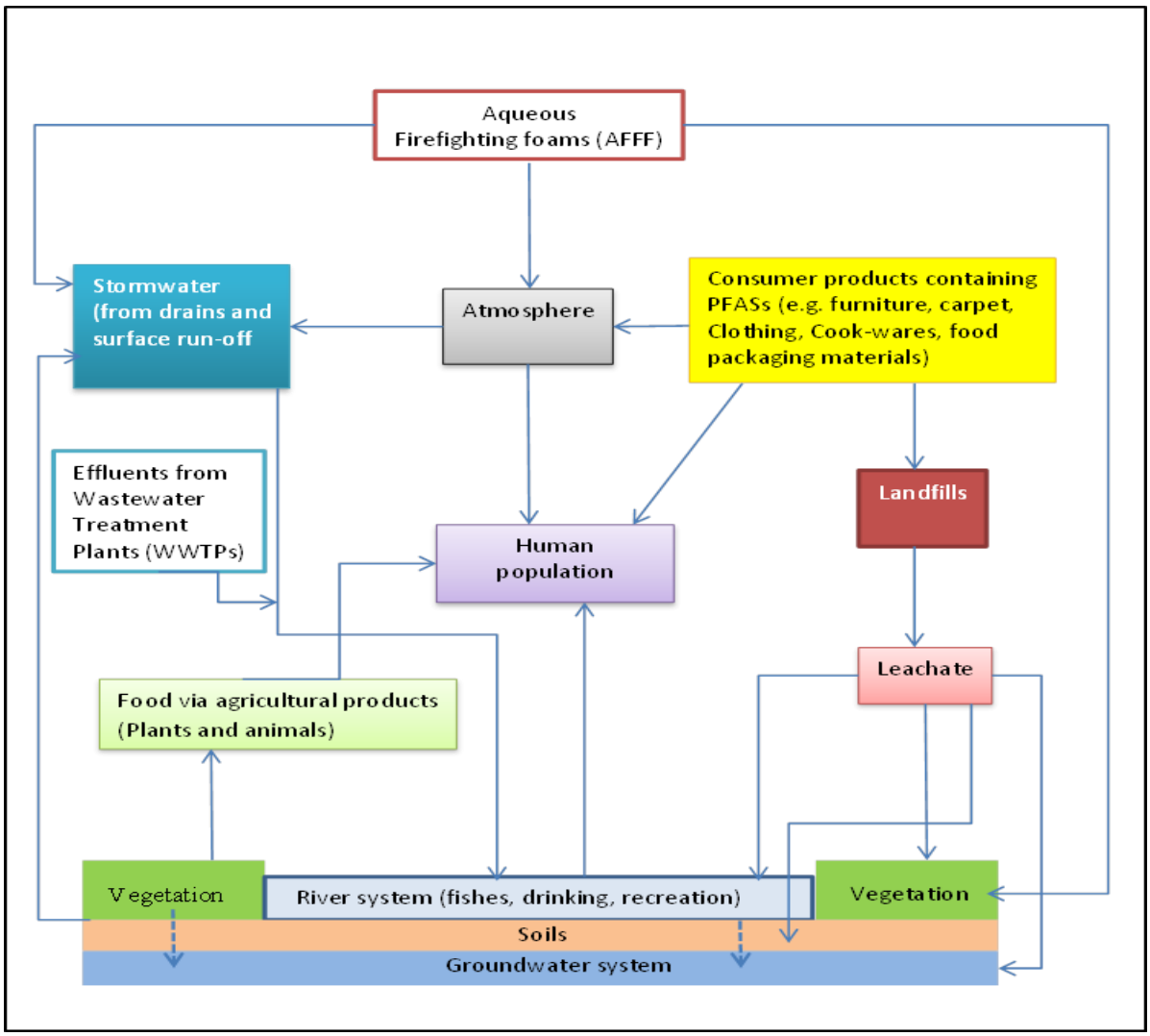


Fig. 2.4: Human exposure routes of FR-Ps and PFASs.

By means of atmospheric transport, when these compounds are dispersed in the air, they can find their ways into aquatic environments by first binding to fine particles of soil and dust on hard surfaces [6], which are subsequently washed-off by stormwater [122] and/or strong winds into receiving waters. Once these compounds are released into water, they are able to bind to sediments [123, 124] and are made available to aquatic organisms through feeding habits, thus finding their ways into humans through the food chain [125]. Sewage sludge used for soil improvement can also contain organic contaminants such as PBDEs and HBCDDs for example, and can contaminate agricultural lands when they are applied to improve soil quality [74]. The contaminants can thus be absorbed directly by plants or washed-off into water systems from the top soils. Stormwater drains and leachate from landfill sites offer other environmental exposure pathways to these organic contaminants.

Due to bioaccumulation and biomagnification potentials, the presence of these compounds in the environment even at very low concentrations is worrying. Also alarming is the fact that FR-Ps can undergo degradation to produce lower molecular weight congeners, which are more toxic due to higher mobility [22, 126]. Some studies have shown the presence of these contaminants in human fluids such as serum and breastmilk [127] [92]. In Australia, for example, Karrman *et al.*, (2006) observed increasing bioaccumulation of PFASs, particularly PFOS (20.8 ng/ml) and PFOA (7.6 ng/ml) in pooled serum with increasing age among 3802 residents [128]. The study provides evidence for the persistence of these organic contaminants.

Also, the use of AFFF for firefighting is a source of PFASs contamination in soils, particularly at firefighting training grounds (FTG). Some studies have shown PFASs contamination in soils, sediment, surface water, biota and even groundwater [21, 40, 44-46] as a result of AFFF applications at FTGs. In Australia, for example, AFFF have been applied for firefighting since the 1970s and increase the potential for PFASs contamination of soils, river bodies and even groundwater.

2.6 Human health risks associated with FR-Ps and PFASs exposure

The presence of organic contaminants in the humans or animals can impact toxic effects by altering some hormones. For example, when brominated flame retardants enter the thyroid system, the bromine atoms compete and replace the iodine atoms at their binding sites due to the similarities in their chemical properties and this leads to the malfunctioning of the thyroid system [129].

Clinical toxicological studies of penta-BDE on rats gave indications of diarrhea, tremors of forelimbs and changes in thyroid size and histology [130]. Adverse liver effects in rats after a dosage of 10 mg/kg body weight of octa-BDE have also been observed [130]. Even though on-going toxicity studies of PBDE to humans are inconclusive, there are indications of several human health effects including cancer, neurodevelopmental and developmental defects as studies on animals have proved positive [55, 131]. PBDEs have been found to potentially produce thyroid stimulating hormones which enhances diabetes in humans [132]. The Agency for Toxic Substances and Disease Registry (ATSDR) have also reported liver tumors in rats, caused by deca-BDE and also neurobehavioral alterations [75].

Similarly, PCBs have been classified by the International Agency for Research on Cancer (IARC) as a human carcinogen [83]. Accordingly, the National Industrial Chemicals Notification and Assessment Scheme (NICNAS) has phased out PCB in Australia [133]. Earlier reports of developmental delays and behavioral problems observed in children born seven years after the occurrence of a PCB rice oil contamination in Taiwan (1979) were attributed to mother-child transfer [48, 134]. Other health effects including skin and eye irritation have been linked to PCB contamination [134]. Dioxin, a by-product of the combustion of flame retardants such as PCBs can impact adverse health effects in humans by binding to the steroid hormone thus leading to a molecular change in the receptor and affecting drug response [135].

Although toxicity studies on HBCDD contamination are still on-going, previous studies have linked increased weights of liver, thyroid and pituitary glands in rats to HBCDD contamination [86, 89, 136]. Induced cancer in rats through non-mutagenic mechanisms due to HBCDD exposure have also been reported [55]. In Australia, HBCDD is recommended for

classification as a potential health risk contaminant to reproduction, potentially to breastfed babies [89].

The toxicity of PFASs on humans is inconclusive. However, PFASs have been implicated in human health risk effects, including immunotoxicity, hepatotoxicity, neurotoxicity and reproductive damage [8-10]. PFASs are also potential human carcinogens [8].

2.7 Influence of some physico-chemical properties of soil and sediment on the transport of organic contaminants

Soil and sediment are important environmental matrices for the distribution of organic contaminants. The soil or sediment physico-chemical properties at a particular study site can influence the sorption processes and subsequent transport of particular organic contaminants. Some of these properties include organic carbon, pH, salinity and mineralogy compositions. These properties can either influence the fate of contaminants independently or depending on the composition of a particular soil or sediment, a combination of two or more of such properties.

Organic carbon

Organic carbon (OC) is the most important soil/sediment content for the sorption of organic contaminants since the OC is the most non-polar solid phase in soils and sediments [137-139]. The hydrophobicity of the organic contaminants allows their sorption to the OC in soil/sediment particle surfaces or inside aggregates [140, 141]. Nonetheless while some studies have found OC to influence the distribution of organic contaminants in soils and sediment [141-143], other studies have found no such relationships and suggest that sorption could be controlled by a combined effect of other soil/sediment properties as well as fresh input, depositional mechanisms and mixing [144, 145]. For example, while OC fraction showed a positive correlation with the sorption of some PFAAs [142, 146, 147] and PBDEs [143], other studies showed poor correlations of OC and the sorption of PBDEs [144] and PFAAs [145]. Notwithstanding, OC has been a significant soil/sediment component for the adsorption of particularly most non-ionic organic contaminants [145].

pH

The pH of soil and sediment is another significant factor that can influence the sorption of organic contaminants. The solution pH thus plays a role by influencing the charged particles on the surface properties of the soils and sediment. As the solution pH increases, the net negative charge density on the soil/sediment surface increases thus increasing cationic adsorption to the soil/sediment surfaces [148]. This effect can control the sorption behavior of ionisable organic contaminants such as PFASs which have hydrophilic end groups [142, 149]. In effect, increased pH will promote the adsorption of anionic PFASs compared to cationic PFASs. Nonetheless, while Kwadijk *et al.,* (2013) observed increased sorption of PFOS in sediment from pH 4 to 6, Millinovic *et al.,* (2015) observed no relationship for the sorption of PFOS and PFOA when the pH was increased from 4.6 to 8.

Salinity

Salinity has been reported to influence the sorption of organic contaminants in soils and sediment particularly when OC is very low or absent due to salting out effect [150]. This effect occurs when the aqueous solubility of organic compounds is reduced due to the presence of dissolved ions [147]. Thus, in the absence of OC, salinity creates electrostatic charges on the soil/sediment surface to increase sorption of organic contaminants. You et al. (2010) observed an increase in the adsorption of PFOS to sediment with increasing salinity.

Mineralogy

The soil/sediment mineralogy can also influence the sorption and transport of organic contaminants particularly at study sites with heterogeneous mineral compositions. For example, while clay content has been found to increase adsorption of contaminants to soils and sediment, high quartz content are poor sorption compartments for the organic contaminants. Clay surfaces such as alumino-silicate clays provide negative charges for adsorption of organic compounds [151]. The presence of metal oxides in soils and sediment can also influence the transport and fate of contaminant. Metal oxides (e.g.Al_2O_3, Fe_2O_3) in soils and sediment can get hydroxylated when exposed to water and cause the net charge on the surface to become either negatively charged at high pH or positively charged at low pH based on the extent of protonation [152, 153].

2.8 Fate of FR-Ps and PFASs in Australia and other parts of the world

Flame retardants have been identified as compounds of high concern due to their ability to infiltrate the food chain and pose hazards to humans [154, 155]. Waste disposal is one of the anthropogenic routes of environmental exposure to FR-Ps and PFASs. The additive chemicals leach out when consumer products are disposed of as domestic wastes to landfills and incineration [135] and consequently deposit in air and dust particles, water, soil, and sediment matrices. The persistent nature of FR-Ps and PFASs coupled with their potential health risk along the food chain makes it necessary to assess the spatial distribution of these compounds and investigate the mechanisms influencing their transport particularly in soils, sediment and water as contaminant sinks.

2.8.1 PBDE contamination

The exposure of FR-Ps due to their semi-volatile properties is evident as BDE-47, a very toxic PBDE congener, was detected in 100% of air samples collected in some selected living rooms within Brisbane, recording a median concentration of 25 pg/m^3 [16]. Another study on human exposure in Australia shows that milk from primiparae mothers and blood serum collected between 1993-2009 were found to contain PBDEs [156]. Correlation of the concentrations of PBDEs in breast milk and faecal samples of babies were also observed in the Australian population [157]. These observations are indicative of the bioaccumulation of PBDEs in the Australian population. However, these studies have not sought to trace the sources of these contaminants. It is therefore necessary to carry out a holistic monitoring of all environmental matrices for these persistent and toxic contaminants.

Sediments and soils serve as a sink for these contaminants and present a good matrix for environmental monitoring [18, 65]. In Australia, not much work has been carried out on sediment samples in particular. However, PBDEs were detected in freshwater, estuarine and marine core sediment samples collected throughout Australia in a maiden study [25]. In that study, only nine sampling points were used to represent Queensland sediment which could not reflect a comprehensive status of PBDE in the Brisbane River. Furthermore, samples were not collected at most tributaries that drain into the Brisbane River. In the maiden study, conducted by Toms *et al.,* (2008), the highest $\sum_7$PBDE (BDE-47, 99, 100, 153, 154, 183, 209) concentrations, 60.9 ng/g dry weight were measured in estuarine sediment from Port

Philip Bay, whilst $\sum_7$PBDE (BDE-47, 99, 100, 153, 154, 183, 209) of 4.42 ng/g dry weight was detected for estuarine sediment from the Bremer River [25] in South-East Queensland. The relatively high concentration (31 ng/g dry weight) of BDE-183 observed in sediment from Port Philip Bay in their study is an indication of a possible point source input. In contrast, BDE-183 concentrations in freshwater sediment from the UK were lower, 0.09-0.12 ng/g dry wt relative to BDE-47 and BDE-209 in sediment samples; 0.69-1.29 ng/g dry wt and 1.09—3.44 ng/g dry wt. respectively [64], suggesting an historic input or possible degradation of BDE-209. It is also alarming to observe that BDE-209 is the most dominant PBDE in the studied Australian sediments since BDE-209 can potentially degrade to form lower and more toxic congeners. Perhaps, the high octanol-water partitioning coefficient (K_{ow}) for BDE-209 might have accounted for its persistence in sediment as it is less soluble compared to other lower congeners.

A recent historic time-series study between 1980-2014 of surficial sediment from Sydney in Australia showed a continuous increase of commercial PBDE congeners, particularly in 2014 [18], regardless of the ban on importation in 2005 [16, 155]. According to Drage *et al.*, (2015), the concentrations of $\sum_6$PBDE (BDE-47, 99, 100, 154, 153, 183) in composite surficial sediment samples were lower than that of BDE-209 in the period 1990-2014. It is possible that BDE-209 in the sediment is degrading to yield the lower congeners. Notably, in 2014, the concentrations at all sampling points were between 0.65-2.5 ng/g dry wt ($\sum_6$PBDE) and 21-65 ng/g dry wt. (BDE-209). BDE-209 concentrations in the study by Drage *et al.,(*2015) were; however, comparable to the BDE-209 concentration (2.1-39.9 ng/g dry wt) that was detected in sediment from the River Cinca, Spain [124]. Deca-BDE (BDE-209) represent about 75% of all PBDEs incorporated as commercial flame retardants [135].

To assess the direct environmental impact of an automobile shredding and metal recycling facility, dust and blood samples from nearby workers and soil samples collected at a depth of 15 cm within 100 m distance around a metal recycling facility in Brisbane, Australia were analysed. The study measured soil concentration of $\sum_{10}$PBDE in the range 29-796 ng/g dry wt [65]. Concentrations in soil and dust were found to be directly impacted by the nearby recycling facility; however, concentrations in blood samples did not show such correlations and may be due to background variations via diet and home environments. Thus, different environmental exposures may have contributed to varying bioaccumulation levels in human

samples. In contrast, a study by Abdallah *et al.*, (2013) on composite soil samples collected at a depth of 10cm from New South Wales in Australia, reported lower levels of PBDE contamination. PBDE concentrations (ng/g dry wt) in the soil samples were predominantly deca-BDE, ranging between 0.037 to 1.0 ng/g dry wt.

Elsewhere, Su *et al.*, (2015) detected BDE-209 concentrations of 3.96-327 ng/g dry wt. in sediment samples from the River Hunhe in Southeast China. The BDE-209 concentrations were higher at the lower stream of the Hunhe River, recording a mean of 148 ng/g dry wt. Continuous environmental input from sewage and industrial effluents is thus suggested as possible contamination source, particularly downstream of the Hunhe River [158]. Similarly, sediment samples from Monzon, an industrialized town in Spain along the River Cinca show $\sum_7$PBDE (BDE-47, 100, 118, 154, 153, 183, 209) concentrations in the range, 2-42 ng/g dry wt [124]. Higher deca-BDE were detected in other parts of the world [49, 58]. However, in Switzerland, relatively lower concentration of BDE-209 in the range 0.1-5.1 ng/g dry wt representing 59-88% of $\sum_7$PBDE (BDE-28, 47, 99, 100, 153, 183, 209) was detected in twelve sediment samples from Lake Thun [159].

2.8.2 HBCDD contamination

While there has not been any report on HBCDD levels in sediment from South-East Queensland, few studies have indicated the contamination of HBCDDs in the Australian environment. Concentrations of HBCDD in surficial sediment from Sydney, Australia show a sharp increase from the late 1990s to 2014 [18]. In 2014, ∑HBCDD (α,β,γ) concentrations in the studied sediment were 1.8 to 5.3 ng/g dry wt compared to 0.12 to 2.9 ng/g dry wt in the early 1990s [18]. This trend is however reflective of the high importation and use of about 90 tonnes of commercial HBCDD between 2006 to 2007 [18]. Urbanization and potential leachate from historic landfill sites and/or run-off were the likely sources of ∑HBCDD (α,β,γ) in the Sydney estuarine sediment. Fresh water sediment collected from the UK in 2009 also measured 1.3 to 5 ng/g dry wt ∑HBCDD (α,β,γ) concentrations [64], similar to the levels measured in surficial sediment from Sydney, Australia. However, sediment samples collected between 2000-2001 in Europe, were found to contain higher HBCDD concentrations; 0.2-950 ng/g dry wt (Belgium), <0.8-9.9 ng/g dry wt (Netherlands), <2.4-16780 ng/g dry wt (UK) and <1.7-12 ng/g dry wt (Ireland) [160]. HBCDD diastereomers were also detected in the range <0.0002-5.6 ng/g for 17 Australian soil samples collected

between 2002-2003 [43]. It is expected that the concentrations of HBCDD in the environment will continue to increase as the ban by the Stockholm Convention exempt the application of HBCDDs for wall insulation in the building industry until 2024 [48]. This also makes it necessary to obtain background data due to HBCDD contamination in environmental samples from South-East Queensland including samples from the Brisbane River estuary.

Apart from sediment and soils, a study conducted in Australia between 1993-2009, measured a maximum HBCDD concentration of 19 ng/g lipid wt. in human milk samples [156]. In Spain, HBCDD concentrations, 3-188 ng/g lipid wt. were also detected in breast milk sampled between 2006-2007, reflecting a daily infant dietary intake of 175 ng/Kg body wt./day [161]. In a related study on human milk in Stockholm between 1980-2002, a time trend increment of about 5 magnitude was observed; 0.17 pmol/g fat (1980) and 1.0 pmol/g fat (2002) for HBCDD [162]. HBCDD has also infiltrated the food basket as shown by a survey in the US, measuring HBCDD concentrations of 0.023 ng/g in canned beef chili and 0.593 ng/g in canned sardines [163]. HBCDDs are thus present in the environment hence the need for continuous investigations of environmental matrices.

2.8.3 PCB contamination

PCBs have been screened in Australian blood serum samples based on a 2003/2009 data subjected to a Ritter population pharmacokinetic model. The outcome suggests a 30- year elimination half-life for PCB-74 [164]. However, the extent of PCB contamination in the Australian environment, notably in sediment, has been given limited attention to date. As early as the 1970s, PCB contamination has been detected in sediment and biota from the Brisbane River estuary at significant concentrations. A collation of data on PCBs by McMahon (1975) indicates PCB contamination of the Brisbane River sediment up to a high concentration of 35 mg/Kg [165]. This study however, did not analyse PCB congeners but only reported total PCB per the brand names. Studies of eight (8) sediment samples collected from the Brisbane River estuaries in the 1980s have reported PCB concentration in the ranges: ND-54 ng/g and ND-58 ng/g for PCB compounds with 54% and 60% chlorination respectively [24]. This earlier study; however, represents a small sampling size and did not account for specific PCB congeners and therefore was not indicative of current contamination trends in the sediments from South Eastern Queensland. In spite of this observation, PCBs can undergo sedimentation in receiving waters and adhere to sediment particles. It is

therefore necessary to evaluate the current status of PCBs contamination in the Brisbane River and report the concentrations of specific congeners.

However, regardless of the ban on production of PCBs in 1979 by the USA and subsequently by the Stockholm Convention, to which Australia is a signatory, PCBs are still persistent in the environment [134]. In Europe, Karzani *et al.*,(2015), reported concentration of $\sum_7$PCB (PCB-28, 52, 101, 118, 138,153, 180) in sediment samples from the Durance River and Berre lagoon in the range: 0.03-13.13 ng/g dw and a high 410.2-514.4 ng/g dw, respectively [166]. This trend is; however, expected as petrochemical and industrial plants have been sited close to the Berre Lagoon. In contrast, low background $\sum_6$PCB (PCB-28, 52, 101, 138, 153, 180) concentrations (1.55-6.71 ng/g dry weight) were measured in sediment samples from UK freshwater lakes [64]. In soil samples from Australia, Abdallah *et al.,* (2013) reported $\sum_6$PCB (PCB-28, 52, 101, 138, 153, 180) concentrations between 0.331 to 3.659 ng/g dry weight for core soil samples collected from urban and industrial sites in New South Wales. However, soil samples collected in 1998 from sites remote to potential sources (urban, industrial, agriculture) in Australia reflect lower background concentrations of 0.14-0.54 ng/g dry weight [167]. PCBs were also detected in mothers milk samples from Australia, measuring mean concentrations between 160 ng/g fat and 480 ng/g fat [168]. Similarly, in Japan, 89 paired samples of serum and human milk measured geometric mean of 15 PCB congeners as 63.9 ng/g lipid for human milk and 2.89 ng/g lipid for serum [169].

2.8.4 PFASs contamination

To date, there are no records of production of PFASs chemicals in Australia except the historic secondary manufacture of consumer products that contain PFASs and the subsequent use of such manufactured products [92]. However, recent studies show PFASs contamination in sediment, water and biota samples from Sydney [27]. Mean concentrations of PFASs in surface sediment samples from the Paramatta River in Sydney, Australia, were 0.03 ± 0.06 ng/g dry weight (PFOA) and 2.1 ± 2.0 ng/g dry weight (PFOS) [27]. Similarly, the only study on PFASs in the Brisbane River estuary by Gallen *et al.,* (2014) measured PFOS and PFOA in the ranges of <LOQ-4.9 ng/L and 1.2-1.4 ng/L respectively in water samples [26]. Gallen *et al.,* (2014) however did not assess the impact of land-use on the PFASs contamination and also did not analyse sediments from the river. The status of possible sediment contamination by PFASs in the Brisbane River is currently not known even though the river is susceptible to

landfill leachates and effluent discharges from WWTPs. PFASs are released from municipal landfills and deposited into water resources which serve as a primary sink due to the ionic and water soluble properties [103]. Armitage *et al.,* (2013) observed that at environmentally favourable pH, the ionogenic PFASs undergo complete ionization. Thus PFASs with high sediment-water partition coefficient (Kd) can settle on sediments as they become less mobile and attach to particulate matter [170]. The previous use of AFFF which contain PFASs at FTGs has been reported as potential primary source of PFASs contamination in soils [44]. Some FTGs studied in Queensland shows PFASs contamination of soils, biota, surface and groundwater [21, 40, 46]. Nonetheless, these studies did not investigate the mobility of PFASs along a depth profile at the FTGs nor account for the distance travelled by PFASs along depths over a period of time. Since PFASs have been reported to impact groundwater, it is necessary to understand the mobility trends and the soil physico-chemical parameters affecting the transport of PFASs along depth profile at FTGs in South-East Queensland.

Elsewhere, PFOA concentrations in the range; 500-640 ng/L were measured in a PFASs contaminated drinking water from Arnsberg, Germany [171]. Holzer *et al,* (2008) also observed that levels of PFOA in blood plasma of residents living in Arnsberg were about 8 times higher than for a reference population, partly from the source of drinking water. In a related study, Oliaei *et al.*, (2010) measured PFOS, PFOA, PFHxS and PFBS concentrations in water, sediment and fish samples collected downstream of a PFASs plant and wastewater treatment plant along the Mississippi River in the USA. It was inferred that PFOS (1.7-27.9 ng/g) in the sediment samples may pose exposure risks to bottom feeding aquatic organisms in the river [103]. The consumption of fish from PFASs contaminated sources is thus another medium of human exposure. Zhao *et al*., (2011) also detected PFASs in fish species from Hong Kong and Xiamen and calculated a hazard ratio of about 0.5 for PFOS in some fish species which may pose a health risk through continuous consumption [172]. Concentrations of PFOS and PFOA were also measured in 36 composite food samples from Catalan (Spain) with a daily intake of 74.2 ng/day/adult for PFOS [171].

2.9 Analytical techniques for monitoring organic contaminants

The modelling of environmental toxicants requires quality analytical data to generate reliable models for a safe environmental management. Analyses of organic contaminants in environmental samples are often confronted with the removal of interfering compounds in the

matrix. Therefore, detection is usually preceded by sample pre-treatment and extraction. Generally, sample analysis steps include; homogenization, lyophilisation, organic solvent extraction, lipid removal, clean-up followed by Gas or Liquid chromatographic (GC or LC) separation and mass spectrometric detection. Soxhlet (liquid-solid extraction) is widely used as a standard extraction technique [173]. Recently, several advanced extraction techniques including: pressurised liquid extraction or accelerated solvent extraction (ASE), supercritical fluid extraction (SFE), microwave assisted solvent extraction, ultra-sonication and alkaline digestion (followed by liquid-liquid extraction) have been used [174]. The ASE is; however, the recent extraction method of choice for PBDEs, PCBs and HBCDDs in soils and sediment due to its advantages [18, 64]. Some of the advantages of ASE are that: extraction can be carried out over a wide range of sample size (1-100g); it offers a reduction of organic solvent by 90%; it is applicable for the extraction of a range of organic contaminants; it has a dionium cell for the extraction of both acidic and alkaline matrices; extracts are simultaneously cleaned-up; and it offers high recoveries and throughput. In a comparative study, recoveries obtained for ASE extraction of soil samples were higher than Soxhlet extraction technique when the same internal standards were used [43]. Desborough *et al.*, (2016) reported the recoveries of HBCDDs via Soxhlet extraction as 79 ± 35% (13C-α HBCDD), 59 ± 28% (13C-β HBCDD) and 46 ± 18% (13C-γ HBCDD) relative to recoveries obtained via ASE extraction; 90 ± 34% (13C-α HBCDD), 63 ± 27% (13C-β HBCDD) and 93 ± 34% (13C-γ HBCDD). While PFASs in soils and sediment have widely been extracted using ultra sonication followed by solid phase extraction (SPE) for clean-up and concentration of analytes [27, 97, 175], extraction of water samples are achieved via direct SPE [26, 29]. Similarly, PPCPs and CUPs are also extracted by SPE [3, 4, 117] as an effective method for isolation, pre-concentration and clean-up for such contaminants at low trace concentrations.

The application of mass analysers such as; ion-trap, quadruple/triple quadruple, orbitraps, and time-of-flight in tandem with the conventional GC-MS or LC-MS, such as High Resolution Gas Chromatography-Mass Spectrometry (HRGC-MS/MS) or High Performance Liquid Chromatography-Mass Spectrometry (HPLC-MS/MS) enhances selectivity and sensitivity for the detection and measurement of organic contaminants. PBDEs and PCBs have been analysed using the HRGC-MS/MS [18, 176, 177]. However, BDE-209 congener is often analysed using the conventional GC-MS, operated in the electron capture negative ionisation

(ECNI) mode and run on a shorter column. BDE-209 is less sensitive on longer columns because at high temperatures and longer retention periods in the column oven, they decompose to form lower congeners thus given a poor peak resolution. Shorter columns circumvent this decomposition and afford a faster run and high sensitivity. Similarly, at GC temperatures >160 ^{0}C, the three diastereomers of HBCDD (α-, β-, and γ-HBCDD) tautomerize due to thermal induced reactions and isomeric rearrangement which result in poor sensitivity [160]. However, HPLC circumvents thermal induced reactions and isomeric rearrangements and is able to separate the diastereomers [178]. HPLC-MS/MS have therefore been used in previous studies to analyse the HBCDD diastereomers [18, 43, 64]. PFASs extracts can contain both ionic and neutral components. The neutral fractions of PFASs following SPE separation can be analysed using either GC-MS or HPLC-MS/MS [179, 180]. However, PFASs observed in environmental samples exist in the anionic form. Therefore, HPLC-MS/MS is the method of choice due to the anionic nature of most PFASs which requires a semi-polar liquid mobile phase and particularly the variation of two solvent systems to achieve optimum gradient elution for highly sensitive detection [26, 27, 97, 181, 182]. The detection and measurement of PPCPs and CUPs have also been achieved via HPLC-MS/MS [3, 106, 117].

2.10 Summary and implications

It is evident that FR-Ps and PFASs are present in environmental compartments regardless of their ban and or restrictions. The literature shows that the distribution of these environmental contaminants in sediment, water and soils have not been given much attention in South-east Queensland. While the status of HBCDDs and PFASs in sediments from the Brisbane River has not been cited in the literature, investigations on PBDEs were last conducted more than a decade ago and did not cover a wide section of the Brisbane River. Similarly, PCB congeners have also not been evaluated in the Brisbane River sediment. Given that the Brisbane River is susceptible to leachates from landfill sites as well as effluent discharges and stormwater, PBDEs, PCBs, HBCDDs and PFASs are likely to be continuously deposited in the river. Aside from the few studies that were previously carried out on these contaminants in the Brisbane River, the effect of sediment physicochemical parameters on the transport and fate of the contaminants have also not been addressed. Further, observations in the literature of PFASs contamination of groundwater arising from the use of AFFF at firefighting training

grounds is also a concern to investigate the mobility and transport of PFASs in soils, particularly along a coring depth. Previous studies carried out at some FTGs in South-East Queensland have also not investigated the influence of soil physico-chemical properties on the sorption of PFASs and its mobility. This research therefore finds it necessary to investigate the transport of PFASs along soil cores in other to understand the distribution along the depths and also assess soil physico-chemical properties affecting PFASs transport. Unlike, previous studies which only measured PFASs concentrations in soils at one depth, this project estimated the travel time of PFASs from the top layer of the soil into lower layers over a period of time as a way of estimating the duration at which PFASs would reach groundwater at a particular study site. It is also important to evaluate the mass load of PFASs in the soils at the selected FTG as this will serve as the basis for the selection of effective remediation strategies. The presence of PPCPs and CUPs in water bodies is also a cause for concern since low concentrations of these contaminants can cause potential adverse effects to biota. Current WWTPs are not able to remove PPCPs and CUPs efficiently from waste streams. Therefore, they are continuously discharged via effluents into receiving waters such as the Brisbane River estuary. Once released into water, the salinity gradient along the Brisbane River due to mixing with sea water can influence the distribution and hence fate of the contaminants in the river estuary via the mixing mechanism. Salinity of the river can therefore serve as a benchmark to investigate the fate of PPCPs and CUPs. Thus unlike previous studies in Sydney, Australia, where only concentrations of PPCPs and CUPs were measured in the Sydney estuary, this work will use salinity to investigate the conservative behaviour of the contaminants as well as deducing point source inputs along the transect of the Brisbane River estuary. Knowledge of the conservative behaviour of specific PPCPs or CUPs will serve as basis for designing effective treatments protocols at WWTPs and thereby safeguarding aquatic organisms.

Chapter 3: Study area and experimental methods

Overview

Assessment of the fate of contaminants in environmental samples requires robust and reproducible methods to generate quality analytical data. Therefore, this chapter describes the experimental methods that were used in this study to generate quality analytical data in order to achieve the aims and objectives stated in Section 1.5 of Chapter 1. Section 3.1 describes the sampling areas; the Brisbane River estuary and a firefighting training ground located in an aviation industry. Concerns for potential contamination of water, sediment and soils from the sampling sites due to varying land-use activities were elaborated. All the chemicals, reagents and standards (mass-labelled, native and instrumental standards) used for the analysis are listed in Section 3.2. The collections of sediment, water and soils samples as well as extraction of the samples prior to analysis are described in Sections 3.3 and 3.4 respectively. Section 3.5 detailed instrumental analysis of the extracts while method of quantitation after the instrumental analysis is presented in Section 3.6. Methods for the determination of physicochemical parameters (pH, salinity, organic carbon and mineralogy) are discussed in Section 3.7. To assess the reproducibility and precision of the analytical methods and results, QA/QC protocols included during sample collection, preparation and analysis are highlighted in Section 3.8. Finally, Section 3.9 describes how the mass loads of PFASs in the bulk core soils were estimated

3.1 Description of sampling areas

3.1.1 The Brisbane River estuary

The Brisbane River estuary, measuring 344 km long [26] flows through a catchment area of about 13560 km^2 located in the Moreton Bay region in South-East Queensland. It flows from Mount Stanley through the Brisbane City and empties into the Moreton bay. The River is susceptible to sea water intrusion from tides pushing from the Moreton bay and moving upwards towards the source where the water is considered fresh. This seawater-freshwater mixing renders the River an estuarine. Dredging of the River has extended the tidal limit to

≥85 km upstream from the mouth of the River (Moreton Bay). The flushing time of the River range from 110 days at the mouth of the River to >120 days further upstream [183]. This relatively slow flushing rate of the estuary could enhance steady state mixing and therefore contaminant retention. In this study, the sampling area, measuring about 71 km long between Karana Downs (designated upstream) and the Port of Brisbane (designated downstream) is characterised by varying land-use types as potential contaminant sources. The land-use types include farmlands and rural residential areas, parklands, wastewater treatment plants (WWTPs), landfills, commercial activities and urban-residential areas as well as industrial activities (Fig. 3.1). Thus contaminant inputs from leachates, surface-runoff, leakages or effluents from WWTPs could be deposited into the River and within the study area. Aside from the major land-use types, tributaries and stormwater also drain directly into the Brisbane River estuary. Recently, two major floods which occurred in 2011 and 2013 were reported to have washed-off household materials and automobiles into the Brisbane River estuary [28]. Following these flood events, Gallen *et al.*, (2014) measured PFASs in water samples from some locations along the Brisbane River estuary. Economic activities along the Brisbane River estuary include agricultural activities and transportation.

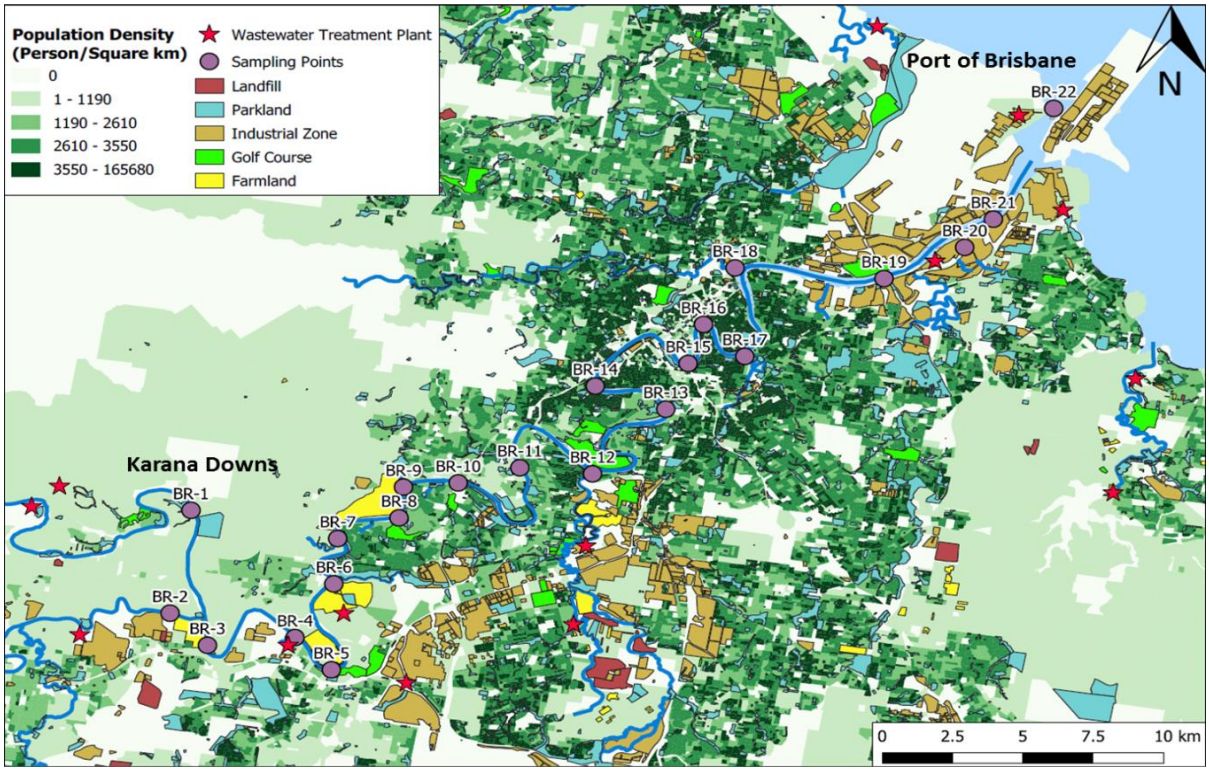


Fig. 3.1: A map of the Brisbane River estuary showing the study sites and land-use activities. The map was drawn using ArcMap 10.3.1 with the base maps of land-use obtained from Imagery ©2019 Google, CNES/Airbus, Map data ©2019 Google Australia under open access.

3.1.2 Firefighting training ground (FTG)

The study area is a FTG located within an airport (the exact name of the airport is withheld due to confidentiality agreement) in South-East Queensland. Firefighting services use such facilities for training to address any fire incidents, in this case incidents arising from aviation activities. Aviation based FTG typically consist of a mock-up plane mounted on a bunded concrete pad. In this study, the area of the concrete pad is ~ 508 m^2. A schematic of the FTG is presented in Fig. 3.2.

At this FTG, AFFF products were used for over two decades starting in the late 1970s until 2010, with 3M Lightwater used for 15 years followed by the use of Ansulite for 7 years. The AFFF formulations used at the site contain both pure perfluoroalkyl substances which can be released directly into the environment and polyfluoralkyl compounds that can breakdown under both biotic and abiotic conditions to form the more persistent perfluoroalkyl acids (PFAAs), such as perfluoroalkyl carboxylic acids (PFCA) and perfluoroalkyl sulphonic acids (PFSA). Despite the discontinuation of AFFF at the training ground, PFASs from past usage can potentially permeate into the surrounding environmental matrices, including soils and groundwater. Note, the concrete pad itself can also be regarded as a potentially ongoing source, as it has become impregnated with PFASs as a result of its long history of contact with AFFF during training activities.

The FTG site is bounded by a network of drainage canals to the West, tidal channels to the South and an estuarine woodland on the North side, while the Eastern side is underlain by an unconfined aquifer which are influenced by tidal flows and provide flow pathways from the FTG to the marine environment via stormwater runoff or groundwater emergence. Notable in the area is the interface between fresh and saline water (brackish water), potentially contributing to the partitioning of PFASs in surface waters and inhibiting subsurface migration of PFASs through the unsaturated zone into groundwater.

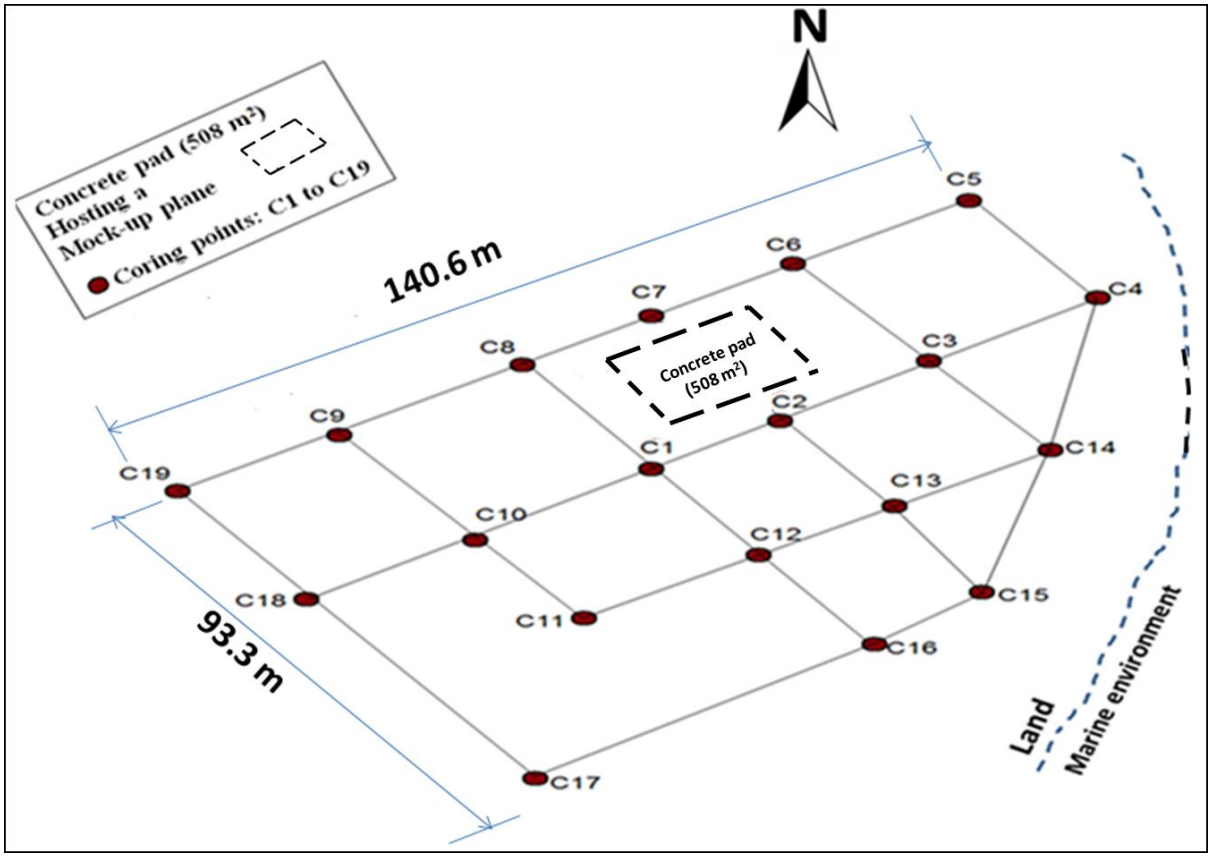


Fig. 3.2: A schematic of the FTG showing sampling collection points; C1 to C19 indicating the individual coring points.

3.2 Chemicals, reagents and analytical standards

Sulphuric acid (98% purity) and HPLC grade organic solvents; n-hexane, dichloromethane and methanol used for sample extractions were supplied by Merck (Darmstadt, Germany). Ammonium hydroxide, ammonium acetate and acetic acid (≥99.7%) were from Thermo Fisher Scientific while copper powder (<425 µm, 99.5 %), Florisil (60-100 mesh) and silica gel (pore size 60A, 40-63 µm, high purity) were obtained from Sigma-Aldrich. ^{13}C-labelled PCBs (marker-7 PCB mix, used as surrogate), ^{13}C-labelled PCB-141 (used as recovery standard) and ^{13}C-labelled PBDEs (method 1614 labelled stock solution) were supplied by Cambridge isotope. Native PCBs (PCB-28, 52, 101, 118, 138, 153 and 180) were obtained from AccuStandard (Dutch seven PCBs standard). Other standards: ^{13}C-labelled HBCDD (MHBCD-MXA, used as surrogate HBCDD isomers), native PBDE (BDE-MXE), native HBCDD (α-, β-, and ϒ-), recovery standards (^{13}C-labelled BDE-77 and TBBPA), ^{13}C-labelled PFASs (MPFAC-MXA), native PFASs (PFAC-MXB), PFASs mass labelled instrumental standards (M8PFOA and M8PFOS) were all supplied by Wellington

laboratories (Guelph, Ontario, Canada). Native and mass-labelled standards for PPCPs and CUPs were supplied by Novachem, Australia.

The mass-labelled PFASs used as surrogate standards (MPFAC-MXA) contain a mixture of: perfluoro[1,2,3,4-$^{13}C_4$]butanoic acid, perfluoro[1,2-$^{13}C_2$]hexanoic acid, perfluoro[1,2,3,4-$^{13}C_4$]octanoic acid, perfluoro[1,2,3,4,5-$^{13}C_5$]nonanoic acid, perfluoro[1,2-$^{13}C_2$]decanoic acid, perfluoro[1,2-$^{13}C_2$]undecanoic acid, perfluoro[1,2-$^{13}C_2$]dodecanoic acid, sodium perfluoro-1-hexane[$^{14}O_2$]sulfonate, and sodium perfluoro-1-[1,2,3,4-$^{13}C_4$]octanesulfonate. The mixture of native PFASs standards (PFAC-MXB) used contain: perfluorobutanoic acid (PFBA), perfluoropentanoic acid (PFPeA) perfluorohexanoic acid (PFHxA), perfluoroheptanoic acid (PFHpA), perfluorooctanoic acid (PFOA), perfluorononanoic acid (PFNA), perfluorodecanoic acid (PFDA), perfluoroundecanoic acid (PFUnA), perfluorododecanoic acid (PFD_oA), perfluorobutane sulphonate (PFBS), perfluorohexane sulphonate (PFHxS) and perfluorooctane sulphonate (PFOS). The PFASs mass-labelled instrumental standards used for recovery calculations contain $^{13}C_8$-PFOS (M8PFOS) and $^{13}C_8$-PFOA (M8PFOS). Instrument recovery standard for PPCPs and CUPs contain D4-acetyl sulphamethoxazole and 2,4 dichlorophenoxyacetic acid.

3.3 Sample collection

3.3.1 Sediment and water

Sediment and water samples were collected at the sample locations (Points 1 to 22) as shown in Fig. 3.1 above. Sampling procedure for sediment followed the Australian/New Zealand standards [184, 185]. Sampling site selection criteria for sediment is based on locations of potential sources of contaminant inputs from agricultural, urban or industrial activities. Grab sediment samples at a depth of 0-3 cm were collected between 2014 and 2015 for this study. At each sampling site, replicate samples were collected and homogenised. Sediment samples in direct contact with the sides of the grab sampler were excluded. Each sample was transferred into a pre-cleaned glass bottle and tightly capped and labelled. Label numerals depict the designated sections along the River with respect to land use as; sites 1-9 (upstream samples), sites 10-18 (mid-stream samples) and sites 19-22 (downstream samples). Diatomaceous earth (hydromatrix) was packed into a pre-cleaned glass bottle and sent to the

field to represent field blank. The samples and field blanks were transported to the laboratory on ice in a thermos insulator box.

The water samples were collected into 500 ml polyethylene bottles at approximately 0.5 m below the water surface using grab water sampler and the samples stored on ice before transporting to the laboratory. Prior to filling the bottles at each sample collection point, the bottles were rinsed 3 times with site water while wearing a new pair of nitrile gloves for each sample. Milli-Q water was used as field blank. Samples were collected in December, 2017 when there were no major rains with the objective of having a near steady state River flow. A steady state condition will mean longer residence time of contaminants in the estuary and also ensure a significant salinity gradient which can be used to assess the behaviour of contaminants along the estuary [111, 112]. Physico-chemical parameters (pH, temperature, salinity, conductivity) of the waters samples were measured *in-situ* (Appendix A, Table A-3.1). Water samples and field blanks were transported to the laboratory on ice in a thermos insulator box where they remained in storage at -20 °C until extraction.

3.3.2 Soil cores

Soil core samples were collected at the FTG in November 2015. Prior to this, exploratory sampling was carried out at depths of 10 cm using a hand auger and the samples analysed to estimate the contamination levels. Following the outcome of the exploration, 19 sampling points, extending from the FTG concrete slab (as a point source), were identified in an optimised grid pattern (Fig. 3.2). Soil cores (C) were collected at depths of 0-0.5 m, 0.5-1 m, 1-1.5 m and 1.5-2 m representing sleeves (S), S1, S2, S3, and S4, respectively. Samples were collected in PVC tubes with a diameter of 7.5 cm using a Geo 305 rig. The soil core samples were transported to the laboratory in coolers and stored in a freezer at -20 °C prior to further preparation and analysis.

3.4 Sample preparations

3.4.1 Extraction of sediment samples for PBDEs, PCBs and HBCDDs

Sediments were freeze-dried for 48 hours using an Alpha 1-4 LDplus freeze dryer (John Morris Scientific, Australia). The dried samples were ground using a mortar and pestle to loosen any lumps and then sieved through a 250 µm meshed sieve. The method of extraction

was as described elsewhere [18, 64] but with a few modifications. One-step extraction and clean-up procedure were achieved by accelerated solvent extraction (ASE) using Dionex™ ASE™ 350 manufactured by ThermoFisher Scientific. Essentially, 66 ml ASE Dionium cells were packed starting from the bottom of the cell in the following order: 2 microfiber filter, 5g activated florisil, 3g hydromatrix, microfiber filter, 10 g 44% acid silica, microfiber filter, 5 g Cu powder and 1g hydromatrix as shown in Fig. 3.3. The packed cells were pre-cleaned by ASE for one cycle at 90 ^{0}C using n-hexane (3): dichloromethane (2) mixture ratio with 2 minutes heating time and static time of 5 minutes. Each cell was purged for 120 seconds at the end of a cycle. After cell clean-up, 8-10 g of dried and ground sediment sample were added onto the pre-extracted packed Dionium cell and spiked with surrogate standards (5 ng of $^{13}C_{12}$-labelled BDEs -28, -47, -99, -100, -154, -153, -183, α- β- and γ- HBCDD, PCBs -28, -52, -101, -118, -138, -153 and -180 and 50 ng of BDE-209). Each batch of 10 samples contains both field and laboratory blanks and a replicate sample. The samples were then extracted per the pre-cleaning protocol but for 3 cycles. Extracts were concentrated to about 1 ml on a rotary evaporator at 40 ^{0}C then reconstituted using n-hexane and blown down to 200 µl under gentle stream of N_2. Finally, the extracts were spiked with 50 µl of 20 ng/ml ^{13}C-labelled BDE-77, PCB-141 and TBBPA instrumental standards and transferred quantitatively into 1.5 ml GC vials for HRGC-MS/MS (PBDEs and PCBs) analysis. Extracts for HBCDDs analysis were solvent changed into methanol before using the HPLC-MS/MS for detection and measurements. SRM-2585 (organic contaminants in house dust) was extracted similarly as the sediment samples for method validation.

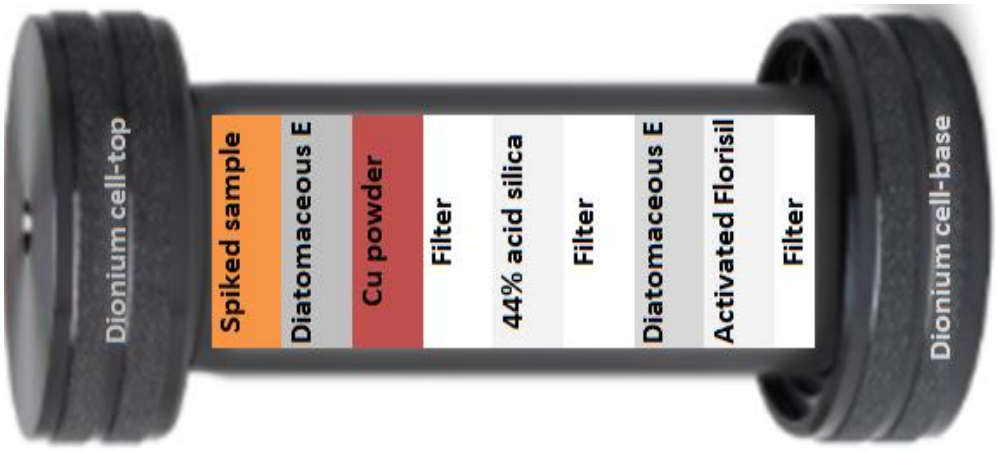


Fig. 3.3: A schematic of a packed 66 ml Dionium cell ready for ASE extraction.

3.4.2: Extraction of sediment and core soils for PFASs

The samples were first frozen at -20 ^{0}C and then freeze-dried. The dried samples were ground using a mortar and pestle and then sieved through a 1 mm sieve. The powdered samples were kept in a cold room prior to extraction and analysis. One gram of dried sample was weighed into a 15 ml falcon tube and spiked with 20 µl of 0.2 µg/ml C-13 labelled surrogate standard mix. In each batch of ten samples, one sample was selected at random and duplicated. The duplicate samples were spiked with 20 µl of 0.2 µg/ml native PFASs standard mix in addition to the surrogate spikes. A 5 ml volume of 99% MeOH/NH_4OH was then added to each sample including the duplicates, sonicated for 20 mins and centrifuged for 10 mins at 3000 rpm. The supernatant were transferred into pre-cleaned falcon tube and the residue re-extracted. The two extract portions were combined and reduced to about 1 ml under a gentle stream of N_2 and 10 % acetic acid added before carrying out clean-up by passing through 100 mg BondElut cartridges (Agilent Technologies) which were pre-conditioned with 99/1 (v/v) MeOH/acetic acid. The cleaned extracts were blown-down to about 400 µl under N_2 and reconstituted using 600 ul of 5 mM ammonium acetate. It is worth noting that for the core soil samples from the FTG (contaminated site), the cleaned extracts were reduced to 200 µl under nitrogen before adding 300 µl 5 mM ammonium acetate thus making the final volume 0.5 ml. Blanks and replicates were treated in the same manner as the samples. Prior to instrumental analysis, 10 µl of 0.2 µg/ml of instrumental standard (M8FOA and M8PFOS) was added to each final extract. A non-extracted side spike (NESS) included in every batch of instrumental analysis was composed of 600 µl of 5mM ammonium acetate in Milli-Q water, 350 µl MeOH, 20 µl 0.2 µg/ml surrogate standard mix, 20 µl of 0.2 µg/ml native standard mix, and 10 µl of 0.2 µg/ml instrumental standard.

3.4.3 Extraction of water samples for PFASs

Water samples were first mixed by inversion and then a 50 ml aliquot taken. The samples, replicates and blanks were spiked with 10 µl of 0.2 µg/ml PFASs mass-labelled standard as surrogate and the content mixed thoroughly by inversion before extraction. In a batch of 10 samples, a replicate of a sample taken at random was spiked with 10 µl of 0.2 µg/ml native PFASs standard. Prior to extraction, the Phenomex Strata X-AW 33 µm polymeric weak anion, 100 mg/6 ml extraction cartridges were mounted on a manifold and preconditioned

using 4 ml of 0.2% NH_3/MeOH followed by 4 ml MeOH and then 4 ml milliQ-water. After draining the preconditioning solutions, the taps were closed and another 4 ml milliQ-water was added to keep each cartridge wet. The spiked samples were then poured stepwise onto the water in the cartridges through a 50 ml syringe barrel, mounted on the cartridges and allowed to drain dropwise. Each cartridge was further rinsed with 4 ml milli-Q water after passing all the 50 ml spiked sample through and allowed to drain to dryness overnight. The PFASs were then extracted from the cartridges into collection tubes by eluting with 0.2% NH_3/MeOH solution and the extracts blown down to about 1 ml under a gentle stream of nitrogen gas. Finally, the 1 ml extracts were transferred using MeOH into a 1.5 ml sample analysis vials and blown down again to 0.2 ml before adding 300 µl of 5 mM ammonium acetate in milli-Q water. Replicates and blanks were all extracted in the same manner as the samples. All extracts were spiked with 10 µl of 0.2 µg/ml instrumental standard (M8FOA and M8PFOS) prior to analysis. A NESS for each batch of extraction was prepared and contains: 180 µl of MeOH, 10 µl of 0.2 ppm mass-labelled surrogate standard, 10 µl of 0.2 µg/ml PFASs native standard, 300 µl of 5 mM NH_4OAc-H_2O and 10 µl of instrumental standard.

3.4.4 Extraction of water samples for PPCPs and CUPs

Sample preparation and analysis were carried out according to methods described by Birch *et al*, (2015), with a few modifications [117]. The water samples were first defrosted and mixed by inversion then poured into 50 ml aliquots of each sample, which were spiked with 10 µl of 0.2 µg/ml ^{13}C mass-labelled standards: D_7-Atenolol, $^{13}C_6$-2,4 D, D3-ibuprofen, $^{13}C_3$-caffeine, D_5-atrazine, D_{10}-simazine, D_5-atrazine desisopropyl, D_6-atrazine desethyl, D_6-hexazinone, D_6-diuron, D_6-metolachlor, D_4-imidacloprid, D_4-acetaminophen, D_2-hydrochlorothiazide, D_{10}-carbamazepine, D_6-MCPA, D_{10}-gabapentin, D_5-temazepam, D_6-fluoxetine, D_6-venlafaxine, D_3-codeine, D_3-cotinine. Samples were acidified (pH 2, by adding 0.12% HCl solution) before passing them through 200 mg/3 ml Strata-X 33 µm SPE cartridges (Phenomenex) which were preconditioned with methanol and HPLC grade water containing 2% formic acid. After sample loading, the cartridges were dried under a stream of nitrogen gas and then eluted with 80:20 (v/v) dichloromethane:isopropanol. The eluted extracts were reduced to dryness under nitrogen gas then reconstituted to 500 µl using a solution of 20 (methanol):80 (Milli-Q water) ratio.

3.4.5 Soil-water partitioning

The soil-water concentration ratio was determined as an estimate of soil-water partition coefficient (K_d). Dried soil core samples (1 g) were weighed into a 15 ml PP tube and 4 ml of Milli-Q water added. The soil-water mixture was equilibrated for 5 days at 25 °C in a horizontal shaker [186, 187]. Each resulting suspension was then centrifuged for 10 mins at 3000 rpm and the supernatant collected, leaving the residual soil portion. The supernatant was prepared for direct LC-MS/MS analysis by mixing 500 µl of the supernatant with 480 µl of methanol, spiked with 10 µl of 0.2 µg/ml of surrogate standard and then filtered using 1 mL NORM-JECT micro filter (0.2 µm pore size) into a 1.5 ml polypropylene (PP) vial. The filtrate was blown down to 0.5 ml under nitrogen gas and spiked with 10 µl of 0.2 µg/ml mass-labelled instrumental standard prior to instrumental analysis. Milli-Q water blanks and sample replicates were included and treated in the same manner as described above for the samples. The residual soil samples were freeze dried and extracted in MeOH/NH_4OH following the protocol described above for the raw soil core samples. The soil-water concentration ratios were then calculated as, $\frac{Cs}{Cw}$ for each PFAS in this analysis where Cs and Cw are the concentrations of a PFASs of interest in soil and water, respectively.

3.5 Instrumental analysis

3.5.1 PBDEs, PCBs and HBCDDs in sediment extracts

Analysis of PBDEs (BDE-28, 47, 99, 100, 153, 154, 183) and PCBs (28, 52, 101, 118, 138, 153, 180) congeners were carried out using triple quadruple HRGC-MS/MS (Thermo Scientific TSQ QUANTUM XLS coupled to Thermo Scientific Trace GC Ultra and Thermo Fisher Triplus autosampler) with spectrometer performed in SRM (selected reaction monitoring) mode. List of parameters for MS/MS measurements are provided in Appendix A (Table A-3.2). 1 µl of cleaned extract was injected at 80 ^{0}C in a splitless mode for analysis on a DB-5MS column (30 m x 0.25 mm x 0.25 µm thickness). The transfer line temperature was maintained at 280 ^{0}C using Helium as a carrier gas at a flow rate of 1 ml/min. The temperature programme, started at 80 ^{0}C was held for 2 min then ramped at 20 ^{0}C /min to 180 ^{0}C and held for 0.5 min before finally ramping at 10 ^{0}C /min to 300 ^{0}C which was held for 10.5 min to complete one run. BDE-209 which decomposes at high temperatures was analysed on a SHIMADZU GCMS-QP2010 Plus with a shorter column length in other to

achieve faster elution time and optimise sensitivity. BDE-209 was run on a DB-5MS column (15 m x 0.25 mm x 0.1 µm thickness). Injection volume was 1 µl in splitless mode and operated in the negative chemical ionisation (NCI) mode with spectrometer in the monitoring reaction mode (MRM). The injection port and transfer line temperature were maintained at 270 ^{0}C. The column oven temperature was started at 100 ^{0}C for 1 min, then ramped at 20 ^{0}C /min to 190 ^{0}C and held 1.5 min and finally ramped at 20 ^{0}C /min to 300 ^{0}C and held for 2.5 min with a total run-time of 15 min.

HPLC-MS/MS was used to analyse HBCDD stereoisomers (α-, β- and γ-HBCDD). Instrumental analysis for HBCDD diastereomers were modified after earlier analysis elsewhere [188]. Separation of HBCDD diastereomers (α-, β- and γ-HBCD) was achieved using a high performance liquid chromatography (HPLC, Shimadzu Corp., Kyoto Japan) coupled to a tandem mass spectrometer (QTrap 5500 AB-Sciex, Concord, Ontario, Ca) operating in negative electrospray ionisation mode and using scheduled multiple reaction monitoring (SMRM) and a Kinetex EVO C18 column (50 x 5.1 mm I.D. x 1.7 µm particle size). A mobile phase of 5% methanol: 95% water (solution A) with 5mM ammonium acetate and 95% methanol: 5% water (solution B). Sample injection volume of 5.0 µl with an initial oven temperature at 45 ^{0}C and kept at a maximum of 160 ^{0}C was used to elute the diastereomers. The gradient elution programme was started at 80% (B) then increased to 100% (B) over 6.5 min, held constant for 5 min, followed by a linear decrease to 80% (B) over 0.1 min and held constant for another 4.9 min.

3.5.2 PFASs in extracts of sediment, soils and water

The PFASs of interest were analysed using high performance liquid chromatography (HPLC, Shimadzu Corp., Kyoto Japan) coupled to a tandem mass spectrometer (QTrap 5500 AB-Sciex, Concord, Ontario, Ca) operating in negative electrospray ionisation mode and using scheduled multiple reaction monitoring (SMRM). For separation, a volume of 5 µl was injected onto a Gemini NX C18 column (50 x 2 mm I.D., 3 µ particle size, Phenomenex, Lane Cove, Australia) held at a constant temperature of 50 ^{0}C. A pre-column (C18, 50 x 4.6 mm I.D., 5 µ particle size, Phenomenex, Lane Cove, Australia) was installed between the solvent reservoirs and the injector to trap and delay the background of PFASs originating from the HPLC system. The method is described fully elsewhere [26]. PFASs were separated by gradient elution on the HPLC using mobile phase 10% (A) and 90% (B) of milli-Q water

and methanol, respectively, with 5 mM ammonium acetate. Solution B was ramped from 25% to 100% for 9 min and held constant till 11 min before decreasing to 25% at 11.1 min and then kept constant till 15 min elution time. Identification and confirmation of peaks was done using retention times and comparing the ratios of MRM transition intensity between the samples and the standards in the same batch of analysis (Appendix A, Table A-3.3). Quantification was conducted using ^{13}C-labelled PFASs. Calibration standards were made up in 1000 µl (400 µl methanol/600 µl using 5 mM ammonium acetate in water for sediment samples) and 500 µl (200 µl methanol/300 µl using 5 mM ammonium acetate in water for the core soils and water extracts). The concentration range of the eight prepared calibration standards was 0.1– 100 ng/ml (0.1; 0.4; 1; 4; 10; 20; 40; 100).

3.5.3 PPCPs and CUPs

Analysis for CUPs, pharmaceuticals and personal care products was conducted on an API 6500+ QTRAP Mass Spectrometer (Sciex, Ontario, Canada), equipped with an electrospray (TurboV) interface coupled to a Shimadzu Nexera HPLC system (Shimadzu Corp., Kyoto, Japan). Analytes were separated using a Kinetex Biphenyl column (2.6 µm, 50x2.1 mm, Phenomenex) run at 45 ^{0}C, and a flow rate of 0.3 mL min-1 with a linear gradient starting at 5% B, ramped to 100% B in 5.2 minutes then held at 100% for 4.3 minutes (A = 1% methanol in HPLC grade water, B = 95% methanol in HPLC grade water, both containing 0.1% acetic acid). Equilibration occurred for 3.4 minutes at 5% B with flow increased to 0.5 mL min-1. The mass spectrometer was operated in scheduled multiple reaction monitoring mode with positive and negative ionisation switching, using nitrogen as the collision gas. List of isotopic masses of analyte and ionization modes for the MS/MS measurements are presented in Table A-3.4 of Appendix A.

3.6 Quantitation of analytes

Isotope dilution method was used to quantify analytes in the sample extracts. This involves the use of calibration standards which contain both native analytes and mass-labelled analytes as (surrogate standards). In each batch of instrumental analysis, calibration standards were prepared and run alongside the samples and blanks. The calibration standard, is composed of a native standard (analyte of interest) at varied concentrations (between 5 to 8 calibration points) spanning the linear range of the analyte of interest and each calibration standard

spiked with an internal standard (^{13}C-labelled surrogate) of the analyte of interest but at a constant concentration (0.2 ppm). The relative response factor is then calculated as:

$$RRF = \frac{A_{NAT}}{A_{IS}} \times \frac{C_{IS}}{C_{NAT}} \qquad \text{Equation 3.1}$$

where A_{NAT} is the peak area for the native (analyte) compound in the standard; A_{IS} is the peak area of the internal standard (^{13}C-labeled) in the standard; C_{NAT} is the concentration of the native compound in the standard; and C_{IS} is the concentration of the internal standard in the standard. The RRF is then applied to determine the concentration of analyte in the sample extracts using the equation:

$$\text{Analyte concentration (sample)} = \frac{A_{NAT}}{A_{IS}} \times \frac{1}{RRF} \times \frac{M_{IS}}{Ms} \qquad \text{Equation 3.2}$$

Where A_{IS} = peak area of internal standard in the sample; A_{NAT} = peak area of target contaminant (analyte) in the sample; RRF = relative response factor for the target contaminant (analyte); M_{IS} = mass of internal standard added to sample (ng) and *Ms* = mass of sample (g).

3.7 Determination of physicochemical and geochemical factors

Physicochemical parameters of the water samples were measured *in-situ* using Multi 3430 meter. In carrying out the in-situ measurements, probes were cleaned and immersed in the grab water samples before filling the bottles for subsequent laboratory analysis. Salinity and pH of soils/sediment were measured by dispersing the sample in Milli-Q water at 1:4 (soil/sediment: water) ratio and the measurements taken using the multi-parameter meter (Multi 3430).

Total organic carbon (TOC) content in the soils were determined by converting the organic matter (% OM) using the relationship: % OM = % TOC x 1.724 [189]. The % OMs were measured based on the loss-on-ignition method [190] after the soils were corrected for moisture content. About 1-2 g of soils were weighed into porcelain crucibles and oven dried at 105 ^{0}C for 24 h. The crucibles were cooled in a desiccator and the weight by difference at 105 ^{0}C noted as W_{105}. The moisture-corrected sample in the crucible was placed in a pre-heated furnace at 550 ^{0}C for 2 hrs, cooled in a desiccator and the weight taken (W_{550}). The % OM was subsequently estimated using: % OM = 100 * [(W_{105}-W_{550}) / W_{105}] [190] .

Mineralogy composition of the soils was determined by X-ray diffraction (XRD) technique. The specimens were prepared for X-ray powder diffraction by the addition of a corundum (Al_2O_3, Baikowski International) internal standard at 10.000 ± 0.001 wt% to an accurately weighed aliquot (2.700 ± 0.001 g) of the samples and micronised in a McCrone mill for 6 min using 10 ml ethanol and zirconia beads. The resultant slurries were dried in an oven overnight at 40 °C and the dry and homogenous powders back-pressed into sample holders. X-ray powder diffraction patterns were collected in Bragg-Brentano geometry on a PANalytical X'Pert Pro diffractometer operating at 40 kV and 40 mA with a cobalt source. Patterns were collected at a step size of 0.016° from 5 – 90° 2θ and the total measurement time was 30 minutes per sample. Phase identification was performed using PANalytical Highscore Plus (V4) and MDI Jade (V4.1) with various databases (PDF4+, American Mineralogist Crystal Structure Database 2010, Crystallographic Open Database, and ICSD FIZ Karlsruhe 2011). Quantitative phase analysis was performed using the Rietveld method as implemented in TOPAS (V5, Bruker). Results for the mineral composition in the soils were reported as the wt% in the original sample.

3.8 QA/QC procedures

To ensure the quality and reproducibility/precision of the analytical methods and hence results, both field and laboratory blanks, replicate samples and certified reference materials (where available) were included. Matrix Spike Recoveries (MSR) of labelled spike standards were also performed. In each batch of 10 samples a replicate and blanks were included. As part of the QA/QC procedures for the analysis of PBDEs and PCBs in sediments, SRM-2585 (NIST, certified household dust) was used in the absence of an appropriate certified sediment reference material (Table 3.1). In the absence of appropriate reference materials for PFASs, PPCPs and CUPs analysis, samples were spiked with mass-labelled standards and instrument recovery standards to measure the respective recoveries. Only HPLC grade reagents were used throughout the analysis. For each batch of MS analysis, a calibration curve with a minimum of 5 different concentration levels was drawn and a minimum R^2 = 0.99 was required before quantitation of analytes. The limit of detection (LOD) for each analyte was computed as 3 times the standard deviation of signal : noise ratio (S/N) while the limit of quantitation (LOQ) were based on a S/N ratio of 10 : 1. Since target analytes were not detected in the blanks, the reported concentrations were not blank corrected. The average

recoveries and ranges in parentheses were as follows for $^{13}C_{12}$-labelled BDEs: BDE-28: 91% (74-118%), BDE-47: 87% (73-119%), BDE-99: 90% (74-119%), BDE-100: 84% (76-102%), BDE-153: 93% (72-120%), BDE-154: 78% (75-104%), BDE-183: 81% (75-112%), BDE-209: 84% (76-112%) and for $^{13}C_{12}$-PCB-28: 100% (80-113%), $^{13}C_{12}$-PCB-52; 96% (73-116%), $^{13}C_{12}$-PCB-101: 73% (71-98%), $^{13}C_{12}$-PCB-118: 84% (71-119%), $^{13}C_{12}$-PCB-138: 75% (72-99%), $^{13}C_{12}$-PCB-153: 85% (74-118%), $^{13}C_{12}$-PCB-180: 81% (76-93%), whereas $^{13}C_{12}$-α-HBCDD: 81%(77-118%), $^{13}C_{12}$-β-HBCDD: 77% (76-110%), and $^{13}C_{12}$-γ-HBCDD: 79% (78-112%). The recoveries of the $^{13}C_{12}$-labelled internal standards for PFASs were in the ranges: PFBA (78-86%), PFPeA (83-105), PFHxA (79-93%), PFHxS (88-108%), PFOA (87-109%), PFOS (86-101%), PFNA (85-105%), PFDA (76-95%), PFUdA (67-81%) and PFDoA (73-84%). Similarly, recoveries of mass-labelled PPCPs and CUPs were in the range 65-113%. For statistical analysis of results, analytes <LOQ were computed as ½ LOQ.

Table 3.1: Method validation results for SRM 2585 (n=9)

	SRM 2585 Organic Contaminants in House Dust		
Analyte	Certified	This work	%Relative accuracy
BDE-28	46.9±4.4	47.3±3.6	101
BDE-47	497±46	469.3±6.6	94.4
BDE99	892±53	823.8±39.4	92.4
BDE100	145±11	155.7±20.4	107
BDE-153	119±1	98.4±11.5	82.7
BDE-154	83.5±2	96.8±10.2	116
BDE-183	43±3.5	46.4±4	108
BDE-209	2510±190	2505±34	99.8
PCB-28	13±1	15±2.5	116
PCB-52	21.8±1.9	18±0.3	82.6
PCB-101	29.8±2.3	26.3±1.4	88.3
PCB-118	26.3±1.7	21.9±0.9	83.3
PCB-138	27.6±2.1	24.2±5.5	87.7
PCB-153	40.2±1.8	39.1±5.6	97.3
PCB-180	18.4±3.2	20.9±3.8	114

3.9 Estimation of PFASs mass load in the bulk soils

The mass load of PFASs within the bulk soil was estimated by first dividing the sampling area into grids; ***a***-l (Fig. 3.4). The area for the mass load estimates excluded the concrete pad area of the FTG, which measured 508 m^2, since the concrete was not sampled in this work.

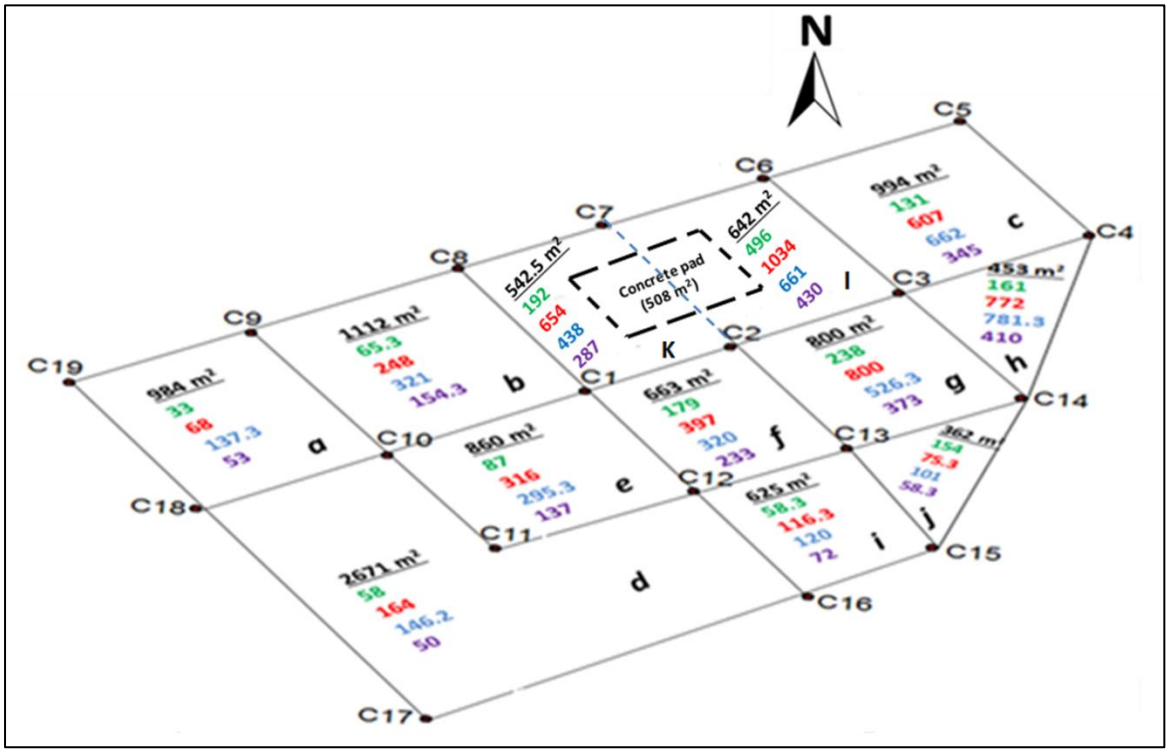


Fig. 3.4: Schematic of the FTG partitioned into grids **a** to l showing the bounded area per grid in **black** and mean concentration of PFOS (ng/g dry weight) in coloured codes for the respective sampling depths: **green (0-0.5 m), red (0.5-1 m), blue (1-1.5 m) and purple (1.5-2 m).**

Mass loads per sampling depth (0.5 m interval up to 2 m) were estimated based on the following assumptions:

i. The mass load of a specific PFAS in each grid (***a***-l) is made up of the concentration of the PFAS per grid which is interpolated as the mean concentration of the sampling points bounding the respective grid, based on Theissen polygon concept [191, 192].

ii. Permeability of PFASs through the concrete pad into lower depths is negligible; based on the findings by Baduel et al. (2015), where PFOS concentration at a

depth of 12 cm into the concrete pad was lower than the concentration at the top (surface) by a factor of 100 [44]. Hence, the total area (508 m^2) of the concrete pad was not included when accounting for areas of grids **k** and **l**. Thus the areas of the bulk soil in grids **k** and **l** were calculated by subtracting the portion of the concrete pad elapsing in each grid from the total area of grids **k** and **l** (Fig. 3.4). Thus the mass loads per sleeve (Ms) were calculated as follows using equations adapted from Baduel et al., (2015):

$$Ms = \sum_{i=1}^{n} \boldsymbol{C}gi * \boldsymbol{A}gi * d * \rho \qquad \text{Equation 3.3}$$

…where Ms = sum of mass of each grid (M_{gi}) $\boldsymbol{C}$ is the concentration in ng/g dry wt., $\boldsymbol{A}$ is area in m^2, d is depth in m, ρ is the soil bulk density in g/m^3, and *gi* is the *ith* grid (***a-l***). The area of each grid was measured using ArcMap 10.3.1 with the sampling point coordinates projected in GDA 1994 MGA zone 56.

The total mass load ($Mtot$) for specific PFASs is computed as:

$$Mtot = Ms1 + Ms2 + Ms3 + Ms4 \qquad \text{Equation 3.4}$$

…where $Ms1$ to $Ms4$ represent sleeves 1-4 for depth profiles 0-0.5 m, 0.5-1 m, 1-1.5 m and 1.5-2 m respectively.

Chapter 4: Distribution of PBDEs, HBCDDs and PCBs in the Brisbane River Estuary Sediment

Contamination of Rivers by PBDEs, PCBs, and HBCDDs even at very low concentrations has been found to pose health implications on aquatic organisms and the general food chain due to the bioaccumulation of these compounds. This chapter presents a baseline data on HBCDD contamination in sediment from the Brisbane River and also bridges the data gap on PBDEs and PCBs contamination for at least the past decade. Chapter 4 is part of the manuscript that was presented and published in Marine Pollution Bulletin by Anim et al., 2017 [193] as part of findings from this thesis. The background and motivation for this study is presented in Section 4.1. The distribution along transects designated as upstream, midstream and downstream based on land-use influences are discussed in detail in Section 4.2 while Section 4.3 summarises the findings of the study.

4.1 BACKGROUND

Polybrominated diphenyl ethers (PBDEs), and hexabromocyclododecanes (HBCDDs) are incorporated as additive brominated flame retardant (BFR) compounds in domestic and industrial products [64] to either retard or prevent burning processes. Consumer products such as plastics, foam and textiles, computer and television casings, carpets and furniture contain PBDEs. HBCDDs have also gained wide applications in polystyrene foam for thermal insulation in buildings and upholstered furniture (automobile interior textiles) and, electric and electronic equipment [86, 89] while polychlorinated biphenyls (PCBs) have been applied historically as heat transfer fluids in transformer and capacitor oils as well as other industrial uses (ink and paints, carbonless copy paper, and wood floor finishers).

Due to the physico-chemical properties, including persistence and potential for bioaccumulation as well as toxicity of these compounds to humans and the ecosystem,

national and global legislations to restrict/ban their production and use have been formulated. PBDEs have been reported to disrupt oestrogen and thyroid hormones [70-72], as well as lead to reduced male fertility and ovarian development [73]. PCBs were classified as human carcinogens in 2013 by the International Agency for Research on Cancer [83]. Toxic effects on immune system disorders, behavioural alterations and reproduction defects have also been attributed to PCBs [78, 194]. Even though studies on human toxicity due to HBCDD contamination are currently inconclusive, developmental neurotoxicity and endocrine disruption have been identified as potential toxicological effects in humans [89, 195, 196]. Accordingly, PCBs, the penta- and octa- BDE commercial formulations and HBCDD have been listed as persistent organic pollutants (POPs) in 2001, 2009 and 2013 respectively under the Stockholm Convention, meaning that these chemicals are subject to legislative bans and restrictions [48, 158]. The application of commercial HBCDD for cavity wall insulation in the building industry is; however, exempt from this ban until 2024 [197]. In Australia, the importation and use of commercial penta-BDE and octa-BDE were banned in 2005 [155, 198]. Although deca-BDE is currently not listed under the Stockholm Convention, both national and international considerations are still on-going for its inclusion on the restricted/banned list [199].

BFRs and PCBs are ubiquitous and can undergo long-range atmospheric transport (LRAT) and deposit in regions far distant from their emission sources [157, 200-202]. As a result, their environmental contamination has been recognised worldwide [49, 131, 203, 204], including regions where they have never been manufactured such as Ghana in West Africa [62] and the Arctic [205]. In Australia, indoor dust has been found to contain high levels of PBDEs [198]. House or airborne dust contaminated with PBDE can settle on hard surfaces such as roofs, roads and other surfaces, and subsequently be washed-off into rivers thereby providing a pathway for the deposition of PBDEs in sediment via stormwater runoff [206].

Sediment can serve as a sink for these pollutants [64, 160, 207], thereby increasing the potential human and health impacts through the food chain. These compounds have low water solubility and can be taken up from sediment by aquatic organisms and subsequently humans through the food chain. However, studies on BFR and PCB contamination in the sediment of Australian Rivers are generally very limited, and this is particularly the case for

the Brisbane River estuarine, which flows through major urban and industrial hubs in the Queensland state with increased susceptibility to contaminated stormwater.

Unlike reports on sediments from the Sydney estuary in Australia, which showed HBCDD contamination of 1.8-5.3 ng/g dry weight [18] and similar reports from other parts of the world [160, 208], no data has currently been cited in literature on HBCDD contamination in Queensland sediment. Data on PCB contamination of the Brisbane River estuary sediment is also very limited and does not represent the contamination status for at least the last decade [24, 42]. A report by Muller et al in 2004 suggests sources of PCBs and distribution of dioxin-like compounds [209] along the Brisbane River estuary. However, a more recent study in 2008, carried out across freshwater, estuarine and marine sediments in Australia only analysed PBDE congeners, reporting a mean concentration of 4.7 ng/g dry weight for $\sum_7$BDE (BDE-47,99,100,153,154,183,209) [25]. That study analysed only a few (n=9) sediment samples from Queensland and this may not reflect the current level of contamination in the study area (Brisbane River) due to subsequent increase in urbanisation and industrialisation over recent years. Furthermore, two major flood events occurred in the Brisbane area in 2011 and 2013. These events reportedly submerged large numbers of buildings for days and washed-off household materials and automobiles into the Brisbane River [28].

Given the ubiquitous and persistent properties of BFRs and PCBs, and the flood events, it is important to investigate the current burden of PBDEs, PCBs and HBCDDs contaminants in sediment along a wider stretch of the Brisbane River. Accordingly, this research study focused on the distribution of PBDEs, HBCDDs and PCBs contamination in surface sediment along the Brisbane River estuarine.

4.2 Results and Discussions

The sums of mean concentrations of analytes across all sampling points were in the ranges: 0.01-12.4 ng/g dry wt. ($\sum_8$PBDE), 0.09-18.8 ng/g dry wt. ($\sum_7$PCB), and 0.04-9.9 ng/g dry wt. ($\sum$HBCDD). Table 4.1 shows the descriptive statistics of the results for PBDEs (BDE-28, 47, 99, 100, 153, 154, 183, 209), PCBs (PCB-28, 52, 101, 118, 138, 153, 180) and HBCDDs (α-HBCDD, β-HBCDD and γ-HBCDD) analyses in sediment samples along the designated sampling sections of the Brisbane River. The distribution of each group of analytes is discussed separately below.

Table 4.1: Mean concentrations (ng/g dry wt) of PBDEs, PCBs, and HBCDDs in sediment across sections of the sampling area [193]. (A) is the data for PBDEs while (B) and (C) presents data for PCBs and HBCDDs respectively

(A)	**PBDEs**		
	Upstream (n=19)	**Midstream (n=20)**	**Downstream (n=6)**
Analyte	**Mean ± SD (range), %detected**	**Mean ± SD (range), %detected**	**Mean ± SD (range), %detected**
BDE-28	<0.013	<0.013	<0.013
BDE-47	0.05 ± 0.03 (<0.003-0.12), 94.74	0.05 ± 0.04 (0.005-0.15), 100	0.03 ± 0.02 (0.006-0.083, 100
BDE-99	0.06 ± 0.05 (<0.003-0.20), 89.47	0.07 ± 0.06 (<0.003-0.23), 95	0.04 ± 0.06 (<0.003-0.16), 83.33
BDE-100	0.01 ± 0.02 (<0.003-0.05), 26.32	0.01 ± 0.02 (<0.003-0.06), 45	0.004 ± 0.001 (<0.003-0.004), 16.67
BDE-153	<0.013,	0.02 ± 0.008 (<0.013-0.025), 10	<0.013
BDE-154	0.02 ± 0.005 (<0.013-0.03), 21	0.024 ± 0.01 (<0.013-0.042), 25	<0.013
BDE-183	<0.030	- (<0.030-0.035), 5	<0.030
BDE-209	4.10 ± 3.09 (<0.30-11.05), 94.74	5.05 ± 3.30 (<0.30-11.91), 95	1.98 ± 0.82 (1.16-3.48), 100
$\sum_8$PBDE	4.2 ± 3.1 (0.01-11.2)	5.2 ± 3.4 (3.4-12.4)	2.04 ± 0.9 (1.2-3.7)
	(B)	**PCBs**	
	Upstream (n=20)	**Midstream (n=22)**	**Downstream (n=9)**
Analyte	**Mean ± SD (range), %detected**	**Mean ± SD (range), %detected**	**Mean ± SD (range), %detected**

PCB-28	0.44 ± 0.41 (<0.003-1.30), 90	0.88 ± 0.56 (0.01-7.71), 100	0.53 ± 0.30 (<0.003-0.97), 88.9
PCB-52	0.25 ± 0.23 (0.01-1.03), 100	0.69 ± 0.49 (0.02-1.65), 100	0.47 ± 0.43 (0.01-1.47), 100
PCB-101	0.34 ± 0.32 (0.01-1.46), 100	0.84 ± 0.82 (0.08-3.62), 100	0.58 ± 0.66 (0.01-2.19), 100
PCB-118	0.26 ± 0.28 (0.01-1.26), 100	0.51 ± 0.43 (0.02-1.95), 100	0.44 ± 0.40 (0.003-1.57), 100
PCB-138	0.79 ± 0.78 (0.03-3.30), 100	1.46 ± 1.03 (0.13-4.19), 100	1.18 ± 1.12 (0.01-3.78), 100
PCB-153	0.86 ± 0.77 (0.03-3.15), 100	1.86 ± 1.33 (0.20-5.63), 100	1.56 ± 1.21 (0.03-5.33), 100
PCB-180	0.48 ± 0.44 (0.01-1.66), 100	1.00 ± 0.66 (0.07-2.32), 100	0.86 ± 0.69 (0.01-2.33), 100
$\sum_7$PCB	3.4 ± 2.9 (0.15-12.6)	7.2 ± 4.9 (0.6-18.8)	5.5 ± 5.3 (0.09-17.6)
	(C) **HBCDDs**		
	Upstream (n=20)	**Midstream (n=20)**	**Downstream (n=8)**
Analyte	**Mean ± SD (range), %detected**	**Mean ± SD (range), %detected**	**Mean ± SD (range), %detected**
α-HBCDD	0.24 ± 0.13 (<0.013-2.00), 95	0.27 ± 0.26 (<0.01-0.84), 95	0.24 ± 0.27 (0.02-0.79), 100
β-HBCDD	0.09 ± 0.04 (<0.01-1.11), 70	0.07 ± 0.05 (<0.01-0.16), 85	0.09 ± 0.10 (<0.01-0.29), 87.5
γ-HBCDD	0.63 ± 1.46 (<0.01-6.75), 95	0.62 ± 0.47 (0.03-2.00), 100	0.87 ± 0.95 (0.04-2.63,) 100
∑HBCDD	0.96 ± 2.1 (0.04-9.9)	0.96 ± 0.7 (0.05-2.7)	1.20 ± 1.3 (0.07-3.7)

4.2.1 PBDEs

Data on PBDE contamination of the Brisbane River sediment is limited and this study provides post 2011/2013 flood data. The data set shows significant differences ($P<0.05$) for all PBDE analytes at each of the sampling points. Mean concentration of $\sum_8$PBDE across all sampling points was 4.4 ± 3.2 ng/g dry weight. The geometric mean (geomean) was therefore calculated to present a common value of reference for each analyte. The geomeans for the most detected PBDEs were: 0.03 ng/g dry wt. (BDE-47), 0.04 ng/g dry wt. (BDE-99), 0.003 ng/g dry wt. (BDE-100), and 2.9 ng/g dry wt. (BDE-209). BDE-28, BDE-153, BDE-154 and BDE-183 represent the least detected congeners in this study (Table 4.1). Maximum levels of the less detected congeners were: 0.03 ng/g dry wt. (BDE-153), 0.04 ng/g dry wt. (BDE-154) and 0.04 ng/g dry wt. (BDE-183), all measured within the midstream section of the Brisbane

River. The observation of low BDE-153, 154 and 183 levels in this study were consistent with earlier studies by Toms et al. which reported 0.038 ng/g dry wt. (BDE-183) and 0.012 ng/g dry wt. (BDE-153) whilst BDE-154 was not detected for Brisbane River sediment [25]. Thus in comparison to this study, the concentrations of BDE-153, BDE-154 and BDE-183 congeners are consistently low in the Brisbane River estuarine sediment. The presence of trace levels of these congeners in the sediment samples may be due to past use of products containing commercial penta-BDE formulation [210].

The percentage relative concentration of each congener along the sampling points indicates that BDE-209 contributes >90% at each sampling point whilst the other congeners contribute <7% to the $\sum_8$PBDE (BDE-28, 47, 99, 100,153,154,183, 209) burden as can be inferred from Table 2 above. BDE-209 represents a larger input due to the much higher usage of commercial deca-BDE (c-decaBDE) products (such as BDE-209 treated drapery fabrics, furniture and carpets).

Toms et al. measured average BDE-209 concentrations of 0.88 ng/g dry wt. about a decade ago in sediment samples collected from the Brisbane River at the CBD and Indooroopilly [25]. This concentration is considerably lower than the average concentration of 5.1 ng/g dry wt. measured for samples collected in the same section of the River (midstream) in our current study. The relatively higher levels measured in this study may reflect continuous input from c-decaBDE containing products, noting that its continuous use although not encouraged, has not yet been banned in Australia. Deposits of household products into the Brisbane River arising from the reported 2011/2013 floods in the study area may also have contributed significantly to the observed BDE-209 levels in the present study.

BDE-47, 99, 100 and 209 represent the most detected (>30%) congeners in the collected samples (Table 4.1). The pattern of PBDE (BDE-47, 99, 100 and 209) distribution along the various sections of the Brisbane River is presented in Fig.4.1. The lower levels of BDE-47, 99, and 100 relative to BDE-209 may be due to the ban on penta-BDE and octa-BDE in 2005 by the Australian Government and the persistence of only traces from previous usage of the compounds in the sediment. Levels of BDE-209 along sections of the river shows no statistical difference (p=0.328, Mann-Whitney rank sum test) between upstream and mid-stream sediment samples with median levels of 3.5 ng/g dry weight and 4.2 ng/g dry weight,

respectively (Fig. 4.1). This trend suggests a similar contamination pathway of BDE-209 in the sediment samples from upstream and mid-stream sections of the Brisbane River probably due to equilibration of contaminant as it moves from the source(s) through these sections. To date, apart from voluntary termination of production by some manufacturers and legislations by few countries, there is no absolute global sanction on the production and use of c-decaBDE suggesting that BDE-209 containing products are still in production and in use in some parts of the world. This development may result in potential leaching of BDE-209 into sediment through recycling and improper disposal of domestic goods such as high impact polystyrene (HIPS) in the television industry, upholstery fabric, and synthetic carpets [74, 211]. Generally, BDE-209 distributions have similar profiles along the study area of the Brisbane River with slightly less variability downstream (Fig. 4.1). Sediment samples collected from downstream registered a relatively lower median concentration of 1.9 ng/g dry weight which could be due to intense tidal activities downstream leading to dispersion of sediment and subsequent dilution of contaminants. Apart from other likely non-point sources, stormwater is identified as a likely transport medium of PBDE deposition into the Brisbane River estuarine sediment considering the fact that stormwater drains are channelled into the River along the study area.

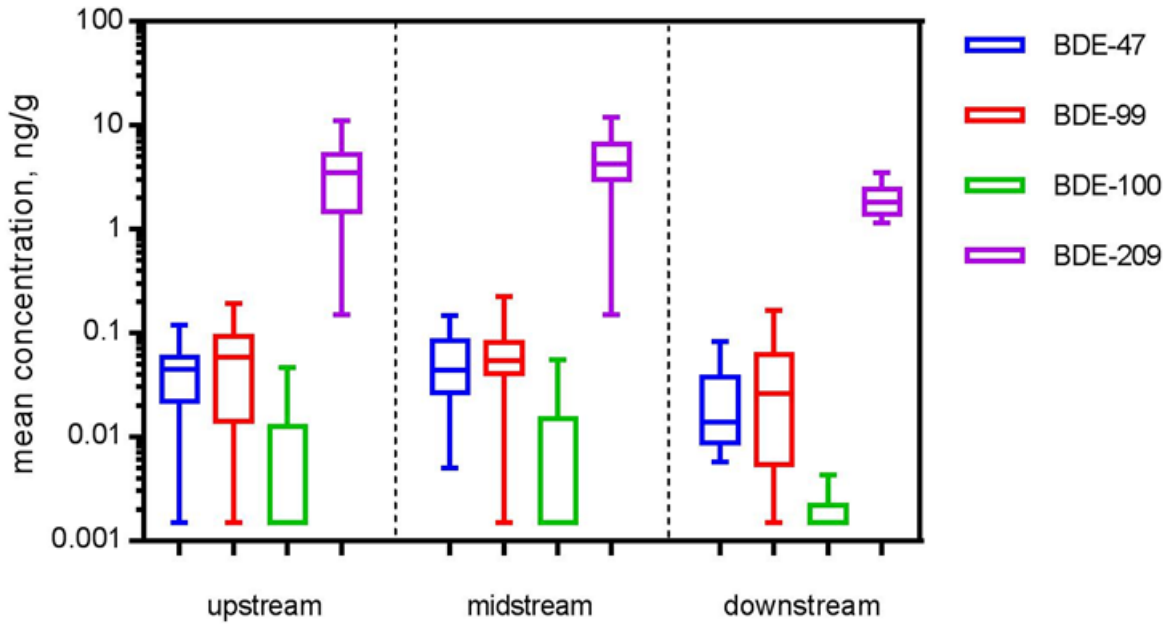


Fig. 4.1: Box and Whisker plots showing distribution patterns of PBDE congeners in sediment along sections of the Brisbane River estuary [193].

In a related study elsewhere in Australia, which was reported by Drage et al. in 2014, PBDE levels for the Sydney estuarine sediment were between 0.65 to 2.5 ng/g dry wt ($\sum_6$PBDE) and 21 to 65 ng/g dry wt. (BDE-209) [18], showing similar congener trends as observed in this work. Nonetheless, the relatively higher levels reported for the Sydney estuarine sediment were attributed to historic accumulation and outflows from active industries sited along the Sydney estuary prior to the ban of penta-BDE and octa-BDE in 2005 [18]. Essentially, Australia has a lower range of PBDE sediment levels, due to its relatively low population and the fact that PBDEs were never manufactured in Australia.

In some other regions around the world, relatively higher levels of PBDE congeners have been measured in sediment samples due to inputs from industrial activities (point sources). For example, in Spain, sediment samples from Monzon, an industrialised town along the River Cinca show $\sum_7$PBDE (BDE-47, 100, 118, 154, 153, 183, 209) concentrations in the range, 2-42 ng/g dry wt [124]. Su *et al.*, (2015) also reported BDE-209 concentrations of 3.96-327 ng/g dry wt. in sediment samples from the Hunhe River in Southeast China due to inputs from sewage and industrial effluents. The BDE-209 concentrations were notably higher at the lower stream of the Hunhe River, recording a mean of 148 ng/g dry wt. [158]. Thus economic development as a result of urbanisation and industrialisation such as evidenced in the Hunhe River catchment is a major factor contributing to the input of PBDE contaminants. Also high levels of BDE-209 (240-1650 ng/g dry weight) in sediment samples collected in 2001 from the Scheldt estuary in Netherlands, one of the most polluted estuaries worldwide, were attributed to tidal hydrodynamics as well as hydrophobic sorption of compounds unto sediment [212]. In San Francisco, where penta-BDE and octa-BDE were banned in 2003, ∑PBDE congeners (BDE-47, 99, 183, 204, 205) were measured from below detection to 212 ng/g dry weight for estuarine sediment [125] due to inputs from past applications (mainly for manufacturing and domestic use) and thus reflecting the greater usage of penta-BDE in California with BDE-47 measuring <0.5-100 ng/g dry wt. in the ∑PBDE. These high trends of BDE contamination in estuarine sediment was also observed in a study conducted in Japan in 1994 where average BDE-209 congener in sediment was 390 ng/g dry weight [51].

It is worth noting that previous studies around the world have observed higher levels of BDE-209 which may reflect its dominant use in industrial applications as well as its exemption

from a global ban (such as the Stockholm Convention) to date. Direct leaching during production and usage, or indirectly from waste streams as well as automotive scrap shredding are some of the routes of PBDE contamination in River estuarine systems [213]. Levels of PBDE congeners (BDE-28,47, 99, 100, 153, 154 and 183) in this study suggests that inputs from commercial penta-BDE and octa-BDE products have reduced significantly or have always been very low. De-bromination of BDE-209 [22, 214] as well as tidal intrusion [215] of the study area may also account for the observed PBDE levels.

4.2.2 PCBs

It is of concern that PCBs, which have been identified as human carcinogens and consequently banned, are still present at measurable levels in the Brisbane River estuary sediment (Table 4.1). 100% detection was observed for PCB- 52, 101, 118, 138, 153 and 180 in all the samples analysed (Table 4.1). Results from this study shows a mean concentration of 5.4± 4.5 ng/g dry weight for $\sum_7$PCB (PCB-28, 52, 101, 118, 138, 153, 180) and a geometric mean of 3.5 ng/g dry weight across all sampling points.

There is; however, a decline of PCB levels when compared to a few previous studies in Queensland and Australian estuarine sediment as a whole. In a previous study covering Queensland estuarine sediments, Richardson et al. reported total PCB levels in the range of 6 - 350 ng/g dry weight [216]. A similar study in the city area of the Brisbane River, reported in 1999 also shows that PCB congeners (PCB-28, 52, 101, 153, 138, 180, 202, 77, 126, 169) were present in estuarine sediment, measuring <0.01-360 ng/g TOC [42]. A more recent study reported by Müller et al. on the Brisbane River estuary (sediment, bivalves and fish) indicates that PCBs contribute >50% of organochlorine compounds studied thus suggesting local contributions of PCBs to the levels of dioxin-like compounds [209]. Other earlier reports have also indicated high levels of PCB mixtures such as Arochlor in the Brisbane River sediment [24]. Shaw and Connell (1980) have reported PCB (Arochlor) concentration for the Brisbane River sediment in the ranges: below detection-54 ng/g dry wt. and below detection-58 ng/g dry wt. for Arochlor with 54% and 60% chlorination, respectively, which were attributed to industrial outflows and leachates from refuse sites. In Sydney, a more industrialised region in Australia, estuarine sediment from Port Jackson recorded higher levels of total PCB (below detection-1921 ng/g dry weight) and were attributed to urban and

industrial inputs within the catchment, particularly from Creeks draining the estuary[217] Apart from local inputs of PCB contaminants in estuarine sediment, long range atmospheric transport (LRAT) has been identified as a possible contamination pathway. For example, low levels (0.32-0.83 ng/g dry wt) of PCBs were measured in the remote area of James Ross Island in the Antarctic Peninsula where PCBs have not been manufactured or used. [218].

In this work, the relative concentration for each PCB congener in the $\sum_7$PCB (PCB-28, 52, 101, 118, 138, 153, 180) was higher in the mid-stream section (40-49%) of the Brisbane River, followed by downstream section (30-35%) and 18-23% for upstream section (deduced from Table 4.1). Thus land-use types, particularly urbanisation exert a major influence on PCBs concentrations in the Brisbane River sediment. This influence is also evident in a related study carried out in Australia where core soil samples collected from urban and industrial sites in New South Wales, reported $\sum_6$PCB (PCB-28, 52, 101, 138, 153, 180) concentrations between 0.3 to 3.7 ng/g dry weight [64] whereas soil samples collected from sites remote to potential sources (urban, industrial) in Australia reflected lower background concentrations of 0.1-0.5 ng/g dry weight [167].

A comparison of observations made in the current work to other regions of the world indicate that PCB contamination in sediment is mostly associated with more industrialised countries apart from improper waste disposal in some less developed countries and also LRAT. For instance, in France, point source contributions from agricultural and industrial activities were found to have caused higher levels of PCBs in sediment samples [166]. According to Kanzari *et al.*, (2015), the concentration of $\sum_7$PCB (PCB-28, 52, 101, 118, 138,153, 180) in sediment samples from the Durance River and Berre lagoon were in the range:0.03-13.13 ng/g dw and a 410.2-514.4 ng/g dw respectively [166]. This trend was attributed to petrochemical and industrial plants that were sited close to the Berre Lagoon as opposed to major agricultural influences on the Durance River. Similarly, very high levels of total PCBs (36-1409 ng/g dry weight) were measured in sediment samples collected from the Mersey estuary in the UK which is within an industrialised and urban catchment [219]. However, the levels in the current work agrees with those in a related study carried out in Bengal, Northeast India which reported that estuarine sediment from 10 sampling stations within the Hugli River estuary were contaminated with $\sum_{14}$PCB (0.18-2.33 n/g dry weight) [220].

The low levels reported in this work in comparison with high levels reported in industrialised parts of Europe, suggests that current inputs into the Brisbane River have ceased or very minimal. Thus apart from LRAT, PCBs present in the Brisbane River estuary may be due to contributions of past deposits from leachates or stormwater considering the persistence and the low water solubility of PCB compounds.

4.2.3 HBCDDs

To the best of our knowledge, no data on HBCDD contamination in the Brisbane River sediment has been cited in the literature. The mean ∑HBCDD (α,β,γ) measured in this work was 1.0 ± 1.5 ng/g dry weight across all sampling points reflecting a geometric mean of 0.6 ng/g dry weight. The level of HBCDD stereoisomers in this work shows a consistent distribution across all sections of the sampling area (Table 4.1). This is probably indicative of diffuse source of contamination.

In this study, mean diastereoisomer concentrations (ng/g dry weight) were 0.25 ng/g dry wt. (α), 0.08 ng/g dry wt. (β) and 0.67 ng/g dry wt. (γ), which corresponds to 25% (α-HBCDD), 8% (β-HBCDD) and 67% (γ-HBCDD) of the ∑HBCDD (α,β,γ) across all sampling points. This observation also agrees with the trends of 23% (α), 5.5% (β) and 72% (γ) reported for Sydney River estuary sediment collected in 2014 [18]. Significantly, higher levels of γ-HBCDD diastereomer were measured across all sections of the studied area (Table 4.1) similar to literature values where HBCDD is usually present in sediment as γ-HBCDD (>90%) [69, 86]. This possibly reflects the fact that commercial/technical HBCDDs consist mainly of 10-13% (α-HBCDD), 1-12% (β-HBCDD) and 75-89% (γ-HBCDD) [86, 221] diastereoisomers.

Although the distribution pattern in this work was similar across the designated sections of the Brisbane River (Table 4.1), the levels were significantly lower compared to earlier reports for the Sydney River estuary sediment. The mean concentration of 3.5 ng/g dry weight measured in the Sydney River estuary sediment in 2014 [18] across all sampling points was higher than the 1.0 ng/g dry weight reported in this work. According to Drage et al., (2015), concentrations of ∑HBCDD (α,β,γ) in surface sediment from Sydney River estuary in Australia show a sharp increase from the late 1990s (0.12 - 2.9 ng/g dry wt.) to 2014 (1.8 - 5.3 ng/g dry wt.) [18], inferring that urbanisation and potential leachate from historic landfill

sites and/or run-off were the sources of ∑HBCDD (α, β, γ) in the Sydney estuarine sediment. The presence of HBCDD in environmental samples should be expected to increase due to its continual application in the building industry until 2024 [48]. Thus demolition of old buildings that contain HBCDD wall cavity insulators may have dispersed these diastereomers into the atmosphere due to LRAT and subsequent deposition in River sediment via stormwater [69].

Elsewhere, higher levels of HBCDDs were measured in sediment samples collected from some parts of Europe: 0.2-950 ng/g dry wt (Belgium), <0.8-9.9 ng/g dry wt (Netherlands), <2.4-16780 ng/g dry wt (UK) and <1.7-12 ng/g dry wt (Ireland) [160] mainly due to urbanisation and industrial outflows. Also in the Pearl River Delta region located in Southern China, ∑HBCDD (α,β, γ) observed concentrations were between 0.03 to 31.6 ng/g dry weight with elevated levels recorded at urban and industrialised locations [208]. Geographical location is thus a significant factor affecting the levels of HBCDD contamination in sediment as HBCDD is reported to be more heavily used in Europe than other parts of the world [222]. Inconsistency in geographical distribution of ∑HBCDD (α,β, γ) contamination was earlier acknowledged by Lam et al. [91].

Currently, in Australia, apart from the building industry, the importation of commercial HBCDD as powder or granules for use in expandable polystyrene foams and polypropylene resins has decreased from 90 tonnes (2006-2007) to about 60 tonnes between 2009-2010 prior to the cessation of imports in 2010 [89] possibly explaining the lower levels measured in this work compared to some parts in Europe. Nonetheless, a continual monitoring of HBCDD in Australian sediment is necessary considering their persistence and potential toxicity coupled with the notable consistent distribution of diastereoisomers across all the studied sections of the Brisbane River estuary.

4.3 Summary

Regardless of the ban on persistent organic pollutants, a suite of PBDEs, PCBs and HBCDDs are present in the Brisbane River estuarine sediment at measurable concentrations. These contaminants seem to be more or less uniformly distributed along the entire studied sections of the River from the upper section of the estuary through the heavily urbanised mid-section to the River mouth. Studies like this contribute to the documentation of the global budget of persistent organic pollutants in environmental matrices.

Chapter 5: Contamination of PFASs in the Brisbane River estuary

Contamination of PFASs in environmental matrices including water and sediment has gained worldwide attention in recent times due to their potential toxicity to ecological life and the fact that PFASs compounds have been incorporated in many consumable and industrial products thus increasing the routes of environmental exposure. Chapter 5 presents a baseline study on PFASs contamination of sediments in the Brisbane River. The motivation and objectives for this study are discussed in the background information section (Section 5.1). Detailed discussion of the spatial distribution the sediments and water are contained in Section 5.2. Section 5.3 summarises the findings of the study.

5.1 Background

Investigations of distribution and fate of per-and polyfluoroalkyl substances (PFASs) in the Brisbane River estuary is of ecological importance as there is evidence of the presence of these compounds in River systems around the world even in countries where there is no record of production. The use of per-and-poly-fluorinated chemicals (PFCs), recently referred to as PFASs in the manufacture of some domestic and industrial products dates back to more than six (6) decades. Previously, PFASs have been used massively as stain protectors (water and oil repellents) in fabric, furniture, carpets and food packaging materials as well as in applications such as flame retardants in aqueous film-forming foams (AFFF) [10, 93]. Other extensive application include the manufacture of polytetrafluoroethylene, a polymer applied as a non-stick in cookware to protect against stain and abrasion [8].

Currently, its usage has been regulated due to the potential of PFASs to leach out from products into environmental matrices including sediment and water and the potential of the leached PFASs to impact the health of humans and wildlife. Some potential health implications associated with PFASs contamination are: immunotoxicity, hepatotoxicity, neurotoxicity and reproductive damage [8-10]. Recent studies have also identified some PFASs as human carcinogenic candidates [8]. As a result, some manufacturers such as the

3M Company in the US have voluntarily phased out production of perfluorooctane sulphonate (PFOS), a PFASs compound. The 3M Company is estimated to have produced about 7.3 million pounds of PFOS in the USA (Minnesota) and Europe in the year 2000 [103], a development which is likely to contribute to the global environmental load of PFASs and its antecedent issues.

To ensure the safety of humans and the environment, long chain PFASs have been characterized as persistent, bioacucumulative and toxic by the OECD/UNEP Global PFC Group under the framework of the strategic approach to International Chemicals Management (SAICM) [34] which seeks to reduce their emissions and eventually eliminate them globally. Two major long chain PFASs: perfluorooctanoic acid (PFOA), a perfluorocarboxylic acid and perfluorooctane sulfonate (PFOS), a perfluoroalkyl sulfonate have been the most widely studied PFASs globally over the years [15, 97-99] thus providing evidence of their persistence in the environment. Recently, other PFASs and their precursors including perfluorohexane sulfonate (PFHxS), perfluorononaoic acid (PFNA), perfluorodecanoic acid (PFDA) and C_{11}-C_{14} chain PFASs are also on the radar of researchers due to their potential human health and environmental toxicity [15, 103]. These PFASs have since been detected in the environment, including sediment, water, biota, food and human fluids [10, 27, 99, 104]. Consequently, PFOS and its precursors have been listed as Annex B compounds in May 2009 under the Stockholm Convention [48] thereby restricting their production and use. Even though Ti/SnO_2-Sb/PbO_2 anodes have been shown to decompose PFOA in water electrochemically [10], no natural degradation pathways have been established for PFASs due to the strong C-F bond, which defines their persistence and thus ubiquitous nature. In June, 2015, Australia adopted the Risk Reduction Approaches of PFASs by OECD/UNEP to reduce the global impact on the environment and health [34] under four themes, namely: Regulatory Approach, Policy Approach, Voluntary Initiatives and Monitoring.

To date, there are no records of PFASs production in Australia, however, sediment, water and biota samples collected from Sydney showed PFASs contamination in the Australian environment [27]. Similarly, following a major flood event that occurred in Brisbane in 2011 [28], PFOS and PFOA were measured in the ranges of 0.18-15 ng/L and 0.13-6.1 ng/L respectively in water samples from the Brisbane River estuary [26] indicating that sediment

samples could as well be contaminated. The 2011 flood, aside from washing off household products and automobile into the estuary, also compromised the integrity of 9 wastewater treatment plants (WWTPs) in Southeast Queensland which resulted in the discharge of untreated sewage due to overflows into the flood waters [26]. Due to the characteristic property of PFASs having a hydrophilic molecule attached to a lipophilic hydrocarbon chain, they can partition in both water and sediment [103]. PFASs with high sediment-water partition coefficient (K_d) can thus attach to particulate matter in water and deposit in sediments as they become less mobile [170]. Notwithstanding the presence of PFASs in water samples from the Brisbane River, studies on PFASs contamination of sediment in the estuary are currently not cited in the literature.

It is therefore necessary to investigate PFASs contamination in a wide range of environmental media in Australia including sediment and water from the Brisbane River estuary since PFASs can partition in a River system and consequently intrude the food chain through bioavailability [142]. For instance, human contamination of PFOS and PFOA has been observed in pooled blood samples in some studies carried out across the Australian population from 2002 to 2011 [92, 223]. This study therefore aims to investigate the spatial contamination of PFASs in sediment and water from the Brisbane River estuary, identifying potential sources along the River and also establish a baseline data for PFASs contamination in sediment.

5.2 Results and discussions

5.2.1 Occurrence and spatial distribution

5.2.1.1 Sediment

The levels of PFASs from this study are the first to be reported for Brisbane River sediment and therefore present a baseline data. The mean concentrations of PFASs: perfluorobutanoic acid (PFBA), perfluoropentanoic acid (PFPeA), perfluorohexanoic acid (PFHxA), perfluoroheptanoic acid (PFHpA), perfluorooctanoic acid (PFOA), perfluorononaoic acid (PFNA), perfluorodecanoic acid (PFDA), perfluorobutane sulphonate (PFBS), perfluorohexane sulphonate (PFHxS), perfluorooctane sulphonate (PFOS),

perfluoroundecanoic acid (PFUnA) and perfluorododecanoic acid (PFDoA) in the estuarine sediment samples are presented in Appendix B: Table B-5.1. Concentrations of PFASs follow the order: PFOS>PFOA, PFDA, PFUnA, PFDoA>>PFHxA, PFHxS>>PFBA, PFBS, PFPA, PFHpA, PFNA. PFPeA, PFHpA and PFNA were not quantified above LOQ whereas % quantitation above LOQ for the other PFASs were: 9% (PFBA), 9% (PFHxA), 77% (PFOA), 77% (PFDA), 18% (PFHxS), 95% (PFOS), 68% (PFUnA) and 73% (PFDoA). For the purpose of data analysis using statistical models, mean levels of analytes below LOQ were computed as ½ LOQ of the respective analyte. A chi-square test shows significant variance in the data set, $p<0.001$ along sampling sites. Thus the geometric means (ng/g dry weight) of the detected PFASs across all sampling sites were computed: 0.02 (PFHxA), 0.02 (PFHxS), 0.10 (PFOA), 0.91 (PFOS), 0.19 (PFDA), 0.08 (PFUnA), and 0.14 (PFDoA).

The most dominant PFASs in the sediment was PFOS measuring a relative high concentration of 2.6 ± 0.8 ng/g dry weight at BR-11, located within the urban section of the Brisbane River. Available data on PFASs distribution in water samples from the Brisbane River, also shows the dominance of PFOS (0.18-15 ng/l) followed by PFOA (0.13-6.1 ng/l) [26]. Incidentally, the highest concentration of PFOS (15 ng/l) in the water samples was observed at a location (Oxley creek) in the urban section of the River similar to BR-11 in this study. This work compares well with a similar study on sediments from the Paramatta River estuary in Sydney, Australia, which also showed PFOS dominance with a range of 0.8-6.2 ng/g dry weight and 1.5 ng/g dry weight geometric mean [27].

In this study, higher chain (C_8-C_{12}) PFASs were observed to be more present in the sediment compared to the lower chains indicating that apart from potential sources, partitioning coefficient could significantly affect the distribution of PFASs in the sediment-water interface. Munoz et al., (2017) observed that shorter chain (C_4-C_6) PFAS partition poorly onto soils and sediment. The higher concentrations of PFOS suggest that PFOS-containing materials have been mostly used and hence have greater inputs along the study area compared to PFOA, PFUnA and PFDoA. Deposition of PFASs contained in leachate from municipal landfills into water resources have also been reported [103]. Recently, PFASs were measured in landfill leachates from municipal waste sites across Australia [29]. Thompson *et al.*, (2011) inferred that PFASs burden in the Australian environment is potentially from localised use and disposal of PFASs-containing products as well as small scale industrial inputs. However,

apart from historic secondary manufacture of PFASs, there are no records of PFASs production in Australia [92]. PFASs can emanate from fire-fighting foams, pesticides, carpets, textiles, and cleaning products which are ubiquitous in urban environments [121]. Notably, PFOS and PFOA have been extensively applied in fire-fighting foams as well as stain repellents in non-stick cook-wares [26]. Elsewhere, urban runoff is identified as a major source of PFASs in Rivers [224, 225]. Considering the study area, the most likely contamination sources could be mainly effluents from WWTPs, stormwater or atmospheric dispersion from PFASs sources such as fire-fighting foams [226].

Generally, the spatial distribution of PFASs shows higher concentrations in areas characterised by urban (midstream) and agricultural (upstream) influences along the River (Fig. 5.1). The Fig. 5.1 shows the distribution of the high chain PFASs that were mostly measured above LOQ along designated sections of the sampling sites. Notably, the highest concentrations for PFOS (2.60 ng/g dry wt.), PFOA (0.38 ng/g dry wt.), PFUnA (0.44 ng/g dry wt.) and PFDoA (0.23 ng/g dry wt.) were measured at the midstream section of the River estuary. In Fig. 5.1, a general decline of PFASs is spatially observed from upstream (agricultural land and WWTPs) to downstream (located seawards). This seaward reduction in PFASs contamination is likely due to long-term mixing with less contaminated sea water and associated dispersed particles arising from higher tides downstream [227]. Thus Fig. 5.1 further suggests that PFASs inputs are from the use of household materials and agricultural products arising from waste discharges and through stormwater runoffs.

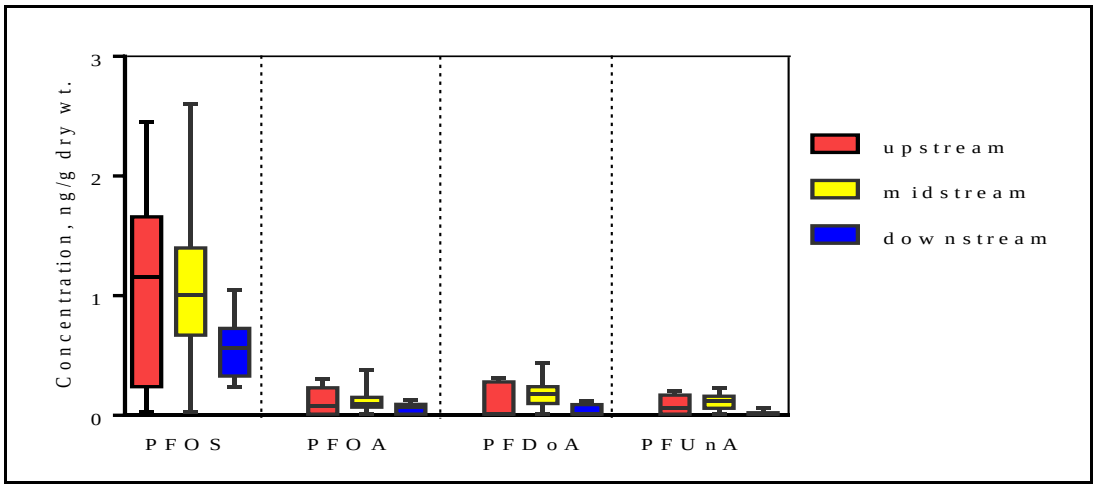


Fig. 5.1: A Box and Whisker plot showing patterns of PFASs distribution in sediment along the Brisbane River estuary. Maximum and minimum concentrations are represented by the upper and lower Whiskers respectively.

To help identify potential sources of PFASs contamination along the sampling points, the spatial distribution of PFOA and PFOS were presented in Fig. 5.2. Concentrations of PFOS along the sampling points show higher variability whereas a fair distribution is observed for PFOA contamination. The lower levels of PFOA relative to PFOS could be due to their relatively higher solubility in water. Similar to observations made by Gallen et al., (2014), the fair distribution of PFOA could also be due to mixing of potential PFOA sources caused by the major flood events in 2011 and 2013 [28] which preceded the sample collection in this work. Sampling point BR-11, in particular, suggests a point source contamination of PFOS. This sampling point is located within the urban area of the Brisbane River that has parklands and a University. Wastewater channelled for treatment at WWTPs can contain high levels of PFASs.

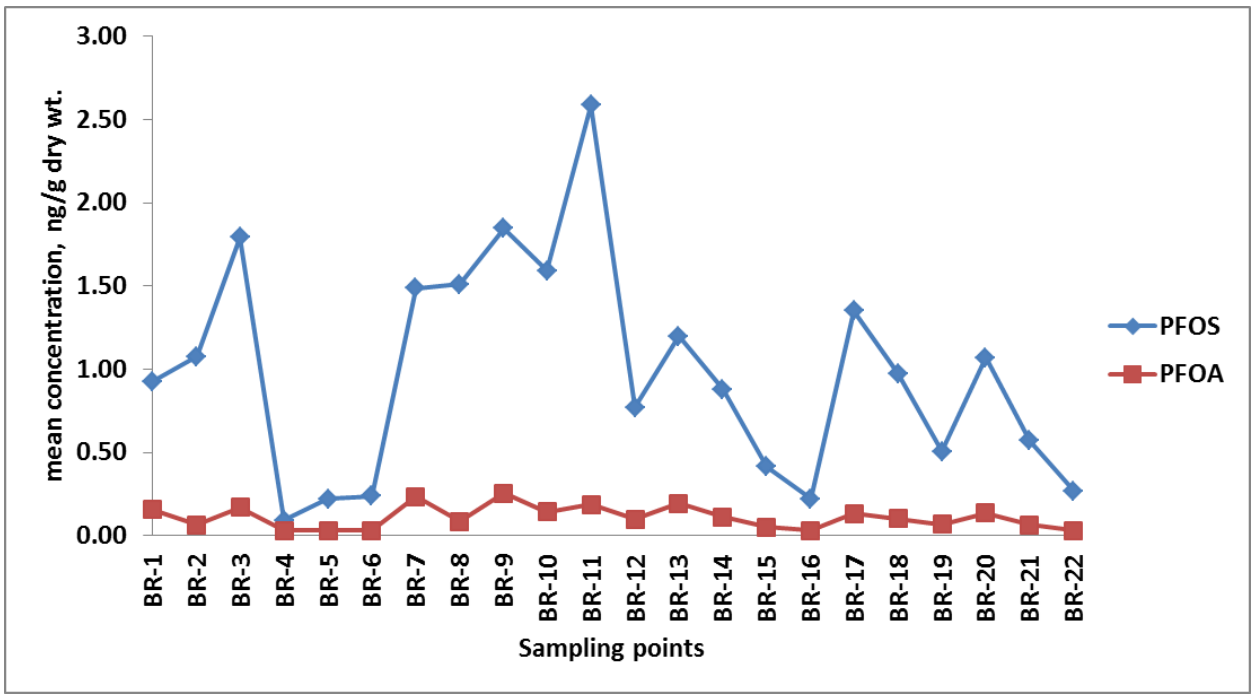


Fig. 5.2: Spatial distribution of PFOS and PFOA in the Brisbane River estuarine sediment

5.2.1.2 Water

This work presents data for water samples that were collected in 2017, about six years after a major flood event in 2011. Descriptive statistics for PFASs contamination in the water samples are presented in Table 5.1. The concentrations of some higher chain PFASs (PFNA, PDA, PFUnA and PFDoA) could not be quantified in the samples as their peaks were below

the respective limits of quantitation. This observation could mean that they have attached more unto sediment rather than dissolve into water due to higher partitioning coefficients.

Table 5.1: Summary of PFASs contamination in water samples from the Brisbane River estuary.

PFASs	LOQ (ng/L)	Qantitation (%)	ng/L								
			Min-Max	Mean	Std. Error	Std. dev.	nth Percentile at 95% CI				
							10^{th}	25^{th}	50^{th}	75^{th}	90^{th}
PFBA	0.1	25	<0.1-7.11	1.38	0.51	2.50	<0.1	<0.1	<0.1	3.14	6.97
PFPeA	0.1	83.3	<0.1-19.04	8.88	1.31	6.41	<0.1	3.24	9.66	15.30	18.05
PFHxA	0.1	29.2	<0.1-15.62	3.38	1.13	5.55	<0.1	<0.1	<0.1	9.56	14.18
PFHpA	0.1	62.5	<0.1-5.27	2.17	0.40	1.97	<0.1	<0.1	2.59	4.65	5.07
PFOA	0.5	83.3	<0.05-14.39	7.42	0.92	4.49	<0.05	3.33	9.17	11.19	12.26
PFNA	0.1	-	<0.1	-	-	-	-	-	-	-	-
PFDA	0.06	-	<0.06	-	-	-	-	-	-	-	-
PFUnA	0.05	-	<0.05	-	-	-	-	-	-	-	-
PFDoA	0.06	-	<0.06	-	-	-	-	-	-	-	-
PFBS	0.05	12.5	<0.05-5.51	0.54	0.31	1.53	<0.05	<0.05	<0.05	<0.05	4.52
PFHxS	0.08	45.8	<0.08-23.96	8.29	1.97	9.65	<0.08	<0.08	<0.08	19.45	23.47
PFOS	0.05	45.8	<0.05-46.77	13.71	3.47	17.01	<0.05	<0.05	<0.05	29.18	43.44

Results obtained from this work (Table 5.1) shows an increase in PFASs contamination in the estuary relative to the earlier study by Gallen et al., (2014) which was based on samples collected in 2011 following a major flood event [26]. While Gallen et al., (2014) reported concentrations of 0.13-6.6 ng/L (PFOA) and 0.18-15 ng/L (PFOS), the PFOA and PFOS in this study were measured in the ranges: <0.05-14.4 ng/L and <0.05-46.8 ng/L respectively. This shows an increase in PFASs contamination of the estuary up to factors of about 2 (PFOA) and 3 (PFOS) since the maiden study. The observed increase is indicative of continual inputs of PFASs compounds into the River, likely from effluents or stormwater.

Tributaries located upstream of the River could also be carrying contaminants from distant localities into the Brisbane River.

The spatial distribution for PFASs contamination in the water samples is presented in Fig. 5.3. Although PFOS concentrations (Fig. 5.3 (f)) were higher than PFOA (Fig. 5.3 (d)) at locations where both contaminants were measured, the occurrence of PFOA was more abundant across the sampling points (Table 5.1and Fig. 5.3) indicating the higher solubility of PFOA in water.

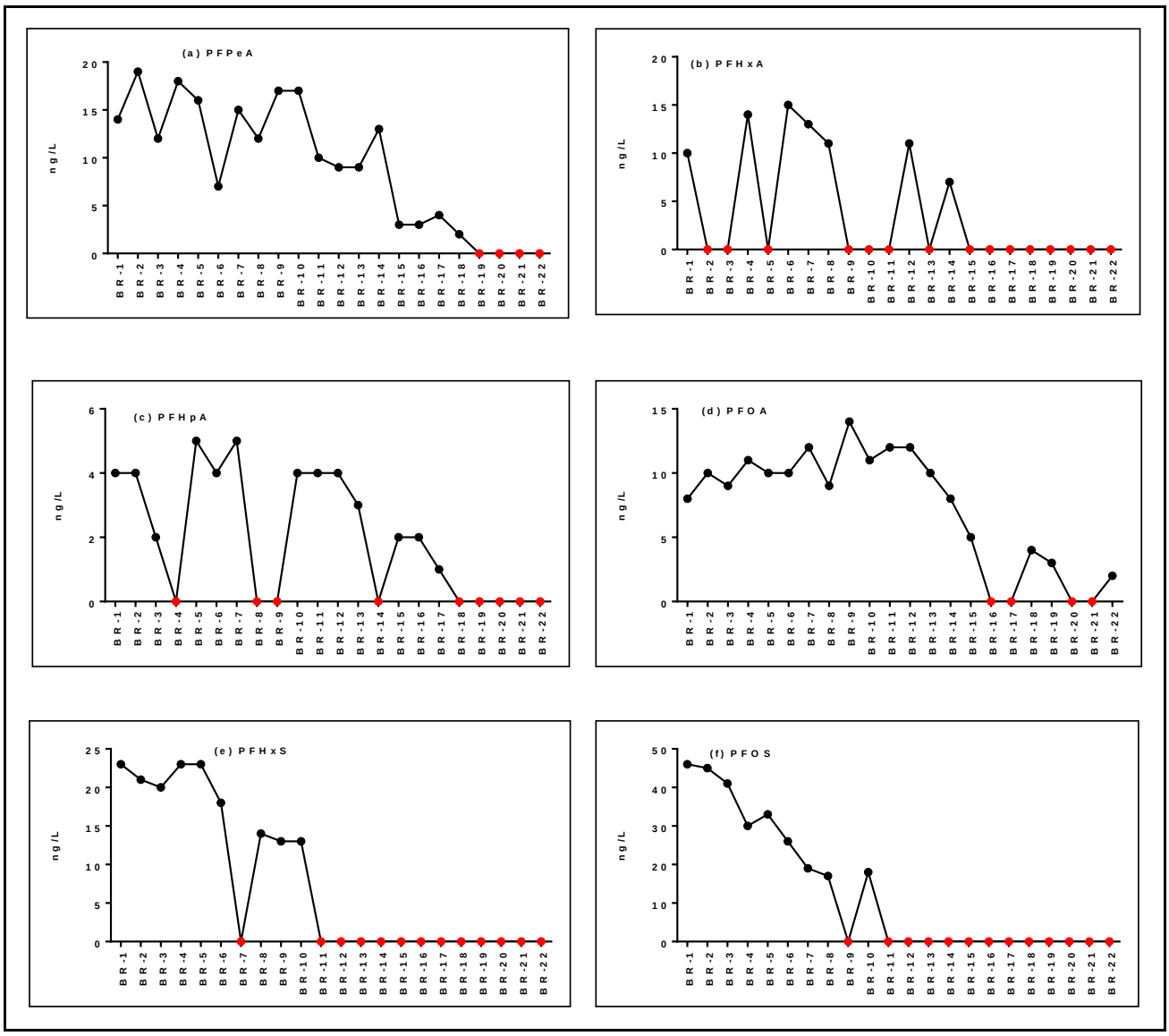


Fig. 5.3: Mean concentration of PFASs; (a) PFPeA, (b) PFHxA, (c) PFHpA, (d) PFOA, (e) PFHxS and (f) PFOS in the water samples across the sampling points along the Brisbane River estuary. The points plotted in red show measurements that were below limit of quantitation.

Generally, spatial distribution of PFASs contamination in the water samples were similar to the sediments showing higher concentrations at the upstream (BR-1-BR-9) and midstream (BR10-BR-18) sections while water samples collected downstream (BR-19-BR-22) were always low or below limits of quantitation. There is the likelihood of more dilution processes at the downstream section due to greater volumes of sea water intrusion at the mouth of the River. The lower concentrations in the downstream samples could also suggest that industrial activities are not the main sources of PFASs contamination in the estuary but rather from upstream where agricultural and urban activities are prevalent.

5.2.2 National and International comparison of PFOS contamination in sediments

The PFOS levels in the Brisbane River sediment compared well with levels reported for sediment in some parts of China, Japan and Spain. Sediments from the Llobregat River in Catalonia which is a drinking water resource to the Barcelona city had PFOS levels of 0.01-3.67 ng/g dry weight [228] whereas 0.1- 4.8 ng/g dry weight (PFOS) were measured in samples from the L'Albufera natural park in Valencia, Spain [229]. In parts of China, PFOS measured in sediment from the Taihu Lake [230] and the Baiyangchian Lake[20] were 0.06-0.31 ng/g dry weight and 0.06-0.64 ng/ng dry weight, respectively. It is; however, worth noting that the Baiyangchian Lake is a receptor of industrial wastewater from the Fuhe River, thus there is a need to periodically monitor PFASs in the Brisbane River since it has no such direct industrial influents. Also in China, ∑PFASs (0.09 - 3.6 ng/g dry weight) in sediments from the Zhujiang River which also passes through the urban city of Guangzhou [181] were consistent with observations in this work. In Japan, sediments from the Ariaka Sea also show comparable levels of PFOS (0.09-0.14 ng/g dry weight) and PFOA (0.84-1.1 ng/g dry weight) [231]. Sediments from the remote Antarctic Peninsula showed no PFASs levels above the method limit of quantitation [15], indicating that industrialisation and urbanisation are the major input of PFASs even though long range atmospheric transport (LRAT) is also possible.

Higher levels of PFASs were measured in sediments in some other parts of the world where the River systems were impacted by industrial activities. Sediments collected from downstream of a PFASs manufacturing and wastewater treatment plant along the Mississippi

River in USA, showed high levels of PFOS (1.7-27.9 ng/g dry weight) [103]. Similarly, industrial discharges have led to PFOA (5.20-203 ng/g dry weight) and PFOS (1.6-8.8 ng/g dry weight) contamination of sediments collected from the Huangpu and Suzhou Rivers in Shanghai respectively [232]. Interestingly, PFOA in the Huangpu and Suzhou River sediments were higher than the PFOS levels indicating that even though partition coefficient favours sorption of PFOS in sediment relative to PFOA, source of contamination and load discharged into the River also plays a major role. Li et al. (2010) identified the discharges from the polytetrafluoroethylene (PTFE) manufacturing plant sited in the Yangtze River Delta as the source of PFOA contamination. This is similarly observed in the Ganges (Hugli) River sediments where concentrations of PFOA were < 0.50-14.1 ng/g dry weight whilst PFOS were <0.50 ng/g at all sampling sites [233]. Further, Xie et al. (2013), acknowledged that the production of PFASs in China for both local and international consumption has accounted for an estimated 70 tonnes of PFOS emission in 2010 [121]. Thus compared to international data, PFASs in the Brisbane River estuary sediment were in the lower ranges of concentration given that $\sum_{12}$PFASs were between < LOQ to 3.6 ng/g dry weight (Table B-5.1).

5.3 Summary

A suite of PFASs are persistent in the Brisbane River estuary. It is worth noting that concentrations in the Brisbane River sediments were comparable to concentrations reported for sediment in Sydney explaining common sources of PFASs contamination in Australian estuarine sediments. The distribution along the study area shows contamination from urban and agricultural environments rather than industrial sources. Thus non-point sources together with mixing of the River estuary arising from hydrodynamics have potentially affected the spatial distribution of PFASs in the water and sediments. This work thus bridges the data deficit on PFASs contamination of sediments in the Brisbane River and also highlights some PFASs contamination sources along the study area. The findings provide necessary inputs for future work along the estuary such as assessing the fate of PFASs contamination.

Chapter 6: Occurrence and fate of target pharmaceuticals, personal care products and pesticides in the Brisbane River estuary

The contamination of Rivers by PPCPs and pesticides is ever increasing due to the continuous inputs from effluents of WWTPs, stormwater channels and surface run-off from agricultural and parklands. Nonetheless, studies on the chemical behaviour of these contaminants in the Brisbane River estuary are very limiting. Although salinity is a good marker for tracing the fate of contaminants in estuaries, its effect on the behaviour of PPCPs and pesticides in the Brisbane River estuary has not been investigated in the literature until this study. Aside from presenting a baseline data on PPCPs and current status of pesticide contamination, this chapter discusses the fate of these contaminants in the Brisbane River estuary along land-use transects. Further, chemical candidates that can be useful for biomonitoring along the River estuary were determined based on their conservative behaviour influenced by salinity. A background highlighting the need for this study and the objectives that defined the study are presented in Section 6.1. Occurrence, spatial distribution and behaviours of the contaminants along the estuary using salinity as a benchmark property are discussed in detail in Section 6.2. The research findings in this study are summarised in Section 6.3.

6.1 Background

Some current-use pesticides (CUPs), pharmaceuticals and personal care products (PPCPs) are ubiquitous, potentially bio-accumulative and are emerging contaminants. Applications of CUPs (herbicides, insecticides and fungicides) to improve agricultural yield (plants and animals), as well as the use of PPCPs (antibiotics, hormones, analgesics, blood lipid regulators, cytostatic drugs and antiepileptic) for therapeutic and cosmetic purposes have resulted in environmental contamination, including water bodies [107, 114-117, 234, 235]. CUPs in receiving waters can bio-accumulate in non-target aquatic fauna and cause potential

adverse effects to the aquatic organisms, as well as humans via the food chain [107, 108]. While pesticides can be washed off into rivers via stormwater drains, or surface runoff (from backyards, farms and parklands), pharmaceutical products, which are often not fully metabolised in the body, end up in wastewater streams as parent compounds, or metabolites through human and veterinary excretion [4, 108, 117, 236]. Similarly, personal care products (insect repellents, UV-filters, anti-microbials or surfactants [107]) also enter the sewage stream as these compounds are washed off during bathing [117], or directly into rivers during recreational swimming. Once these compounds are released into water ways, they may be transported across a wide salinity gradient into both fresh and marine environments. For example, the pharmaceutical product, carbamazepine, is conserved in both fresh and saline waters and hence does not significantly breakdown [4, 108] along the water course. Nonetheless, some breakdown routes, such as biodegradation, photodegradation or adsorption to particles and subsequent sedimentation is possible for some contaminants [109, 110]. Even though waste streams are channelled through wastewater treatment plants (WWTPs), significant amounts of the contaminants are not removed by treatment processes and are continuously transported into river and estuarine systems via effluent discharges.

Although increasing contamination of CUPs and PPCPs in both fresh and marine water bodies from other parts of the world at concentrations of ng/L to µg/L have been reported in literature [5, 237-244], investigations of the occurrence and distribution of these contaminants in Australian receiving waters are relatively limited [4, 41, 117] to date. The few studies conducted, however, showed evidence of the presence of these emerging contaminants in water bodies. For example, Birch *et al.* (2015) measured diuron (a herbicide) in Sydney estuarine waters up to 3100 ng/L, while the concentrations of fluoxetine (anti-depressant) and acesulfame (food sweetener) were up to 36 ng/L and 114 ng/L respectively. In Sydney estuary, the source of these contaminants was leakage from stormwater systems. Similar to findings from other parts of the world, studies in some parts of Australia also agree that WWTPs are contributing sources of these emerging contaminants in receiving waters due to their presence in both influent and effluent materials [245-249].

In Australia, sewage and wastewater are channelled through treatment plants before discharge into waterways. The waste streams can be generated from domestic households, industries, hospitals, commercial farmlands or aquaculture. Nonetheless overflows or leakages may

occasionally arise during high rainfall, storm seasons, or major flood events [26, 117]. Stormwater, however, discharges into the river systems throughout the year and is thus a potential route of pesticide contamination from agricultural fields, parklands and households with backyard lawns.

In this study, the Brisbane River estuary (Queensland) was investigated for contamination of CUPs and PPCPs. Estuaries vary from freshwater bodies to saline waters that are freely connected to the open sea resulting in characteristic tidal flows and salinity gradients [250]. This connection creates a mixing interface between rivers and the sea for some chemicals, where salinity may be used as a tracer to assess the conservative behaviour of contaminants [111]. Even though the Brisbane River is an estuary that presents a pronounced salinity gradient, investigation of the fate of contaminants using salinity as a marker in the estuary have not been cited in the literature. The estuarine waters sampled in this study receive surface runoff via stormwater drains, as well as effluents from sewage/wastewater treatment plants that are sited along the river. Land adjacent to sampling sites are also characterised by varying land-use, including agricultural, residential and commercial, and industrial. Water samples were collected in December 2017 when there were no major rains in the preceding 4 months. This results in a longer residence time of the contaminants in the estuary, and also ensures a pronounced salinity gradient that could be used to interpret the sources and degradation of contaminants along the estuary.

With growing population and subsequent urbanisation, the incentive to utilise CUPs for increased food production and the use of PPCPs to improve physical health and socio-psychological well-being will continue to increase with associated contamination of river estuaries. Accordingly, this research sought to achieve the following: (1) assess the distribution of pharmaceuticals and pesticides in estuarine waters, (2) use salinity as a property to assess fate of PPCPs and CUPS, (3) identify pharmaceutical biomarkers of WWTPs as a means of verifying the efficiency/integrity of sewage/wastewater treatment, (4) verify potential contribution of stormwater (drains, parklands, farms) to pesticide contamination. Knowledge from this study is necessary to understand the fate and potential risk of these environmental contaminants.

6.2 Results and Discussions

6.2.1 Quantitation frequency of target analytes

A total of 82 chemical compounds were targeted for analysis in the water samples. The raw data is presented in Appendix C (Tables C-6.1 and C-6.2). Descriptions and a summary of the analytes that were quantified are presented in Table 6.1, while the descriptions of the analytes below limits of quantitation and therefore could not be quantified were presented in Table C-6.3 (Appendix C). Out of the 25 pharmaceuticals analysed, 9 (atorvastatin, fluoxetine, naproxen, sildenafil, verapamil, hydroxycotinine, ibuprofen, furosemide, caffeine) were below limits of quantitation in all samples. Thus 16 pharmaceuticals were detected in at least one sample ranging between 5% (nicotine) and 100 % (carbamazepine, gabapentin, tramadol, iopromide, venlafaxine, and temazepam). The quantitation of target CUPs in at least one sample was 23 out of 53. Quantitation frequencies of the CUPs were in the range 23% (DCPMU, a soil degradation product of diuron) to 100 % (atrazine, diuron, metolachlor, simazine, imidacloprid, hexazinone, tebuconazole, simazine hydroxyl, 2,4 D, and ametryn hydroxy). Although 3 personal care products (DEET, triclosan and salicylic acid) were analysed in the samples, the detection of DEET could not be confirmed due to poor chromatograms for the second transitions, whereas salicyclic acid contents were below quantitation levels after blank corrections. Triclosan was quantified in 18% of the samples.

Table 6.1: List of target analytes quantified in at least one sample, with descriptions and concentration ranges. The group of target analytes are: (A) Pharmaceuticals, (B) Pesticides and (C) Personal Care Products.

(A) Pharmaceuticals			
Analyte	***Use***	***quantitation frequency(%)***	***min-max(ng/L)***
Paraxanthine	Stimulant of the central nervous system and a caffeine metabolite)	86	<1.2-10.5
Atenolol	Lowers high blood pressure, prevents stroke, heart attacks, kidney problems	50	<2-7.7
Carbamazepine	Treats epilepsy and neuropathic pain	100.00	5.3-106.4
Citalopram	Treats depression	18	<1-2.6
Codeine	Depressant	5	<0.1-3.0
N-Desmethylcitalopram	Metabolite of antidepressant drugs citalopram and escitalopram	14	<2-5.0
N-Desmethyldiazepam	Amnesic, sedative, muscle relaxant properties and a metabolite of diazepam (Valium)	27	<1-1.9
Gabapentin	Treats partial seizures in adults and children, nerve pain	100	<7.8-117.6
Iopromide	X-ray contrast medium which permits radiographic visualization of internal organs	100	3.9-94.3
Paracetamol	Mild pain relief	45	<1-6.0
Temazepam	Treats short-term sleeping problems (insomnia)	100	2.5-37.8
Tramadol	Pain relief	100	3.1-81.1
Venlafaxine	Treats depression and social anxiety disorder	100	4.5-86.2
Cotinine	Metabolite of nicotine	9	<1-9.6
Nicotine	Stimulant	5	<1-1.6
Hydrochlorthiazide	Lowers high blood pressure	45	<1-31.7

	(B) Pesticides (herbicide, fungicide, insecticide)		
Analyte	***Use***	***Quantitation frequency (%)***	***min-max(ng/L)***
Tebuconazole	Fungicide (plant pathogenic fungi)	91	<1-16.5
Fluroxypyr	Herbicide(broadleaf weeds and woody brush)	50	<5-21.2
Atrazine	Herbicide (control weeds in summer crops: maize, sorghum, sugarcane)	100.0	1.7-39
Clopyralid	Herbicide (control broadleaf weeds)	73	<2-19
Desethyl Atrazine	Herbicide (metabolite of atrazine)	59	<1-5.1
Desisopropyl Atrazine	Herbicide (metabolite of atrazine: controls broadleaf weed for maize, sugarcane, golf courses, residential lawns)	68	<1-5.5
Diuron	Herbicide	100	6.3-56.7
Hexazinone	Herbicide (weeds and woody plants for mostly on non-crop areas)	96	<1-10.7
Imazapic	Herbicide (broadleaf weed and grass in pasture, rangeland etc)	68	<1-9
Imidacloprid	Insecticide (control sucking insects, termites, soil insects and fleas on pets)	96	<1-46
Metolachlor	Herbicide (broadleaf weeds and grass)	100.0	4-128.2
Metsulfuron-Methyl	Herbicide (broadleaf weeds and grasses)	64	<5-12.3
Picloram	Herbicide (control woody plants, broadleaf weeds)	77	<5-28
Propiconazole	Fungicide (cereals and stone fruit)	55	<2-7
Simazine	Herbicide(broadleaf weeds and annual grass)	100	1.5-34.2
Terbuthylazine	Herbicide	59	<10-32
Simazine hydroxy	Herbicide (broadleaf and annual grass)	91	<1-8

DCPMU	Pesticide (soil degradation product of diuron)	23	<1-2.5
Ametryn hydroxy	Herbicide (metabolite of atrazine)	96	<1-12.8
MCPA	Herbicide (2-methyl-4-chlorophenoxyacetic acid, control weeds in cereals, rice, pastures)	73	<5-68
2,4 D	Herbicide (2,4-Dichlorophenoxyacetic acid)	100	4.2-58.6
Triclopyr	Herbicide (broadleaf weeds, woody weeds and melon)	41	<50-226.4
Haloxyfop	Herbicide	68	<1-11
	(C) Personal Care Products (PCP)		
Analyte	***Use***	***Quantitation frequency (%)***	***min-max(ng/L)***
Triclosan	Anti-bacterial/antifungal (toothpaste, soaps, detergents, surgical cleaning treatments)	18	<1-2.8

6.2.2 Occurrence of contaminants

PPCPs

Descriptive statistics of the pharmaceuticals with quantitation ≥ 50 % across all sampling points is presented in Table 6.2, with the respective 25th, 50th, 75th and 90th percentiles. The mean concentrations of analytes with quantitation frequencies ≥ 50 % across the sampling points show that carbamazepine (46 ng/L), gabapentin (42 ng/L), iopromide (28 ng/L), tramadol (26 ng/L) and venlafaxine (24 ng/L) were the most dominant pharmaceuticals in the Brisbane river estuary.

Table 6.2: Descriptive statistics of pharmaceuticals with quantitation frequencies ≥ 50 % across the 22 sampling points.

PPCPs and Food additive	Quantitation Frequency(%)	Mean ng/L	Std.dev ng/L	Min ng/L	Max ng/L	nth Percentile				
						10th	25th	50th	75th	90th
Paraxanthine	86	6.0	2.5	1.2	11	1.4	3.9	6.9	7.7	8.3
Atenolol	50	3.6	2.5	<2	7.7	<2	<2	3.6	6.1	6.7
Carbamazepine	100	46	30	5.3	106	8.8	13.2	48	73	78.1
Gabapentin	100	42	34	7.8	118	9.1	12.9	34	61	94.2
Iopromide	100	28	25	3.9	94	4.6	6.9	18	42	61.2
Temazepam	100	16	10	2.5	38	3.5	5.1	17	26	26.7
Tramadol	100	26	20	3.0	81	4.4	6.8	24	39	47.0
Venlafaxine	100	24	19	4.5	86	5.1	7.5	23	35	37.5

When compared to earlier results for the Sydney river estuary reported by Birch *et al.*, (2015), mean concentrations of pharmaceuticals in the samples were always higher by order of magnitude, with the concentrations reported in this work higher, by the following order of magnitude: 42 (carbamazepine), 6 (iopromide) and 5 (venlafaxine). This could be due to to lower dilution in the Brisbane River as it is a smaller estuary than the Sydney River estuary. Alternative, the Brisbane River could be receiving higher inputs of pharmaceutical contaminants via higher rate of effluent discharges. Triclosan, a personal care product, which is used as an anti-bacterial in a wide range of products (toophpaste, soaps, detergents, and

surgical cleaning treatments) was; however, less present or at lower concentrations in Brisbane River (maximum concentration 2.8 ng/L) (Table 1).

CUPs (herbicides, fungicides and insecticides)

Descriptive statistics for the CUPs with the nth percentile concentrations are presented in Table 6.3. Notably, atrazine, diurion, metolachlor, simazine and 2,4 D were detected and quantified in all the samples. The mean concentrations of the dominant CUPs with > 50% detection rates across all sampling points were in the order: 50 ng/L (metolachlor) > 33 ng/L (2,4 D) > 28 ng/L (diuron) > 22 ng/L (imidacloprid) > 21 ng/L (atrazine) > 20 ng/l (MCPA) > 17 ng/L (simazine) > 15 ng/L (picloram) > 10 ng/L (clopyralid).

Table 6.3: Descriptive statistics of CUPs with quantitation frequencies ≥ 50 % across the 22 sampling points

Current-use pesticides (CUPs)	Quantitation Frequency(%)	Mean ng/L	Std.dev ng/L	Min ng/L	Max ng/L	n^{th} Percentile				
						10^{th}	25^{th}	50^{th}	75^{th}	90^{th}
Tebuconazole	91	6.7	4.9	<1	17	1.5	2.5	5.6	11	14
Fluroxypyr	50	7.6	6.1	<5	21	2.5	2.5	4.5	11	16
Atrazine	100	21	11	1.7	39	4.6	13	22	29	33
Clopyralid	73	9.8	6.7	<2	19	<2	2.1	9.4	16	18
Desethyl Atrazine	59	2.0	1.4	<1	5.1	<1	<1	2.2	2.8	3.4
Desisopropyl Atrazine	68	2.8	1.9	<1	5.4	<1	<1	3.3	4.5	5.1
Diuron	100	28	14	6.3	57	10	18	29	37	45
Hexazinone	96	6.0	3.2	<1	11	2.0	3.4	6.0	9.3	10
Imazapic	68	3.5	2.9	<1	9.0	<1	<1	2.6	5.6	7.7
Imidacloprid	96	22	15	<1	46	3.0	10	19	33	41
Metolachlor	100	50	36	4.1	128	8.3	20	55	63	84
Metsulfuron-Methyl	64	6.5	3.5	<5	12	<5	2.5	7.0	9.0	10
Picloram	77	15	9.5	<5	28	<10	6.3	17	24	25
Propiconazole	55	3.2	2.2	<2	6.9	<2	1.0	3.3	5.0	6.2
Simazine	100	17	11	1.5	34	3.9	7.7	19	24	31
Terbuthylazine	59	14	9.4	<10	32	<10	<10	13	23	27
Simazine hydroxy	91	4.5	2.1	<1	8.0	1.7	3.5	5.0	6.0	6.9
Ametryn hydroxy	96	7.9	3.9	<1	13	2.2	5.3	10	11	11
MCPA	73	20	21	<5	68	<5	3.4	14	27	54
2,4 D	100	33	18	4.2	59	11	16	33	50	53
Triclopyr	41	73	67	25	226	25	25	25	134	170
Haloxyfop	68	4.3	3.7	<1	11	<1	<1	3.4	7.5	9.6

To obtain more information on the possible sources of the CUPs, the spatial distribution of the chemicals along the River were examined more closely in the following section.

6.2.3 Spatial distribution and effect of salinity on the fate of contaminants in Brisbane River estuary

Although direct measurement of chemical behaviour in environmental matrices is possible, the complications associated with spatiotemporal variabilities may be avoided by using bench-markers [251]. Bester at al., (1998) used the linear dependency of salinity gradient and concentration to assess the conservative behaviour of 2,5-dichloroanaline in a German river estuary [112]. The use of salinity to assess the conservative transport of contaminants is based on the assumption that the Brisbane River estuary is at a steady state at the time of sample collection over periods greater than the flushing time of an estuary [111, 252]. This is possible due to the flushing time of about 120 days for the Brisbane River and the fact that the samples were collected at a time when there were no preceding major rainfalls within the flushing time of the estuary. Thus, there were no major inflows to alter the mixing of the estuary. To deduce the behaviour of contaminants in the Brisbane River estuary, a modified mixing plot model shown in Fig. 6.1 was applied to interpret the spatial distribution and fate of the contaminants along the salinity gradient. This modified mixing plot model is triangular and can be used to assess conservative contaminants from two directions with respect to the contaminant source (Fig. 6.1) which is an advantage over the linear mixing plot model used by Bester et al., (1998). The curves were plotted between the two end-members (the least salinity and the highest salinity along the River) and also the point along the River where the concentration of contaminant was highest (major input). Thus, the definitions in Fig. 6.1 were used to identify whether the contaminants behave conservatively, or not as well as to identify sources along the River.

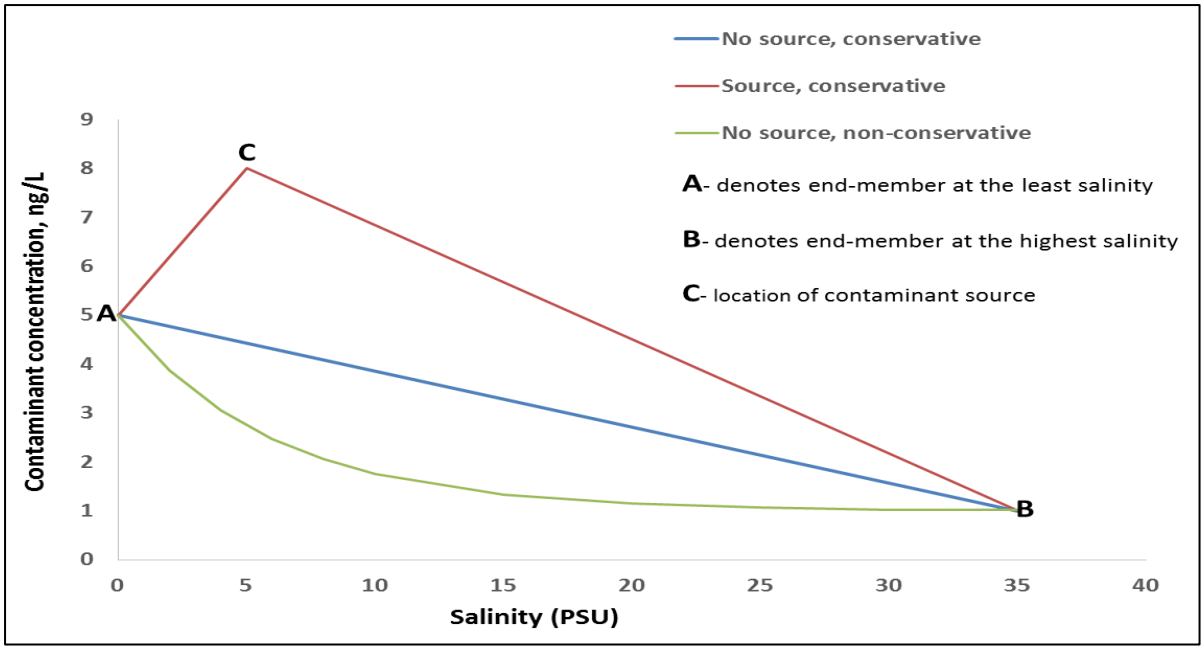


Fig.6.1:A modified mixing plot model illustrating the behaviour of conservative and non-conservative contaminants in estuarine waters as well as the influence of a contaminant source along the estuary on the mixing plot. Source in the legend means contaminant input along the estuary.

Pharmaceuticals

It is hypothesised that the major source of pharmaceutical products in Rivers is via WWTPs. Fig. 6.2 presents mixing plots of the variations of the concentrations of the pharmaceuticals along sampling points in the estuary (BR-1 to BR-22) with respect to salinity. Generally, the distribution of pharmaceutical products in the Brisbane River estuary shows high concentrations at BR-4. This satisfies the hypothesis since there is a major WWTP (Fig. 3.1) at point BR-4, which releases effluents into the River. This is suggests that wastewater streams and sewage in Brisbane are channelled for treatment at WWTPs. Therefore, the efficiency of these treatment plants as secondary contaminant sources should be an area of focus and scrutiny. The presence of carbamazepine (5 –106 ng/L) and gabapentin (7.8-118 ng/L), known bio-markers of effluents, indicates that the River could indeed be receiving these contaminants via leakages from the WWTP, or discharges of effluents. Carbamazepine (an anti-epileptic medication) and gabapentin (anticonvulsive medication) can both be excreted by human subjects and find their way into sewage and wastewater streams [247].

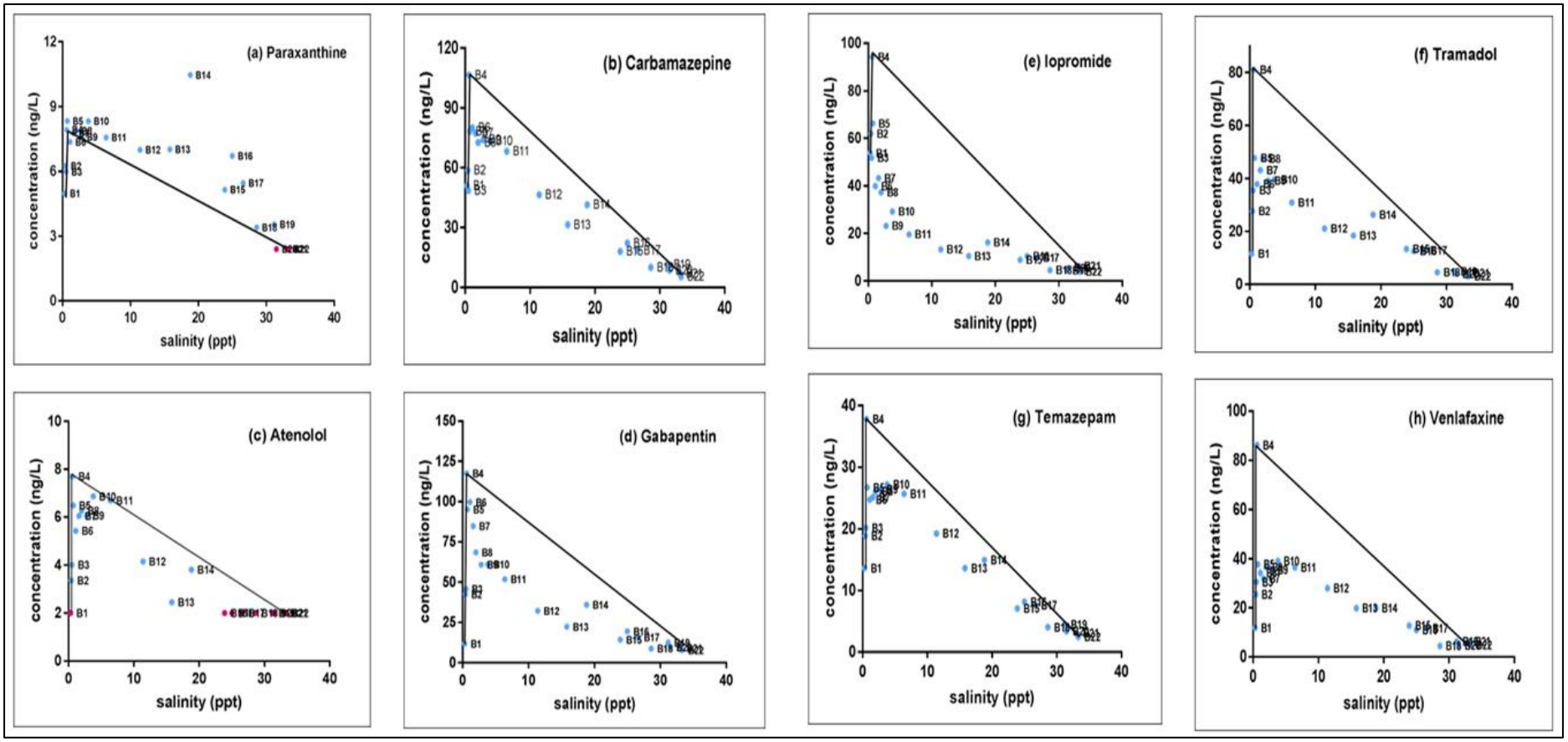


Fig. 6.2: A mixing plot illustrating the fate of 8 pharmaceuticals: (a) Paraxanthine, ((b) Carbamazepine, (c) Atenelol, (d) Gabapentin, (e) Iopromide, (f) Tramadol, (g) Temazepam, and (h) Venlafaxine, along the Brisbane River estuary. The labels B1 to B22 on the plot represent sampling points BR-1 to BR-22 respectively. The lines were drawn using the end-members at lowest salinity (0.3 PSU) and highest salinity (33.3 PSU) with point B4 hypothesised as the major source of contamination. Plotted points in red indicate the respective limits of quantitation (LOQ) since the concentrations in the respective samples were < LOQ.

Nevertheless, the concentration of paraxanthine was highest at point BR-14 (Fig. 6.2 (a)), where there is no WWTP and this does not support the hypothesis. Thus, the noticeable increase in the concentration of paraxanthine at BR-14, which is located between a University and an urban community, could be due to factors such as leakages from wastewater channels (pipes).

Comparison of the mixing plots in Fig. 6.2 with the interpretation in Fig. 6.1, shows that the pharmaceuticals: atenolol (Fig. 6.2 (c)), gabapentin (Fig. 6.2 (d)), iopromide (Fig. 6.2 (e)), tramadol (Fig. 6.2 (f)) and venlafaxine (Fig. 6.2 (h)) appear to be non-conservative with no additional sources as they move from upstream (with BR-4 as the major source) towards downstream (BR-22). Apart from BR-4, there are no further inputs of these chemicals along the River. Both venlafaxine and tramadol may potentially undergo photo-degradation in the presence of strong UV light [253]. While gabapentin, an anticonvulsant, can undergo slow degradation at room temperature and at a pH of 6 in aqueous medium [254], iopromide (an X-ray contrast medium) can undergo both photolytic [255] and microbial degradation [256-258]. Nonetheless, the observed non-conservative behaviour of gabapentin could also be due to pseudo-degradation processes such as the uptake by aquatic organisms [106]. Thus, gabapentin could be re-distributed into the estuarine by the aquatic organisms when they are dead. The apparent non-conservative behaviour of atenolol in Fig. 6.2 must be accepted with caution since the concentration of atenolol could not be quantified in 50% of the samples and therefore would present a significant uncertainty in that data set.

Although carbamazepine (Fig. 6.2 (b)) and temazepam (Fig. 6.2 (g)) appear to be non-conservative, a steady dilution trend can be observed as these chemicals move downstream (BR-22) where concentrations were lowest. Thus, within the margins of measurement uncertainties, carbamezepine and temazepam can be classified as conservative chemicals in the River (Fig. 6.2). This conservative tendencies of carbamazepine and temazepam could be due to their potential resistance to biodegradation [259].

Remarkably, paraxanthine (a metabolite of caffeine) shows a conservative behaviour, but with multiple source inputs notably at the midstream section indicating the potential to be present in the River over a longer period (Fig. 6.2 (a)). Although the concentration of paraxanthine is relatively high at points BR-4 and BR-6 where WWTPs are located, the relatively higher concentrations at midstream (sites BR-10 to BR-17) with the highest

concentration at BR-14 (10.5 ng/L) where there are no WWTPs is indicative of non-conventional inputs of caffeine along the River (i.e. other than via WWTPs alone). Sampling locations along the midstream section of the River hosts recreational parklands. Therefore, the presence of paraxanthine could be due accidental spillages of coffee beverages on the park by patrons, which could subsequently be washed into the River during precipitation events.

In general, it appears the major source of pharmaceutical products in the River is effluent from the WWTP around sampling point BR-4, which is located upstream.

Current use pesticides (CUPS)

The variation of salinity with the concentrations of CUPs along the sampling points of the Brisbane River estuary is presented on the mix plots in Fig. 6.3. The inputs of CUPs along the River vary between upstream and midstream sites. With the exception of diurion, samples collected at the downstream section had lower concentrations of the CUPs (Fig. 6.3). The relatively lower concentrations of most CUPs in the downstream section (BR-18 to BR-22), apart from diuron, can be attributed to lower inputs or high measurement uncertainties for the chemicals that were below limits of quantitation (Fig. 6.3). Apart from the CUPs, atrazine, desethyl atrazine, desisopropyl atrazine, metolochlor, MCPA and simazine, which have maximum concentrations at upstream sites where there are farmlands and agricultural activities, all other CUPs have maximum concentrations at the midstream section (particularly at sampling points BR-10 and BR-11) where there are active parklands. (Fig. 6.3 & Fig. 3.1). The concentration of metolachlor is highest (128 ng/L) at BR-1 (upstream) before reducing gradually to 4 ng/L downstream (BR-22) where there are mostly industrial activities, few parklands and no agricultural lands. The higher concentrations of metolachlor upstream, which is consists predominantly of agricultural lands (crop and animal grazing fields) coupled with parklands and backyard lawns is indicative of the application of herbicides in that transect. However, the distribution of other pesticides (atrazine, clopyralid, diuron, imidacloprid, picloram,simazine, MCPA and 2,4 D) suggests direct surface wash-offs from farmlands, lawns and backyards into the Brisbane river as well as inputs from herbicides from roadside lawns via stormwater drains.

To further understand the spatial distribution and the effect of salinity on the behaviour of the CUPs, the mix plots in Fig. 6.3 were examined based on the interpretations presented in Fig. 6.1. However, some limitations may arise from compounds that could not be quantified in >20 % of the samples with respect to sampling points as this may increase the uncertainties. Consequently, the behaviour of CUPs such as fluoroxypyr, cloppyralid, desethyl atrazine, desisopropyl atrazine, imizapic, metsulfuron-methyl, picloram, propiconazole, terbuthylazine, MCPA and haloxyfop, presented in Fig. 6.3, should not be accepted with caution since > 20 % of the samples were below limits of quantitation. Only the CUPs with quantitation frequencies >80% across all sampling points were interpreted for their behaviour along the River. The behaviour of terbuconazole, simazine and 2,4 D were shown to be conservative with no additional inputs along the River. This means that they are being diluted at a fairly steady ratio along the river, indicating their suitability as markers. Degradation of simazine in water, for example, has been observed to be poor [260]. While the concentrations of terbuconazole and 2,4 D were maximum at BR-10 (midstream section), the maximum concentration for simazine was at BR-4 (upstream section with agricultural influence). These observations agree with their patterns of their respective applications along the River. Terbuconazole is used to suppress fungal growth mainly at parklands, whereas simazine is mainly used as an herbicide in farming areas to control broadleaf weeds and annual grass. The behaviour of metolachlor, which has maximum inputs at the upstream section, was observed to be non-conservative along the River. Since there are no further inputs along the River apart from the upstream source, its behaviour could be attributed to its ability to undergo biodegradation.

Other CUPs such as imidacloprid, hexazinone, simazine hydroxyl, ametryn hydroxyl and atrazine also showed conservative behaviour, but with additional source inputs, apart from those from the major point sources. Additional source inputs were mainly observed at the midstream section and could be due to contributions from surface runoffs from the parklands. The similar behaviour of atrazine and its degradation product ametryn hydroxyl suggests on-going application of atrazine.

Interestingly, the concentration of diuron is higher downstream (high salinity transect) compared to upstream (low salinity transect), indicating diuron contamination of the sea. This

observation is in agreement with earlier findings where diuron was measured in samples from the Great Barrier Reef, a marine environment [261].

Generally, inputs of the pesticides into the Brisbane River are likely due to discharges from stormwater drains and/ or from direct surface run-offs from agricultural and recreational fields.

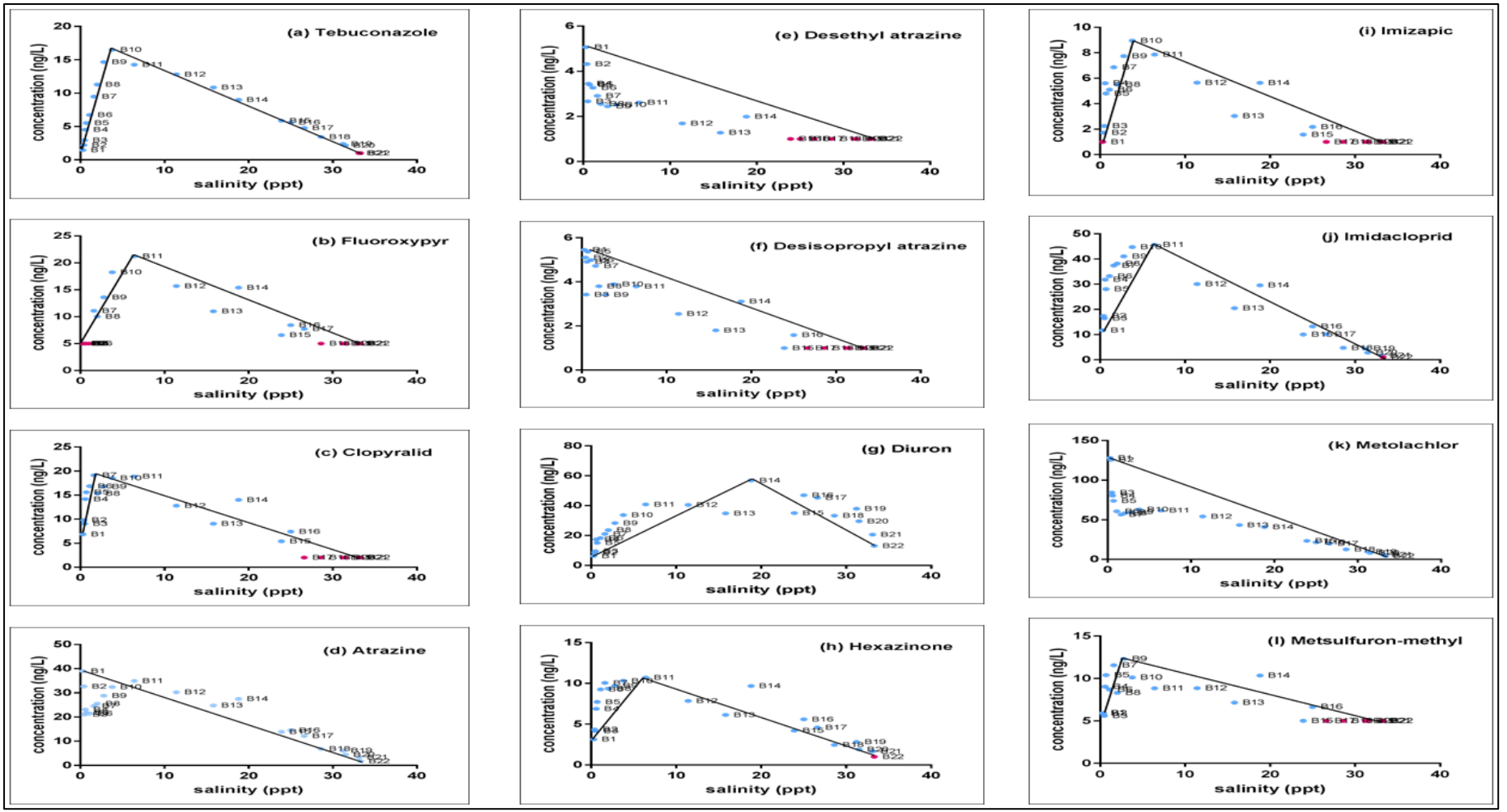


Fig. 6.3: A mixing plot illustrating the fate of respective CUPs along the Brisbane River estuary; numbered as sub-figures (a)-(u). The labels B1 to B22 on the plot represent sampling points BR-1 to BR-22 respectively. The lines were drawn using the end-members at lowest salinity (0.3 PSU) and highest salinity (33.3 PSU) and the maximum concentration of the CUPs. Plotted points in red indicate the respective limits of quantitation (LOQ) since the concentrations in the respective samples were < LOQ.

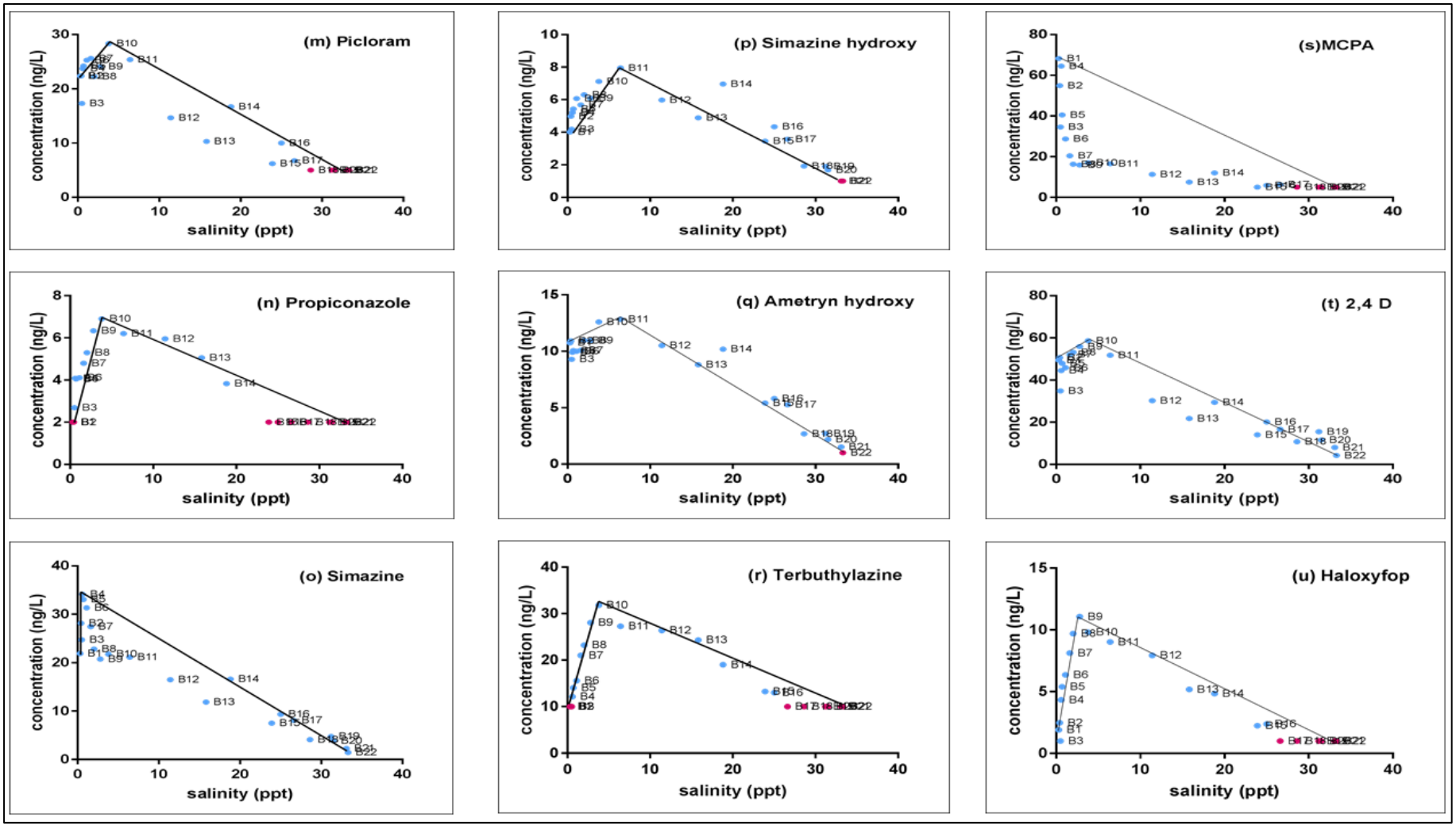


Fig. 6.3 continued over. Sub-figures (a) to (u) represent the plot for individual analytes.

6.3 Summary

The study shows the presence of a range of PPCPs and CUPs in the Brisbane River estuary. Pharmaceutical products, which are primary generated from domestic and hospital waste streams were at measurable levels in the water samples; this suggests secondary inputs of these contaminants from WWTPs. CUPs measured in the water samples are potentially influenced by land-use via direct surface wash-offs from agricultural and parklands or stormwater drains. Salinity of the River is a useful marker property in investigating the fate of contaminants. Conservative transport was observed for three CUPs (tebuconazole, simazine and 2, 4 D) and three pharmaceuticals (carbamazepine, temazepam and paraxanthine). Thus these contaminants can be used as markers to investigate the impact of pharmaceuticals and CUPs on aquatic organisms since they are not readily degrading and thus can maintain a steady dilution ratio when released into water bodies such as river estuaries.

Chapter 7: Distribution and transport of PFASs in core soils impacted by aqueous film-forming foams

The application of AFFF at FTGs is a primary source of PFASs contamination to aquatic environments and subsequent effects on ecological life. Nonetheless, most FTGs in Australia have not been investigated for PFASs contamination. Worst still, the few studies that have been reported have not accounted for the migration mechanisms of PFASs along soil depths at the FTGs. This chapter therefore presents in-depth investigation on the fate of perfluoroalkyl substances in soils impacted by aqueous film-forming foams along a depth profile. Residual PFASs load at the FTG under study have also been estimated and provides a significant input for remediation. The concerns that motivated this study and the objectives are presented in the background information (Section 7.1). Section 7.2 discusses the results in detail; transport of the PFASs in the core soils along a 2 m depth at 0.5 m intervals and the soil physico-chemical factors that affects migration of PFASs at the site, and also estimated transport time of PFASs from the top to 2 m depth is provided. Finally, Section 7.3 summarises the findings of this study, providing inputs for monitoring similar impacted sites in Australia and elsewhere in the world.

7.1 Background

The distribution and mobility of per- and polyfluoroalkyl substances (PFASs) in the environment have attracted attention due to their persistence and potential for bioaccumulation and toxicity [98, 186, 262, 263]. PFASs are characterised by strong covalent C-F bonds. The C-F bonds are formed by either the replacement of all hydrogen atoms attached to the carbon-carbon chain apart from the functional group carbon with fluorine atoms (perfluoroalkyl substances) or at least having one

hydrogen atom remaining on the C-C bond (polyfluoroalkyl substances) [94, 264]. This strong, thermally and chemically stable C-F bond explains the persistence of PFASs. The unique electronegative property of fluorine confers both lipophilic (aliphatic chain end) and hydrophilic (functional group end) properties on PFASs and makes them useful chemicals as surfactants for firefighting aside from other useful applications (including textile treatment, pesticides, paints and food packaging materials) [95, 265]. Consequently, these compounds were used to develop firefighting foams, known as aqueous film-forming foams (AFFF) in the 1960's. The fluorinated surfactants in AFFF create a film that spreads across the liquid fuel surface as the foam bubbles break down, the lipophilic end in the fuel and hydrophilic end in the air/water above it, keeping the water in contact with and preventing oxygen access to the hot fuel [266]. As a result of their effective performance, AFFFs are commonly used to extinguish fire in industries such as aviation, petrochemical, asphalt, bulk fuel and chemical storage facilities. The AFFF formulations contain both pure perfluoroalkyl substances which can be released directly into the environment as a result of AFFF usage, or polyfluoralkyl moieties that can transform under both biotic and abiotic conditions to form the more persistent perfluoroalkyl acids (PFAAs), notably, perfluoroalkyl carboxylic acids (PFCA) and perfluoroalkyl sulphonic acids (PFSA) [9, 100-102, 267]. PFASs contamination of soils can therefore result from the use of AFFF, particularly at locations such as fire training grounds (FTG) where the use of AFFF has been common [21, 267-270].

Two major AFFF products: 3M Lightwater produced by 3M company and Ansulite produced by Tyco have been widely used for firefighting at the location investigated in the present study. These AFFF formulations ranged in PFASs content; from perfluorooctane sulphonate (PFOS) as an active ingredient in 3M Lightwater and including perfluorooctanoic acid (PFOA) and perfluorohexane sulphonate (PFHxS) as minor components, to fluorotelomers in Ansulite [265, 271]. Fluorotelomer precursors such as 6:2 or 8:2 fluorotelomer alcohols [$F(CF_2)_nCH_2CH_2OH$; n = 6 or 8], 6:2 fluorotelomer thioether amido sulphonate (FtTAoS) and 8:2 fluorotelomer sulfonate (8:2 Fts) are polyfluoroalkyl substances that can break down upon their release into the environment during firefighting or emergency response to form

PFCAs and PFSAs, including perfluorohexanoic acid (PFHxA), perfluoropentanoic acid (PFPeA) and perfluorobutanoic acid (PFBA) [100-102]. For example, fluorotelomer betaines (FtB) a proprietary component of Ansulite [272] and including fluorotelomer sulphonamide betaines and 6:2 fluorotelomer sulfonate (6:2-FTSA) were reported in AFFF impacted sediment and fish [273]. Despite 3M company's voluntary decision to cease production of formulations containing PFOS, and the advent of national and international conventions [48, 274, 275] that restrict their production, fluorinated AFFF compounds are still commercially available products. Furthermore, the use of AFFF products purchased prior to production restrictions is often still allowed, with very limited controls. Therefore AFFF contamination of soils, surface/ groundwater as well as aquatic fauna potentially continues [2, 37, 265, 276-278].

The distribution of these fluorinated compounds, however, depends on their physicochemical properties as well as the characteristics of the environmental matrix. Although AFFF can contain a mixture of PFASs in many states (zwitterionic, anionic or cationic), they are often found in media such as soils in their anionic states [279-282]. Therefore, distribution coefficients such as the soil-water partitioning coefficient (K_d) plays a key role in determining the fate and transport of PFASs in soils. While soil-water partitioning is influenced by soil physicochemical properties [147, 268, 283], salinity, pH, cation/anion exchange ratio, temperature, mineralogy and organic carbon content are the key factors [142, 145, 186, 283-285] that can influence PFASs transport.

Although few studies in Australia have reported PFASs contamination in soils, water, sediment and fish [26, 27, 40, 46, 286], investigations of soil contamination at AFFF impacted FTGs in Australia is very limiting [21, 44], with many FTGs remaining uncharacterised. Furthermore, investigations of the migration of PFASs in soils along depth potential at AFFF impacted sites in Australia have not been cited in the literature. This study therefore investigated PFASs contamination arising from the past use of AFFF at the site to understand the factors influencing their transport through environmental matrices. The primary aim of the study was to assess the mobility of these compounds in the soil and to identify factors that influence their

distribution. The study findings are expected to contribute to the development of effective intervention/remediation strategies to contain these environmentally persistent and hazardous compounds at PFASs impacted sites.

7.2 Results and Discussion

7.2.1 PFASs concentrations in the soils

A descriptive statistics of the concentrations for the suite of PFASs analysed in the samples is shown in Table 7.1. While the PFSAs (PFOS, PFHxS and PFBS) present in the soils could emanate as primary compounds and/ or breakdown of polyfluoralkyl precursors in 3M Lightwater [100], the PFCAs measured (PFDA, PFNA, PFOA, PFHpA and PFHxA) are likely breakdown products of polyfluoroalkyl precursors in Ansulite [287]. It is worth noting that PFOA in the soils can also arise as a primary compound from the usage of 3M Lightwater. The spatial distribution of all PFASs analysed in the soils at each coring point is shown in Appendix D: Fig. D-7.1.

The PFASs were dominated by PFOS, accounting for 49-98% of the $\sum_8$PFASs in all the soils analysed. This was followed by PFHxS (0.2-30%) and PFOA (0.5-14%). Apart from the 0-0.5 m depth where PFOS was highest at C14 and followed by C6, the concentration of PFOS was consistently highest at C3 across the remaining depths (0.5-1 m, 1-1.5 m and 1.5-2 m). The lowest concentrations for PFOS across all depths were measured at C17, the coring point furthest removed from the FTG. Consistently, the highest concentrations of PFOS (and other PFASs) were observed in samples closest to the concrete pad of the FTG (inner cores): C1, C2, C3, C6, C7, and C8 (Fig.3.2 & Fig. D-7.1). These coring points, closer to the concrete pad (hosting the mock-up plane), are related to the spots where the firefighting trucks are parked during training. The highest PFOS concentration (2170 ng/g dry weight) representing 98% of $\sum_8$PFASs for coring point C3 was measured at 0.5-1 m depth close to the FTG. Interestingly, the highest concentration of PFOA, 86 ng/g dry weight, was also measured at 0.5-1 m depth, but in C1. The concentration of PFHxS was highest (122 ng/g dry wt.) in C2 at 1.5-2 m coring depth. Fig. D-1 also shows

that concentrations of PFOS at the coring points were always the highest followed by PFHxS and then PFOA (both at 0.5-1 m depth), at the coring points C11 and C14 (both at 0-0.5 m depth) where PFDA was higher than both PFHxS and PFOA.

Table 7.1: Summary of concentrations of PFASs in the soils

Linear PFASs		Concentration in ng/g dry weight			
	LOQ	Min	Max	Mean	Geomean
PFHxA	0.08	<0.08	17.3	3.11	1.23
PFHpA	0.10	<0.10	19.7	1.99	0.67
PFOA	0.05	0.1	85.8	7.42	2.03
PFNA	0.09	<0.09	14.8	1.86	0.63
PFDA	0.06	<0.06	70.5	2.69	0.34
PFBS	0.08	<0.08	3.80	0.55	0.23
PFHxS	0.07	0.1	122	14.3	2.96
PFOS	0.05	1.0	2170	233	93.5
$\sum_8$PFASs	-	1.96	2216	265	110

Nonetheless, some sampling points farther away from the concrete pad, particularly C4, C11, C12 and C14, also showed high concentrations of PFOS which could be due to the reception of AFFF overthrows or spillages during training. It is worth-noting, that the pad can overflow during high rainfall events such as the Brisbane floods in 2011 and 2013 [28] and the south-east of the pad is on the downhill side of the FTG where runoff would go towards the marine environment. Generally, the highest concentrations are to the east and south of the FTG (Fig. 3.2 & Fig. D-7.1).

The AFFF formulations (3M Lightwater and Ansulite) used for firefighting training at this FTG contain pure PFASs as well as precursors which can undergo conversion to form persistent PFAAs [287]. Thus, PFASs observed in this AFFF impacted soil consist of pure ingredients (eg. PFOS and PFOA), unintentional by products formed during production processes (eg. PFBS, PFHxS), and breakdown products (eg.

PFHxA, PFHpA) from the past application of 3M Lightwater and Ansulite. The lower concentrations of these PFASs at the top (0-0.5 m depth) relative to the lower depths (Fig. D-7.1) suggests that the PFASs are migrating downwards at the FTG. Nonetheless, lateral transport of PFASs could also explain some of the inconsistent distribution patterns between the soil cores. It is also worth noting that chain length can play a role in the migration of PFASs as it can affect sorption [288]. For example, the proportion of PFBS at 2 m depth is higher than at 0.5 m depth, unlike the observed result for PFOS (Fig. D-7.1).

7.2.2 Transport of PFASs along the depth profile

The distribution of PFOS along depth profiles at each coring point indicates a vertical transport from the top of the FTG to the lower depths (Fig. 7.1). The mean concentration of PFOS along the vertical transects varied in the order: 312 ng/g dry weight (0.5-1 m) > 299 ng/g dry weight (1-1.5 m) > 167 ng/g dry weight (S1.5-2 m) > 70 ng/g dry weight (0-0.5 m). Similar to the PFOS distribution along the depths, concentrations of PFHxS, PFOA and PFHxA were always lowest at the upper level (0-0.5 m depth) as shown in Fig. 7.1. The distribution of PFDA and PFNA were however higher at the top levels and is due to the relatively higher concentrations of these analytes in the top level at C11 and C14.

The variations in the depth profile of PFASs concentrations including PFOS between cores may be due to differences in the retention of the PFASs as well as differences in the history of AFFF application on the surface. The water table at this site never rises above 2 m, therefore only minor horizontal transport of PFASs in the first 2 m of soil would be expected. Highest levels of PFOS and most other PFASs were typically found in lower parts of the core, specifically in the 0.5 -1 and 1-1.5 m sections (Fig. 7.1). This suggests that a substantial fraction of the PFASs has moved vertically.

To assess potential movement of PFASs from the site we identified historic data on PFAAs spanning similar spatial locations within the site [45, 289] and found that concentrations of PFOS were statistically significantly higher in the first sampling

campaign in 2008 (Fig. 7.2 & Fig. D-7.2), whereas subsequent sampling periods resulted in comparable data.

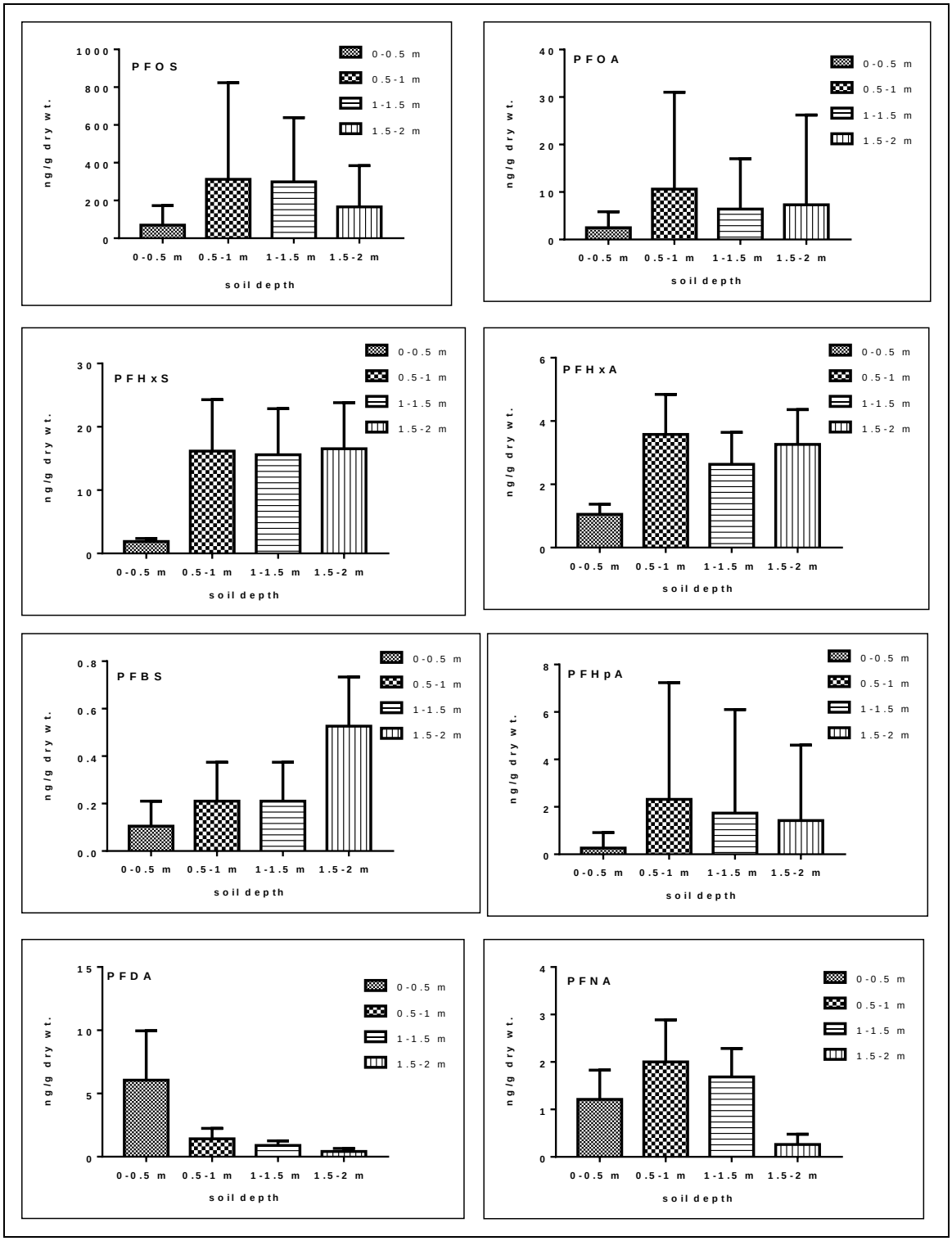


Fig. 7.1: Distribution of mean concentration of analyte observed per depth of soil cores

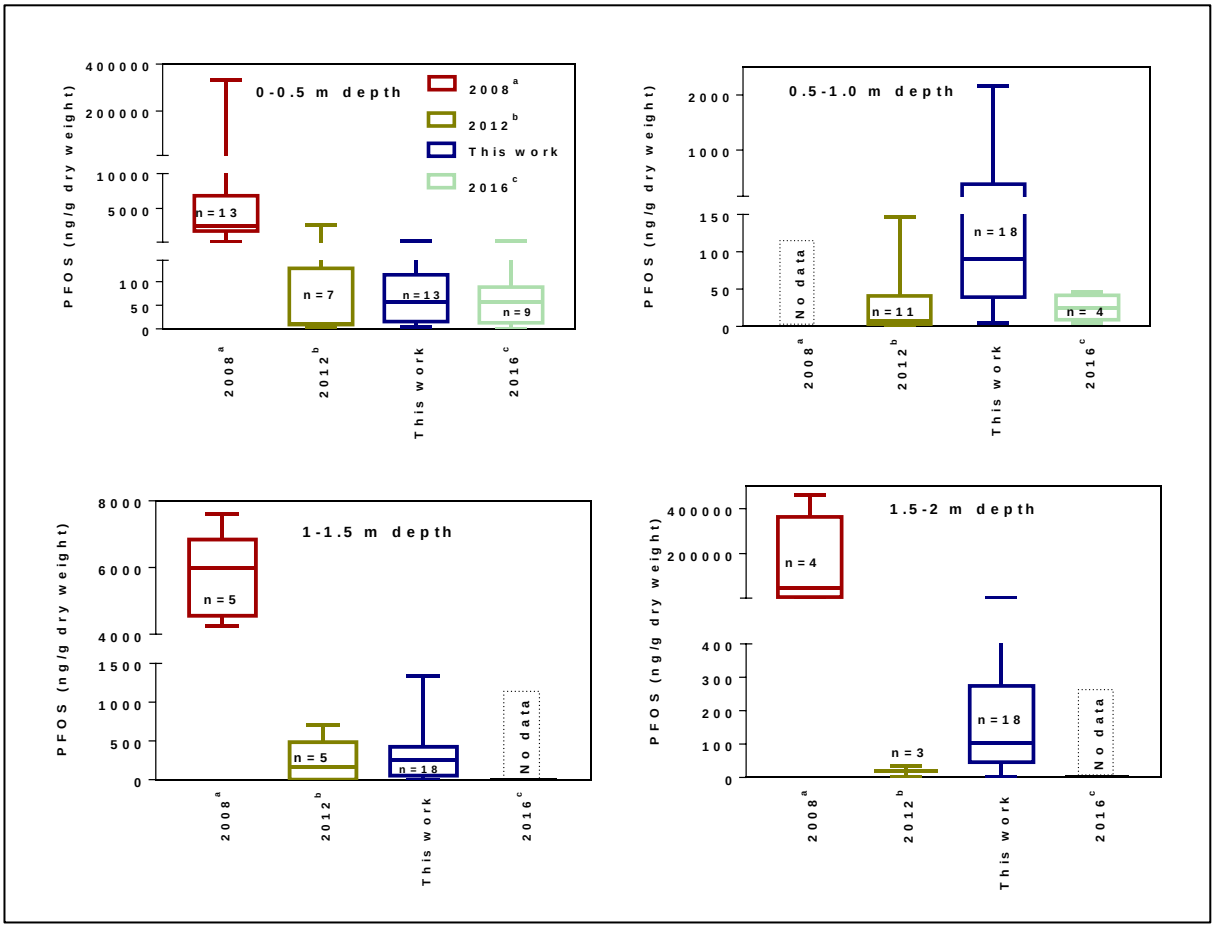


Fig. 7.2: Comparison of PFOS levels at different soil depth profiles in this work with previous studies at the FTG. [a][45], [b][289], [c][290].

Fig. 7.2 shows that the concentrations in soil have decreased dramatically as PFOS must have been transported out of the top 2 m of soils. Apart from the potential vertical transport into depths >2 m, the 2011/2013 flood events in Brisbane could also have impacted the substantial reduction in concentrations between 2008 and the subsequent years due to lateral transport of PFASs into a wider environment, particularly at the top (0-0.5 m). Factors that could influence the vertical distribution of PFASs will be discussed in the following paragraphs.

7.2.3 Estimation of PFOS mass load

Knowledge of mass load and any trends along the depth profile are important determinants in selecting or developing effective remediation/containment strategies, if necessary. The mass load of PFOS in the soils was estimated to determine how much PFOS is present in the bulk soil (0-2 m depth at the FTG) to date. The selection of PFOS, a primary component of 3M Lightwater is based on its higher concentrations in the soils due to persistence and slower migration rate as well as its potential to be the most toxic r PFASs analysed. Notwithstanding, the estimated mass load of PFOS can be related proportionally to other PFASs since the calculations were mostly influenced by soil PFASs concentrations (equation 3.3) since all other parameters will remain same. Based on Equations 3.3 and 3.4., the parameters used for the PFOS mass load estimations are presented in Appendix D (Fig. D-7.3 and Table D-7.1). The results show that the sum of PFOS loading in the bulk soils were highest at S2 (0.5-1 m depth), accounting for 38% of the total mass load (Table D-7.1). Soils at the top, S1 (0-0.5 m depth), had the lowest PFOS mass load of 12% contribution to the total mass load. PFOS mass load at the lower depth (1.5-2 m) is approximately twice the load at the top (0-0.5 m depth). The total mass load of PFOS in the 21 x 10^3 m^3 bulk soil from the FTG was estimated as ~6.5 kg.

It is possible that the vertical distribution of PFASs may be influenced by variability in soil physicochemical properties along the depths of the different coring sites. Therefore, a comparative assessment of the differences in the soil properties and sorption of PFASs to soil in specific soil core samples was undertaken. In addition, the potential migration of PFASs into lower depths and or/ groundwater was also estimated, as this could be indicative of vertical transport.

7.2.4. Measured soil properties and their association with soil-water concentration ratios of PFASs

To assess the sorption of the different PFASs in the specific soils, soil-water concentration ratios (C_s/C_w) were determined for PFASs in the four sections (0-2 m depth) from 10 cores (39 samples) which served as an approximation of the soil-

water partitioning coefficient (K_d) for the respective substance of interest. That is, the K_d measures the amount of perfluoroalkyl substance adsorbed to soil with respect to the amount of perfluoroalkyl substance in water.

The relationships between the estimated K_d values and soil physicochemical properties were then investigated to assess the possible factors that may influence the distribution of PFASs. Results of the K_d estimation, TOC, pH and salinity are presented in Table D-7.2. K_d values were found to be independent of soil PFASs concentrations. The absence of a significant correlation between K_d and concentration is highlighted for PFOS in Fig. D-7.4. Overall, there was no consistent apparent trend in K_d either within a layer or vertically. Summary of the K_d values is provided in Table 7.2. Soil-water concentration ratios for PFOS were the highest, ranging between 1.3 to 30 L/kg. K_d values for PFOA (0.2-6.4 L/kg) and PFHxS (NA-6.1 L/kg) were similar but lower than that for PFOS. The higher K_d value observed for PFOS indicates a slower transport rate from the surface of the FTG to lower depths relative to the other PFASs.

Table 7.2: Statistical summary of soil-water concentration ratios (K_d)

PFASs	Mean (L/Kg) n=40	Std dev.	Min-Max (L/Kg)	n^{th} Percentile				
				10%	25%	50%	75%	90%
PFHxA	1.4	1.0	0.1 – 4.9	0.3	0.5	1.3	2.0	2.4
PFHpA	1.4	1.1	0.2 – 5.2	0.3	0.5	1.3	2.1	2.5
PFOA	1.8	1.3	0.2 – 6.4	0.4	0.7	1.7	2.5	3.2
PFNA	4.0	4.8	0.1 – 18	0.6	1.1	2.5	4.7	8.0
PFDA	7.2	4.1	NA-20	1.3	3.6	7.4	10	12
PFBS	0.8	1.0	NA – 4.4	0.1	0.4	0.6	1.4	1.9
PFHxS	1.7	1.4	NA – 6.1	0.3	0.7	1.7	2.3	3.3
PFOS	10.6	7.3	1.3 – 30	3.1	6.2	8.8	14	20

*NA here means K_d could not be calculated as the respective PFASs in the water portion could not be quantified (<LOQ) in a sample.

In terms of soil properties the total organic carbon fraction (TOC in %) was overall very low when compared to many other soils [145] and varied by more than an order of magnitude, ranging from 0.06 – 1.7%. Li et al. (2018) found that organic carbon (OC) in various soils from published data ranged between <1% to ~75%. TOC is one

of the important sorption compartments for PFASs in soils. Interestingly, there was no obvious spatial (horizontal nor vertical) trend in soil TOC composition across the various depths (Appendix D: Table D-7.2). For example, the highest TOC in C1 (1.2%) was in the bottom section (1.5-2 m depth) whereas in C6 the highest TOC (0.7%) was in the top section (0-0.5 m depth) of the core. This probably reflects the heterogeneous character of the sampling site with more than 50% being built on reclaimed land filled with heterogeneous material. Thus, the TOC in the soils do not exhibit the normal trend expected of a natural site due to the anthropogenic impact of reclamation.

The measured K_d values are in good agreement with data from the literature for soils with TOC content <2% [145]. For example, Li et al. (2018) consistently found K_d values for PFOS to be < 20 L/Kg for soils with TOC < 1%. Other studies have reported that sorption of PFASs to soils and sediment can increase significantly with increasing proportion of TOC [142, 170, 284]. A regression of TOC versus K_d, presented in Appendix D (Fig. D-7.5), did not show any apparent relationship, as R^2 values were: 0.02 (PFOS) and 0.002 (PFOA). This could be due to the relatively higher TOC values observed for a few samples. To minimise this variability, the K_d values were normalised by TOC and expressed as K_{oc} [291]. The effect of K_{oc} was then assessed on other soil properties since increasing K_{oc} favours adsorption of PFASs to soils. It could be expected, particularly for soils with low TOC as observed in this work, that other soil compartments play an important role for the sorption of PFASs [145].

The pH was in the range 6.2-8.8 (Appendix D: Table D-7.2), with an average of 7.5. There was no observed correlation between pH and soil-water concentration ratios in this work (PFOS (R^2=0.05) and PFOA (R^2=0.1)). Hence, pH did not contribute to the observed results and the calculated K_d values. When pH increases, the proportion of anionic PFASs increases as the positive charges on the mineral surface reduces [148], which in effect decreases sorption of PFASs to mineral surfaces [145, 148]. A modelling approach by Li et al. (2018) based on Lee et al. (1990) [149] suggested that sorption of PFASs may predominantly be affected at pH <6 (which we do not have in the present study). Thus, our findings are not in disagreement with Li et al.

(2018) where no correlations were observed between pH and K_d values (PFOS (R^2=0.06) and PFOA (R^2=0.07)).

Salinity in the soils ranged between 0.1 to 0.5 PSU (Table D-7.2). Salinity contributes to the increase of electrostatic charges on the mineral's surface which increases the net positive charge available for sorption of PFASs in soils. A relationship between salinity and log K_{oc} was observed (Fig. 7.3), indicating that salinity played a role in the soil-water partitioning of PFASs at the FTG. Since the K_{oc} values reflects sorption and hence mobility, the positive relationship with salinity indicates that increasing salinity will lead to increase adsorption of PFOS and PFOA in the soils [146].

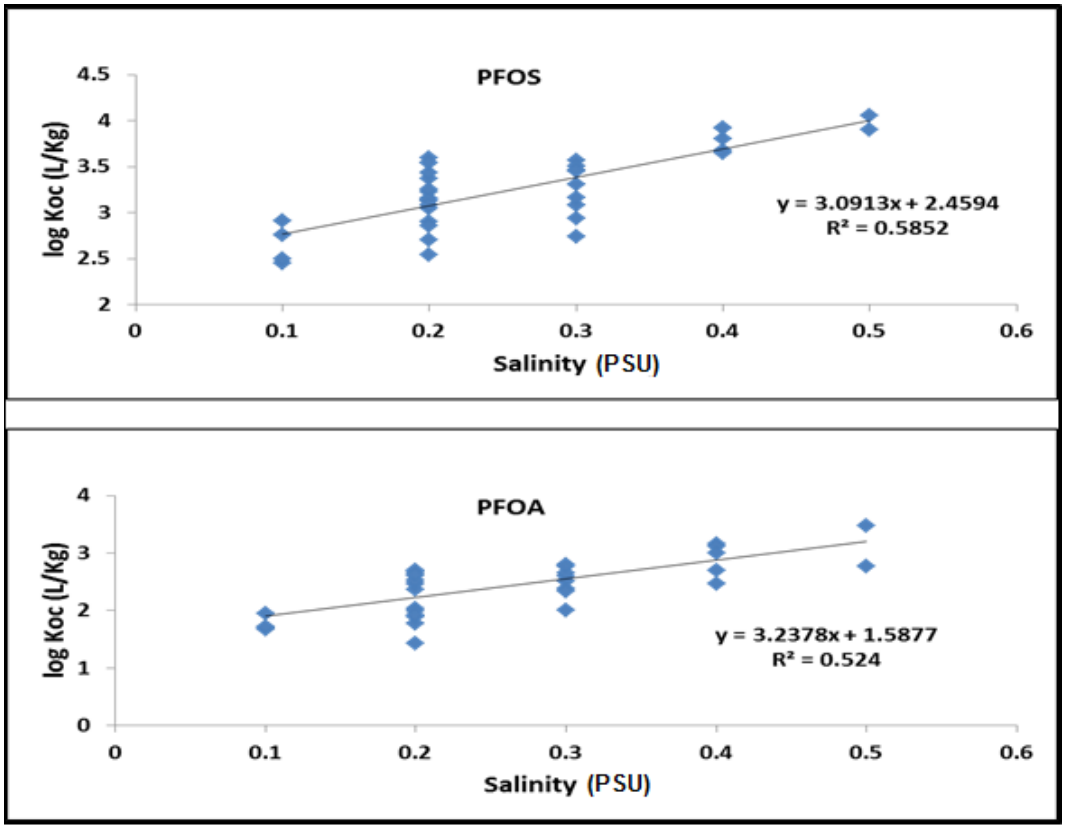


Fig. 7.3: Correlation of salinity with log K_{oc} for PFOS and PFOA in the soils

It is known that PFASs can adsorb to mineral surfaces [138], particularly aluminosilicate rich-clay (kaolinite, illite and montmorillonite). The mineral composition of the soils presented in Appendix D (Fig. D-7.6) shows high levels of quartz with very low clay content. Quartz was found in 100% of the samples with percentage compositions ranging between 38.3 -95.7% (quartz) while clay content

were: 3.4-5.6% (illite) and 1.2-10% (kaolinite) (Appendix D: Table D-7.3). In a similar work at a FTG, Weber et al. (2017) observed high quartz content (>92%) with only up to 0.2% clay composition in soils [267]. The proportions of amorphous mineral (with no definite crystalline structure) were between 2.8 and 23.1%, greater than the clay content (Table D-7.3). This composition of amorphous minerals may further reflect the heterogeneous nature of the FTG which is primarily a reclaimed site.

The general mineralogy composition in this work follows the order: quartz>>plagioclase>amorphous minerals>>clay (kaolinite, illite). There is, however, no observed consistent trend in composition either in depth or space. Notably, irregular compositions were observed for plagioclase, K-feldspar and amorphous minerals along the depths at individual coring sites, particularly C6, C11, C14 and C15 (Fig. C-6.6). Similar to TOC, the low clay content in the soils could imply that a combination of these soil physico-chemical factors could be influencing the soil-water partitioning of PFASs along the various depths of the quartz –rich soils [138, 145, 283].

To investigate the independent factors influencing the partitioning of PFOS and PFOA, the data in Table D-7.2 was mean-centred and subjected to a multilinear regression analysis using SigmaPlot 13.0. Initial results are presented in Appendix D (Table D-7.4). After the elimination of parameters with $p>0.05$, the final regression report suggests that salinity is affecting the partitioning of PFOS and PFOA in the soils. This outcome further supports the observation in Fig. 7.3. Both log K_{oc} (PFOS) and log K_{oc} (PFOA) can be predicted by salinity at $p<0.001$ as shown in Table D-7.4. Soil salinity can increase the electrostatic charges on soil surfaces for PFASs adsorption [146]. This could be further enhanced when there are calcium ions present [147], likely from Ca-bearing minerals such as the calcite and amphibole content measured in the soils. Calcium ions can thus provide a conduit for the adsorption of PFASs onto soils [292, 293]. Thus, salinity could be contributing to the residual PFASs content in the soils, particularly PFOS due to slow desorption processes.

7.2.5. Leaching of PFASs through the surface soil

The use of, and ultimately contamination by, PFASs-containing AFFFs at FTGs around Australia has occurred over several decades from the 1970s and has only ceased in the late 2000s. At the study site, AFFF was used from the late 1980s until 2010. It is estimated that about 150 – 300 x 10^3 L of AFFF containing approximately 5000 – 10000 kg of PFOS were used at this site. Furthermore, during a typical training exercise approximately 90 – 95% of the AFFF used was re-captured with approximately 5 – 10% spilled over the edge into the soil surrounding the FTG. Hence, between 250 – 1000 kg of PFOS entered the soil surrounding the FTG.

In contrast, the top 2 m of soil surrounding the FTG as calculated in this work contains approximately 6.5 kg of PFOS (Table D-7.1), or ~0.5 – 2% of the historical releases to the soil. This suggests that most of the PFASs have been removed from the top 2 m of the soil via leaching since PFOS, for example, is non-volatile.

To explore whether the measured soil-water partitioning of the PFASs is consistent with extensive removal via leaching, the distance of migration of a contaminant spill on the soil surface within 10 years was determined. The choice of 10 years is based on the approximate length of the period between cessation of PFASs use at the site and the soil sampling. The distance of migration of the centre of mass of the contamination d (m) was estimated according to

$$d = \frac{Q_R(1-f_E)t}{\theta R} \qquad \text{Equation 7.1 [294]}$$

where Q_R is the annual precipitation (m yr^{-1}), f_E is the fraction of precipitation lost to evapotranspiration, Θ is the soil porosity, t is time (yr), and R is the retardation factor. R is calculated according to

$$R = 1 + \frac{K_d \rho}{\theta} \qquad \text{Equation 7.2 [138]}$$

where ρ is the soil bulk density (kg L^{-1}). Θ and ρ were measured to be 0.3 and 1.2 kg L^{-1}, while the average precipitation at the site is 1.2 m yr^{-1} [43, 44]. Setting f_E to 0.5, and utilizing the median K_d values from Table 6.2, we estimate d values ranging from 6 m for PFBS to 0.6 m for PFOS in these soils (Table D-7.5). This suggests that most perfluroalkyl substances have moved down the soil and may well have

migrated beyond our sampling depth during the past decade. Thus, the measured soil-water partitioning behaviour supports other observations that suggest a substantial fraction of the PFASs that may have entered the soil following training with AFFF is not accounted for in the area sampled in this study. This therefore supports the hypothesis that much of the PFASs, including PFOS has leached out of the top 2 m of soil into the underlying saturated zone. Residual PFASs in the top profiles could be due to ongoing leakage from the training pad or be a consequence of slow desorption kinetics for a portion of the PFOS associated with the soil.

7.3 Summary

This study shows that even though the first application of AFFF at the study site occurred in 1988, with the last application in 2010, a perfluoroalkyl substance burden can still be measured in soils from the area. The dominant PFAS is PFOS which is due to its higher content in early AFFF formulations (3M Lightwater was last applied ~15 years ago) as well as its relatively high soil-water concentration ratio. The downward plume of PFOS along the depth profile in the core soils indicates depletion of deposits from the top of the FTG into lower depths. The estimated average distances of PFASs migration in the soils from a point of contamination over 10 yrs were 0.6 m (for PFOS) and 2.6 m for both PFOA and PFHxS, suggesting that PFASs released at the site which started >20 yr ago would have migrated well into lower depths at the FTG. Thus, the estimated mass load of PFOS currently in the top 2 m bulk soil at the FTG represents a residual burden which could be due to slow desorption of PFOS or on-going leaching process from the top of the impregnated concrete pad. The effect of physico-chemical parameters on PFASs mobility shows the potential of salinity to contribute to sorption, whereas mineralogy (clay) content and pH did not show any relationship with K_{oc}. Salinity can affect mobility by increasing the electrostatic forces on the surface of the soils to make available Ca^{2+} which could serve as a bridge for the adsorption of PFASs and hence any slow desorption kinetics. This could partly account for the residual PFASs present in the soils.

Although the concentrations of PFOS observed in this work were significantly lower compared to those observed in 2008, the persistence of PFASs and the estimated migration distances indicate a potential short to medium term transport into ground and surface waters. These findings provide inputs for future development of effective containment strategies at this and other FTGs.

Chapter 8: Conclusions and Recommendations for future research

8.1 Conclusions

This research work presented a holistic understanding of the occurrence of PBDEs, PCBs, HBCDDs, PFASs, PPCPs and CUPs and also investigated their fate in both the Brisbane River estuary and a firefighting training ground. Physico-chemical properties, particularly, salinity was found to affect the distribution and transport of the contaminants. The investigations provided baseline data for HBCDDs and PFASs contamination in sediments from the Brisbane River and determined typical PPCPs and CUPs that warrant monitoring in the Brisbane River estuary based on a modified mixing plot model. The residual PFOS in soils at the AFFF impacted FTG was quantified which is a useful input for developing effective site remediation strategies. Spatial distribution of contaminants along the River shows various sources of contamination including WWTPs, agricultural fields, parklands as well as contributions from stormwater drains.

8.1.1 Brominated flame retardants

The interpretation of the results were based on spatial distribution of these contaminants along land-use transects. Generally, concentrations of PBDEs, PCBs, and HBCDDs were found to be relatively higher at the midstream section (urban and commercial activities) of the river compared to downstream sections, where industrial facilities are located. However, the concentrations of PCBs and PBDEs in the sediments were relatively low when compared to other urbanised countries around the world. It is likely that there are no fresh inputs of the contaminants into the River aside from residuals emanating from landfills and effluents from WWTPs. Nonetheless, PBDEs and PCBs could also have settled at lower depths of the sediment beneath the 5 cm depth of surficial sediment that was sampled in this study. Although the use of HBCDDs is still permitted as a flame retardant material, particularly in the building industry its concentration in the sediments were relatively

low when compared to results from regions such as China but comparable to earlier results in sediments from Sydney. This could either be attributed to low usage of HBCDD in Australia or the fact that buildings which have HBCDD-containing flame retardants are still intact. Nonetheless, since HBCDDs contamination in the Brisbane River sediment was not assessed until this study, its sources cannot be immediately deduced. Therefore further investigations of HBCDDs in other environmental matrices including stormwater inlets which flow directly into the Brisbane River is warranted. The results of PBDEs, PCBs, and HBCDDs contamination in the sediments was published thereby enhancing knowledge base on these contaminants worldwide and providing, for the first time, a baseline for HBCDD contamination in the Brisbane River sediment.

8.1.2 PFASs contamination

The results show PFASs contamination in water, sediment and soils from the studied sites. Although the contamination of PFASs in water samples from the Brisbane River was previously assessed, this study provided the first data on PFASs contamination in the Brisbane River sediments. However, in comparison to other estuaries around the world, lower concentrations of PFASs was observed in both the water and sediments, indicating that PFASs contamination in the Brisbane River is due to secondary sources. Nonetheless, there has been a marked increase in PFASs concentration in the water up to a factor of 3 when compared to a previous study that was carried out during the 2011 floods in Brisbane. While the downstream section of the River appears to be less contaminated, probably due to higher rates of dilution due to sea water, sites located upstream of the River showed higher PFASs concentrations. This observation suggests that periodic monitoring of PFASs contamination in the estuary is warranted. Also worth noting is the higher concentrations of PFASs in the water samples relative to the sediments. Potentially, effluents from WWTPs are the major inputs of PFASs aside from possible inputs from agricultural and landfill sites.

The contamination of PFASs in the AFFF impacted soils indicates that FTGs can serve as primary sources of PFASs contamination to the wider environment. This is observed as the inner cores (around the concrete pad) have higher PFASs burden

compared to the coring sites farther away from the concrete pad where the mock-up plane was mounted. The observation of higher PFASs concentrations at the lower depths relative to the top is indicative of PFASs migration into groundwater and the wider environment including surface waters. The study has shown that appreciable amount of PFOS (6.5 kg) is still present at the FTG up to 2 m depth. This estimate is a useful input when designing effective remediation strategies for the site and the method adopted in this study can be replicated at AFFF impacted FTGs in both Australia and other parts of the world to quantify residual PFASs at similar sites. The residual amounts of PFOS at the FTG, for example raises very useful questions for future studies such as: where is the PFASs coming from and are there precursors remaining at the site? Assessment of the impact of soil physico-chemical properties on PFASs transport at the studied site indicate that salinity is the major factor affecting PFASs migration at the FTG. The mobility of PFASs in the soils also indicates that a greater proportion of PFASs deposited at the top of the FTG due to AFFF applications > 20 years ago could have migrated beyond the 2 m depth and that the concentrations measured are residuals. Quantitation of residual PFASs at the FTG is a valuable input into the design of an effective remediation strategy for the site.

8.1.3 PPCPs and CUPs in the Brisbane River estuary

This study provided the first assessment of the fate of PPCPs and CUPs in the Brisbane River along a salinity gradient and further adds to knowledge by developing a modified mixing plot model which can be utilised to assess the fate of other contaminants outside of this study both in the Brisbane River estuary and elsewhere around the world where a steady state condition can be obtained for an estuary, River or dam.

The contamination of the Brisbane River by a suite of PPCPs and CUPs in this study is indicative of varying source inputs. The distribution of PPCPs however shows contamination from WWTP indicating that WWTPs does not provide an effective barrier to the contaminants due to varying solubility properties and therefore removal inefficiencies of the WWTPs. Consequently, it is advisable to contain effluents for further treatment before releasing them into the wider environment since these contaminants can affect the development of aquatic fauna when exposed even at

trace concentrations. Distribution of the CUPS; however, indicated non-point source contributions. The major sources include agricultural fields and parklands along the River. It is also worth noting that some contaminants such as diurion are possibly entering the Brisbane River from the sea. This is possibly due to sea water intrusion up to about 85 km further upstream. Studies elsewhere have highlighted the contamination of diuron at the Great Barrier Reef and therefore a potential source of contamination in the Brisbane River estuary.

This study has also provided evidence that the first application of salinity to investigate the fate of contaminants in the estuary has been very effective as CUPs such as tebuconazole, simazine and 2,4 D and pharmaceuticals, especially carbamazepine, temazepam and paraxnathine were found to be conservative and therefore useful candidates for future monitoring. While it is possible to study the conservative behaviour of contaminants in the laboratory, it is often laborious and susceptible to chemical interferences. Thus salinity as a water physico-chemical property in the estuary presents a ready to use alternative for evaluating the persistence of chemical contaminants.

8.2 Recommendations

8.2.1 PBDEs, PCBs and HBCDDs

Although the concentrations of PBDEs, PCBs and HBCDDs were lower in the sediments compared to other parts of the world, it is possible that historic deposits have been sorbed onto sediments at lower depths in the Brisbane River estuary. Therefore, future studies should include sediment cores in order to assess contamination at depths where benthic organisms may potentially be at risk after feeding on contaminated sediment. Studies on core sediments can also provide a historic data on these contaminants and this can be useful for their global budgeting.

Since chemical contaminants can be resuspended from sediment into water, it is also important to carry out bioavailability studies as this will provide inputs for estimating potential toxicity levels to aquatic organisms in the Brisbane River estuary. In this regard, future studies can deploy solid phase microextraction (SPME) fibres coated

with polydimethylsiloxane (PDMS) as a passive sampler for the measurement of organic contaminants that can readily desorb from sediment into the water. Following deployment of the glass fibres in sediment and achieving equilibration, the fibres can be washed with milli-Q water and analysed directly in the GC-MS/MS by the thermodesorption technique. Results from such studies can be useful in evaluating toxicity indices of the contaminants in the Brisbane River estuary.

Noting that HBCDDs contamination in the sediments have first been reported in this work and the fact that the use of HBCDD-containing materials for building applications is allowed until at least 2024 in Australia, studies HBCDD contamination in various environmental matrices needs to be carried out periodically across Australia. For example, during building demolishing at sites where HBCDD-containing flame retardant materials have been used, the HBCDDs can get into the wider environment via air or particulate matter. These HBCDDs impacted particulate matters can eventually settle on hard surfaces (roads, roofs) and be washed into water systems such as the Brisbane River estuary during precipitation events via stormwater. In order to investigate sources of HBCDDs contamination, future studies should include air quality monitoring of HBCDDs both in indoor and outdoor environments as well as stormwater. Outdoor monitoring should target building demolishing sites as these compounds can disperse into the air and undergo long range atmospheric transport. In addition, leachates should also be continuously monitored as aquatic environment can be impacted.

8.2.2 PFASs

The presence of residual PFASs in the soils at the FTG makes it necessary to carry out further investigations to conclude whether the available perfluoroalkyl substances is due to past breakdown products or the breakdown of precursor compounds is on-going. This will provide a holistic input into the design of the most effective remediation protocols for the site in order to prevent future contamination of the wider environment including surface and groundwater. Although some historic products such as AFFF manufactured by 3M company contain some perfluoroalkyl substances (e.g. PFOS), most perfluoroalkyl acids in the environment are formed

from precursors as a result of breakdown of polyfluoroalkyl substances. For example, precursors including fluorotelomer betaines, fluorotelomer sulphonamide betaines and 8:2 or 6:2 fluorotelomer alcohols that were historically used in AFFF formulations can undergo breakdown in soils to form the more persistent perfluoroalkyl substances. Therefore, future studies should involve analyses of PFASs precursors as this can assist source identification and assist the formulation of effective mitigation strategies.

Also in future works, coring depths at the FTG should go beyond 2 m depth up to 4 m where the water table can be reached. In addition, surface water and groundwater samples from around the FTG should also be investigated. Since PFASs are soluble in water, future works should also include pore water analysis. Pore water extracted from soils in the unsaturated zone can serve as a good medium for the determination of soil-water partitioning coefficient (K_d) as equilibrium in PFASs partitioning will better be achieved compared to the use of milli-Q water in the laboratory batch analysis.

Noting that TOC is very low in the soils and the fact that AFFF formulations contain hydrocarbons, future works should include the assessment of hydrocarbons in the soils and investigate their contribution to PFASs migration along the soil depths.

8.2.3 PPCPs and CUPs

The presence of PPCPs and CUPs in the Brisbane River can lead to the exposure of these contaminants to aquatic fauna thereby changing their physiology and having potential implications on their development, particularly during reproduction. Therefore, future studies should be focused on investigating potential health risk impacts of the contaminants on fauna/ecological life in the Brisbane River estuary. In this regard, future studies should include biological samples such as fishes in the estuary to evaluate the toxicity potential of the contaminants to the organisms, particularly carbamazepine, temazepam, paraxanthine, tebuconazole, simazine and 2,4 D which were observed to be conservative.

This study has also shown that salinity is an effective marker to evaluate the fate of contaminants in the Brisbane River estuary; however, the use of salinity is possible

only if the estuary is at a steady state where thorough mixing of the contaminants can be achieved. Achieving this steady state depends on contaminant input and hydrodynamics of the estuary. Therefore in future studies, the hydrology of the Brisbane River estuary should be monitored closely to optimise steady state conditions based on the inflow and outflow rates of the estuary. The fate of other environmental contaminants in the Brisbane River estuary should utilise the modified mixing plot model designed in this work.

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Appendices

Appendix A

The study area and experimental methods

Table A-3.1: Physico-chemical properties of the Brisbane River water samples

Site ID	pH	Temp (^{o}C)	Conductivity (mS/cm)	Salinity (PSU)
BR-1	7.61	29.9	0.78	0.32
BR-2	7.54	28.9	0.96	0.43
BR-3	7.70	29.8	1.13	0.51
BR-4	7.66	28.7	1.33	0.60
BR-5	7.72	28.7	1.54	0.72
BR-6	7.65	28.5	2.12	1.14
BR-7	7.69	27.9	3.09	1.61
BR-8	7.61	28.2	3.74	2.01
BR-9	7.64	28.1	5.14	2.84
BR-10	7.61	28.2	6.92	3.81
BR-11	7.57	28.1	11.17	6.44
BR-12	7.56	27.9	19.10	11.41
BR-13	7.72	27.7	25.81	15.83
BR-14	7.84	28.5	30.10	18.81
BR-15	7.99	27.2	37.60	24.10
BR-16	8.02	27.3	39.01	25.04
BR-17	8.04	27.6	41.30	26.62
BR-18	8.05	28.7	43.90	28.64
BR-19	8.12	27.4	48.00	31.25
BR-20	8.10	27.6	48.10	31.53
BR-21	8.20	27.7	50.50	33.14
BR-22	8.14	27.9	50.70	33.30

Table A-3.2: Transitions and collision energies for PBDEs and PCBs

Analyte	**Start ion**	**End ion**	**Collision energy (eV)**	**Time window starts (minutes)**	**Time window end (minutes)**
Native Tri BDE	405.8	245.89	15	12.8	14.8
Native Tri BDE ref	407.8	247.89	20	12.8	14.8
C-Tri BDE	417.84	258.04	10	12.8	14.8
C-Tri BDE ref	419.84	260.04	40	12.8	14.8
Native Tetra BDE ref	483.71	323.84	20	14.55	18.05

Native Tetra BDE	485.71	325.84	20	14.55	18.05
C-Tetra PBDE	497.71	337.84	15	14.55	18.05
C-Tetra BDE ref	499.75	339.95	25	14.55	18.05
native Penta BDE	563.65	403.81	25	15.75	19.25
Native Penta BDE ref	565.62	405.8	20	15.75	19.25
C-Penta BDE	575.66	415.86	25	15.75	19.25
C-Penta BDE ref	577.66	417.86	25	15.75	19.25
Native Hexa BDE ref	641.53	481.7	20	17.65	21.15
NativeHexa BDE	643.53	483.73	25	17.65	21.15
C-Hexa BDE	655.57	495.77	25	17.65	21.15
C-Hexa BDE ref	657.57	497.77	15	17.65	21.15
Native Hepta BDE	721.44	561.76	20	21.3	23.3
Native Hepta BDE ref	723.43	563.63	25	21.3	23.3
C-Hepta BDE	733.48	573.68	30	21.3	23.3
C-Hepta BDE ref	735.48	575.68	30	21.3	23.3
Native Tri PCB	255.96	186.02	25	9.52	11.52
Native Tri PCB ref	257.96	186.02	30	9.52	11.52
C-Tri PCB	267.9	198.02	30	9.52	11.52
C-Tri PCB ref	269.9	198.02	30	9.52	11.52
Native Tetra PCB	289.92	219.98	25	10.45	13.95
Native Tetra PCB ref	291.92	219.98	20	10.45	13.95
C-Tetra PCB	301.96	232.02	30	10.45	13.95
C-Tetra PCB ref	303.96	232.02	30	10.45	13.95
Native Penta PCB ref	323.9	253.95	30	11.2	16.2
Native Penta PCB	325.88	255.95	25	11.2	16.2
C-Penta PCB	335.92	265.91	30	11.2	16.2
C-Penta PCB ref	337.92	267.91	35	11.2	16.2
Native Hexa PCB ref	357.84	287.88	25	13.35	16.85
Native Hexa PCBs	359.84	289.87	30	13.35	16.85
C-Hexa PCB	369.84	299.88	30	13.35	16.85
C-Hexa PCB ref	371.84	301.87	35	13.35	16.85
Native Hepta PCB ref	391.81	321.84	25	14.45	17.95
Native Hepta PCB	393.81	323.84	25	14.45	17.95
C-Hepta PCB ref	403.83	333.84	30	14.45	17.95
C-Hepta PCB	405.81	335.84	30	14.45	17.95

Table A-3.3: MRM operation parameters for LC-MS/MS analysis of PFASs.

Analyte	Acronym	Transitions	Q1 Mass (Da)	Q3 Mass (Da)	Time (msec)	DP (Volts)	CE (volts)	CXP (volts)	Retention time (min)
Perfluorobutanoic acid	PFBA	1	213	169	10	-50	-13	-16	1.73
Perfluoropentanoic acid	PFPeA	1	263	219	10	-50	-13	-23	4.27
Perfluorohexanoic acid	PFHxA	2	313	269, 119	10	-60	-13, -29	-27, -10	5.90
Perfluroheptanoic acid	PFHpA	2	363.1	319, 169	10	-60	-15, -26	-12, -10	6.86
Perfluorooctanoic acid	PFOA	3	413.1	369, 169, 219	10	-90	-16, -27, -20	-15, -15, 20	7.55
Perfluorononaoic acid	PFNA	2	463.1	419.1, 169	10	-95	-16, -27	-17, -10	8.11
Perfluorodecanoic acid	PFDA	2	513.1	469.1, 269	10	-100	-17, -26	-18, -10	8.60
Perfluoroundecanoic acid	PFUnDA	2	563.2	519.1, 269	10	-100	-20, -26	-20, -10	9.08
Perfluorododecanoic acid	PFDoDA	2	613.1	569.1, 169	10	-105	-20, -36	-23, -10	9.61
Perfluorobutane sulphonate	PFBS	2	299.1	80, 99	10	-90	-58, -45	-10, -10	4.80
Perfluorohexane sulphonate	PFHxS	2	399.1	80, 99	10	-90	-75, -58	-10, -10	6.93
Perfluorooctane sulphonate	PFOS	6	499.1	80, 99, 130, 419, 219, 169	10	-120	-105, -85, -54, -37, -42, -44	-10, -10, -10, -10, -10, -10	8.11
^{13}C-Labelled Spike Standards									
MPFOA		2	417.1	372, 169	10	-65	-16, -27	-15, -21	7.54
MPFOS		2	503.1	80, 99	10	-100	-100, -75	-10, -10	8.10
MPFBA		1	217	172	10	-50	-13	-16	1.71
MPFHxA		1	315	270	10	-60	-13	-27	5.89
MPFNA		1	468	423	10	-70	-16	-17	8.09
MPFDA		1	515	470	10	-70	-17	-18	8.58
MPFUnDA		1	565	520	10	-80	-18	-20	9.07
MPFDoA		1	615	570	10	-80	-20	-23	9.60
MPFHxS		1	403	103	10	-90	-75	-10	6.92
M8PFOA		2	421.2	172, 223	10	-100	-25, -25	-12, -10	7.53
M8PFOS		2	507.1	80, 99	10	-100	-100, -75	-10, -10	8.10

Table A-3.4: List of target analytes: (A) Pharmaceuticals, (B) Current-use pesticides, and (C) Personal care products and including (D) internal standards, with their respective retention times and ionization modes for the HPLC-MS/MS analysis. The m/z ratios for 1st and 2nd (in brackets) transitions are shown both for the precursor (Q1) and fragment (Q3) ions. *t_r mean analyte was monitored over the entire run, rather than a scheduled window.

(A) Pharmaceuticals	**m/z ratio,Q1 (precursor)**	**m/z ratio, Q3 (fragment)**	**Retention time (min)**	**+ve/-ve ionization mode**
Paraxanthine	181 (181.1)	124 (96)	3.9	+ve
Atenolol	267.2 (267.2)	190 (145)	*t_r	+ve
Atorvastatin	559.5 (559.5)	(440.3 250.2)	6.67	+ve
Carbamazepine	237.2 (237.2)	194 (193)	6	+ve
Citalopram	325.3 (325.3)	109 (262.2)	5.35	+ve
Codeine	300.2 (300.2)	215.1 (165.1)	4	+ve
N-Desmethylcitalopram	311.3 (311.3)	109 (262.2)	5.38	+ve
N-Desmethyldiazepam	271.2 (271.2)	140.1 (165.1)	6.45	+ve
Fluoxetine	310.1 (310.1)	44 (148)	*t_r	+ve
Gabapentin	172.1 (172.1)	154 (137)	3.3	+ve
Iopromide	792 (792)	573.1 (559.1)	4	+ve
Naproxen	123.2 (231.2)	185.1 (170.1)	6.31	+ve
Paracetamol	152.1 (152.1)	110 (65.1)	1.5	+ve
Temazepam	301.2 (301.2)	255.1 (283.1)	6.53	+ve
Tramadol	264.2 (264.2)	58 (42)	4.72	+ve
Venlafaxine	278.2 (278.2)	58 (121)	5.1	+ve
Sildenafil	475.2 (475.2)	58 (283.1)	5.73	+ve
Verapamil	455.2 (455.2)	165.1 (303.2)	5.75	+ve
Hydroxycotinine	193.1 (193.1)	134.1 (80.1)	1.37	+ve
Ibuprofen	205.1 (205.1)	161 (159)	6.41	-ve
Furosemide	329 (329)	285 (205)	5.66	-ve
Caffeine	195.1 (195.1)	138.1 (110.1)	4.63	+ve
Cotinine	177.1 (177.1)	80 (98)	3.5	+ve
Nicotine	163.1 (163.1)	132 (106.1)	1.13	+ve
Hydrochlorthiazide	296 (296)	269 (205)	2.8	-ve

(B) Current-use pesticides	**m/z ratio,Q1 (precursor)**	**m/z ratio, Q3 (fragment)**	**Retention time (min)**	**+ve /-ve ionization mode**
Tebuconazole	308.15 (310.15)	70 (70)	6.55	+ve
Fluroxypyr	255 (255)	209 (181)	5.21	+ve
Pendimethalin	282.1 (282.1)	212.1 (194.1)	7.42	+ve
Fluazifop	328.2 (328.2)	282.2 (254.1)	6.23	+ve
Propazine	230 (230)	146 (188)	6.01	+ve

3,4 DiCl Aniline	162 (162)	127 (74)	5.51	+ve
Ametryn	228.2 (228.2)	186 (116)	*t_r	+ve
Asulam	231 (231)	156 (108)	3.81	+ve
Atrazine	216.1 (216.1)	174 (96)	5.77	+ve
Bromacil	261.2 (263.2)	205 (207)	5.42	+ve
Carbofuran	221.1 (221.1)	165.2 (123)	5.77	+ve
Chlorpyriphos	350.1 (350.1)	198 (97)	7.3	+ve
Clopyralid	192 (192)	110 (146)	2.55	+ve
Desethyl Atrazine	188 (188)	146 (104)	4.68	+ve
Desisopropyl Atrazine	174 (174)	104 (96)	4	+ve
Diazinon	305.3 (305.3)	169.1 (249.1)	6.75	+ve
Dichlorvos	221 (221)	109 (127)	5.4	+ve
Diuron	233.05 (233.05)	72 (46)	5.7	+ve
Fenamiphos	304.15 (304.15)	217.1 (202.1)	6.55	+ve
Flumeturon	233.1 (233.1)	72 (46)	5.45	+ve
Hexazinone	253.2 (253.2)	171 (71)	5.95	+ve
Imazapic	276.1 (276.1)	231.1 (163)	5.19	+ve
Imazethapyr	290.1 (290.1)	177.1 (106)	5.6	+ve
Imidacloprid	256.1 (256.1)	209.1 (175)	5.14	+ve
Malathion	331.1 (331.1)	99 (127)	6.6	+ve
Methomyl	163.1 (163.1)	88.1 (106)	4.26	+ve
Metolachlor	284.2 (284.2)	252 (176)	6.75	+ve
Metribuzin	215.1 (215.1)	187 (47)	5.6	+ve
Metsulfuron-Methyl	382.1 (382.1)	167 (199)	6.19	+ve
Picloram	243 (243)	197 (143)	3.6	+ve
Prometryn	242.2 (242.2)	158 (200.1)	*t_r	+ve
Propiconazole	342 (342)	159 (41)	6.85	+ve
Propoxur	210.1 (210.1)	168.1 (111)	5.58	+ve
Simazine	202.1 (202.1)	132 (124)	5.42	+ve
Tebuthiuron	229.2 (229.2)	172 (116)	5.55	+ve
Terbuthylazine	230.1 (230.1)	174 (104)	6.12	+ve
Terbuthylazine-desethyl	202 (202)	146 (104)	5.42	+ve
Terbutryn	242.2 (242.2)	91.2 (71.1)	*t_r	+ve
Simazine hydroxy	184.1 (184.1)	114 (69)	3.68	+ve
DCPU	205.03 (205.01)	127 (162)	5.33	+ve
DCPMU	219.01 (219.02)	127 (162)	5.33	+ve
Ametryn hydroxy	198.11 (198.11)	156 (86)	4.3	+ve
Metalaxyl	280.2 (280.2)	220.1 (192.1)	6.29	+ve
Pyrimethanil	200.1 (200.1)	107 (183)	6.2	+ve
Mecoprop	213 (215)	141 (143)	6.05	-ve
Dicamba	219 (221)	175 (177)	*t_r	-ve
2,4,5-T	252.9 (254.9)	194.9 (196.9)	6.1	-ve
Bromoxynil	273.8 (275.8)	78.9 (78.9)	5.75	-ve
MCPA	199 (201)	141 (143)	5.86	-ve
2,4 D	219 (221)	161 (163)	5.76	-ve
Triclopyr	254 (256)	196 (198)	6.1	-ve
Haloxyfop	360 (362)	288 (290)	6.62	-ve

Diketonitrile	360.1 (360.1)	251.1 (69.2)	5.39	+ve

(C) Personal care products	**m/z ratio,Q1 (precursor)**	**m/z ratio, Q3 (fragment)**	**Retention time (min)**	**+ve or -ve ionization mode**
DEET	192.1 (192.1)	119 (91)	6.12	+ve
Triclosan	287 (289)	35 (35)	6.82	-ve
Salicylic acid	137 (137)	93 (65)	4.24	-ve

(D) Internal standards	**m/z ratio,Q1 (precursor)**	**m/z ratio, Q3 (fragment)**	**Retention time (min)**	**+ve or -ve ionization mode**
Atenolol-D7	274.1 (274.1)	145.1 (190.1)	*t_r	+ve
2,4-D-Ring13C6	225 (227)	167 (169)	5.75	-ve
Ibuprofen-D3	208.1 (208.1)	164 (161)	6.41	-ve
Caffeine-13C3	198.3 (198.3)	140.1 (112.1)	4.61	+ve
Atrazine-D5	221.1 (221.1)	179 (101)	5.75	+ve
Simazine-D10	212 (212)	137 (134)	5.38	+ve
Atrazine desisopropyl-D5	179.1 (179.1)	137.1 (101.2)	3.9	+ve
Atrazine desethyl-D6				
Hexazinone-D6	259.3 (259.3)	177.2 (77.2)	5.95	+ve
Diuron-D6	240.9 (240.9)	78.2 (52.1)	5.7	+ve
Metolachlor-D6	290.2 (290.2)	258.2 (182.2)	6.71	+ve
Imidacloprid-D4	260.2 (260.2)	179.3 (213.2)	5.1	+ve
Acetaminophen-D4				
Hydrochlorothiazide-13C,D2	298.9 (298.9)	269.9 (205.9)	2.75	-ve
Carbamazepine-D10	247.2 (247.2)	204.1 (202.1)	5.97	+ve
MCPA-D6	205.1 (207.1)	147.1 (149.1)	5.83	-ve
Gabapentin-D10	182.1 (182.1)	164 (147)	3.1	+ve
Temazepam-D5	306.2 (306.2)	260.1 (288.1)	6.52	+ve
Fluoxetine-D6	316.2 (316.2)	44 (154.2)	*t_r	+ve
Venlafaxine-D6	284.2 (284.2)	64 (121)	5.02	+ve
Codeine-D3	303.3 (303.3)	152 (115)	4	+ve
Cotinine-D3	180.1 (180.1)	80 (101)	3.47	+ve
Paracetamol-D4	156.1 (156.1)	114.1 (69.1)	1.5	+ve
DEET-D7	199.2 (199.2)	126.1 (98.2)	6.11	+ve
Hydroxycotinine-D3	196.1 (196.1)	134.1 (80)	1.4	+ve
Nicotine-D4	167.1 (167.1)	136 (121)	1.13	+ve

Appendix B

Contamination of PFASs in the Brisbane River estuary

Table B-5.1: Mean concentrations of PFASs (± 1 standard deviation, n=3) in sediments collected from 22 sites along the Brisbane River estuary

Sites	Mean ± SD (ng/g dry weight) of PFAS in sediment, (range)												
	PFBA (LOQ=0.08)	PFPeA (LOQ=0.08)	PFHxA (LOQ=0.08)	PFHpA (LOQ=0.10)	PFOA (LOQ=0.05)	PFNA (LOQ=0.09)	PFDA (LOQ=0.06)	PFBS (LOQ=0.05)	PFHxS (LOQ=0.07)	PFOS (LOQ=0.05)	PFUnA (LOQ=0.05)	PFDoA (LOQ=0.06)	$\sum_{12}$PFASs
BR-1	<0.08	<0.08	0.13 ± 0.01 (0.11-0.14)	<0.10	0.16 ± 0.01 (0.15-0.17)	<0.09	0.08 ± 0.02 (0.06-1.01)	<0.05	<0.07	0.93 ± 0.07 (0.86-1.01)	<0.05	<0.06	1.3
BR-2	<0.08	<0.08	<0.08	<0.10	0.06 ± 0.01 (0.05-0.07	<0.09	0.10 ± 0.02 (0.09-0.12)	<0.05	0.09 ± 0.01 (0.07-0.10)	1.08 ± 0.60 (1.02-1.20)	0.06 ± 0.01 (0.06-0.07)	0.07 ± 0.03 (0.06-0.09)	1.46
BR-3	<0.08	<0.08	<0.08	<0.10	0.17 ± 0.08 (0.08-0.20)	<0.09	0.15 ± 0.02 (0.13-0.16)	<0.05	0.09 ± 0.02 (0.08-0.11)	1.80 ± 0.60 (1.20-2.45)	0.09 ± 0.02 (0.06-0.12)	0.13 ± 0.02 (0.11-0.16)	2.43
BR-4	<0.08	<0.08	<0.08	<0.10	<0.05	<0.09	<0.06	<0.05	<0.07	<0.05	<0.05	<0.06	<LOQ
BR-5	<0.08	<0.08	<0.08	<0.10	<0.05	<0.09	<0.06	<0.05	<0.07	0.22 ± 0.01 (0.21-0.24)	<0.05	<0.06	0.22
BR-6	<0.08	<0.08	<0.08	<0.10	<0.05	<0.09	<0.06	<0.05	<0.07	0.24 ± 0.02 (0.21-0.26)	<0.05	<0.06	0.24
BR-7	<0.08	<0.08	0.11 ± 0.03 (0.09-0.14)	<0.10	0.23 ± 0.07 (0.17-0.23)	<0.09	0.39 ± 0.05 (0.33-0.43)	<0.05	0.09 ± 0.02 (0.08-0.11)	1.49 ± 0.17 (1.32-1.66)	0.18 ± 0.04 (0.15-0.17)	0.33 ± 0.01 (0.28-0.41)	2.82
BR-8	<0.08	<0.08	<0.08	<0.10	0.09 ± 0.03 (0.06-0.11)	<0.09	0.23 ± 0.02 (0.21-0.25)	<0.05	<0.07	1.51 ± 0.07 (1.44-1.58)	0.19 ± 0.02 (0.17-0.21)	0.26 ± 0.03 (0.23-0.28)	2.28
BR-9	<0.08	<0.08	<0.08	<0.10	0.25 ± 0.07 (0.23— 0.29)	<0.09	0.42 ± 0.06 (0.37-0.48)	<0.05	<0.07	1.84 ± 0.18 (1.66-1.87)	0.21 ± 0.03 (0.19-0.23)	0.31 ± 0.05 (0.27-0.36)	3.03
BR-10	<0.08	<0.08	<0.08	<0.10	0.14 ± 0.02 (0.12-0.17)	<0.09	0.18 ± 0.03 (0.16-0.21)	<0.05	<0.07	1.60 ± 0.24 (1.34-1.81)	0.21 ± 0.03 (0.18-0.23)	0.25 ± 0.06 (0.20-0.31)	2.38
BR-11	0.11 ± 0.03 (0.09-0.14)	<0.08	<0.08	<0.10	0.18 ± 0.03 (0.14-0.21)	<0.09	0.29 ± 0.08 (0.23-0.38)	<0.05	<0.07	2.58 ± 0.77 (1.96-3.44)	0.17 ± 0.03 (0.14-0.20)	0.23 ± 0.07 (0.17-0.31)	3.56
BR-12	<0.08	<0.08	<0.08	<0.10	0.09 ± 0.03 (0.06-0.12)	<0.09	0.19 ± 0.02 (0.16-0.20)	<0.05	<0.07	0.77 ± 0.18 (0.66-0.98)	<0.05	0.14 ± 0.02 (0.12-0.16)	1.19
BR-13	<0.08	<0.08	<0.08	<0.10	0.19 ± 0.16 (0.08-0.38)	<0.09	0.36 ± 0.25 (0.15-0.64)	<0.05	0.10 ± 0.02 (0.09-0.12)	1.19 ± 0.62 (0.67-1.87)	0.14 ± 0.08 (0.07-0.23)	0.24 ± 0.09 (0.18-0.44)	2.22
BR-14	<0.08	<0.08	<0.08	<0.10	0.11 ± 0.02 (0.09-0.13)	<0.09	0.33 ± 0.05 (0.28-0.39)	<0.05	<0.07	0.87 ± 0.11 (0.80-1.01)	0.10 ± 0.02 (0.09-0.13)	0.18 ± 0.05 (0.14-0.24)	1.59
BR-15	<0.08	<0.08	<0.08	<0.10	<0.05	<0.09	0.08 ± 0.01 (0.07-0.08)		<0.07	0.41 ± 0.06 (0.35-0.47)	<0.05	0.09 ± 0.02 (0.07-0.11)	0.58
BR-16	<0.08	<0.08	<0.08	<0.10	<0.05	<0.09	<0.06	<0.05	<0.07	0.22 ± 0.05 (0.18-0.27)	<0.05	<0.06	0.22
BR-17	<0.08	<0.08	<0.08	<0.10	0.13 ± 0.05	<0.09	0.31 ± 0.15	<0.05	<0.07	1.35 ± 0.17	0.15 ± 0.04	0.19 ± 0.03	2.13

					(0.09-0.17)		(0.13-0.41)			(1.16-1.49)	(0.10-0.18)	(0.16-0.22)	
BR-18	0.12 ± 0.03 (0.10-0.15)	<0.08	<0.08	<0.10	0.10 ± 0.02 (0.08-0.13)	<0.09	0.33 ± 0.08 (0.27-0.42)	<0.05	<0.07	0.97 ± 0.06 (0.91-1.02)	0.42 ± 0.20 (0.16-0.97)	0.32 ± 0.06 (0.26-0.38)	2.26
BR-19	<0.08	<0.08	<0.08	<0.10	0.07 ± 0.01 (0.06-0.08)	<0.09	0.09 ± 0.03 (0.07-0.12)	<0.05	<0.07	0.51 ± 0.13 (0.36-0.62)	<0.05	<0.06	0.67
BR-20	<0.08	<0.08	<0.08	<0.10	0.14 ± 0.01 (0.13-0.14)	<0.09	0.24 ± 0.01 (0.22-0.25)	<0.05	<0.07	1.07 ± 0.06 (1.02-1.14)	<0.05	0.13 ± 0.01 (0.12-0.14)	1.58
BR-21	<0.08	<0.08	<0.08	<0.10	0.06 ± 0.01 (0.06-0.07)	<0.09	0.14 ± 0.02 (0.12-0.17)	<0.05	<0.07	0.57 ± 0.04 (0.52-0.60)	0.06 ± 0.01 (0.05-0.07)	0.08 ± 0.01 (0.07-0.08)	0.91
BR-22	<0.08	<0.08	<0.08	<0.10	<0.05	<0.09	<0.06	<0.05	<0.07	0.27 ± 0.03 (0.24-0.30)	<0.05	<0.06	0.27
Max	0.12	-	0.13	-	0.25	-	0.42	-	0.1	2.58	0.42	0.33	3.56
Min	<0.08	-	<0.08	-	<0.05	-	<0.06	-	<0.07	<0.05	<0.05	<0.06	<LOQ

*LOQ means limit of quantitation

APPENDIX C

Occurrence and fate of target pharmaceuticals, personal care products and pesticides in the Brisbane River estuary

Table C-6.1: Raw data of pharmaceuticals and personal care products (PPCPs) and the limits of quantitation (LOQ). All values are reported in ng/L. Results for sampling sites BR-1 to BR-10 are presented in sub-table **(A)** while results for sampling sites BR-11 to BR-22 are presented in sub-table **(B)**

(A) Analyte	LOQ	Pre sample blank	Post sample blank	Trip Blank	Lab Blank	BR-1	BR-2	BR-3	BR-3 Repeat	BR-4	BR-5	BR-6	BR-7	BR-8	BR-9	BR-10	BR-10 repeat
Paraxanthine	2	<2	<2	<2	<2	4.98	6.26	6.50	5.47	7.93	8.34	7.37	7.76	7.88	7.60	8.61	8.05
Atenolol	1	<1	<1	<1	<1	<1	3.36	4.02	3.99	7.70	6.49	5.43	6.05	6.27	6.06	7.01	6.73
Atorvastatin	1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1
Carbamazepine	1	<1	<1	<1	<1	50.96	58.69	49.60	47.46	106.42	78.23	80.08	77.32	72.66	74.03	76.78	70.02
Citalopram	1	<1	<1	<1	<1	<1	1.30	1.27	<1	2.61	1.52	<1	<1	<1	<1	<1	<1
Codeine	2	<2	<2	<2	<2	<2	<2	<2	<2	3.02	<2	<2	<2	<2	<2	<2	<2
N-Desmethylcitalopram	1	<1	<1	<1	<1	2.67	2.36	2.50	2.60	5.00	2.18	1.88	1.73	2.25	1.44	2.11	1.94
N-Desmethyldiazepam	2	<2	<2	<2	<2	<2	<2	<2	<2	2.00	2.00	<2	2.00	1.54	2.00	2.00	2.00
Fluoxetine	1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1
Gabapentin	2	<2	<2	<2	<2	11.86	42.61	45.91	45.52	117.57	95.22	99.78	84.80	68.49	60.88	62.84	59.09
Iopromide	2	<2	<2	<2	<2	53.76	61.98	50.41	53.22	94.30	66.25	39.89	43.28	37.24	23.20	26.62	31.69
Naproxen	1	<1	<1	3.36	<1	<1	<1	<1	<1	7.40	<1	<1	<1	<1	<1	<1	<1
Paracetamol	2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2
Temazepam	1	<1	<1	<1	<1	13.63	18.84	21.03	19.26	37.80	26.72	24.75	25.13	26.07	26.38	27.69	26.69
Tramadol	1	<1	<1	<1	<1	11.55	27.67	37.67	33.32	81.06	47.74	37.84	43.10	47.41	39.19	40.24	39.08
Venlafaxine	1	<1	<1	<1	<1	11.88	25.45	30.82	30.30	86.19	37.65	34.23	31.85	36.54	35.46	38.55	39.50
Sildenafil	1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1
Verapamil	1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1
Hydroxycotinine	10	<10	<10	<10	<10	<10	<10	<10	<10	<10	<10	<10	<10	<10	<10	<10	<10
Ibuprofen	2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2
Furosemide	1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1
Caffeine	1	15.22	7.02	6.24	3.37	15.63	16.14	16.65	21.53	18.88	19.58	22.07	20.06	22.95	24.47	27.84	25.67
Cotinine	1	<1	<1	<1	<1	<1	<1	8.33	<1	<1	<1	<1	<1	9.55	<1	<1	<1
Nicotine	1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	1.19	<1	<1	<1
Hydrochlorthiazide	1	<1	<1	<1	<1	<1	<1	<1	<1	31.72	6.89	4.31	<1	<1	7.88	8.23	10.36

DEET	1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1
Triclosan	1	<1	<1	<1	<1	1.63	<1	<1	<1	2.79	2.04	<1	<1	<1	2.26	<1	<1
Salicylic acid	1	62.09	35.07	31.96	67.24	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1

(B) Analyte	LOQ	Pre sample blank	Post sample blank	Trip Blank	Lab Blank	BR-11	BR-12	BR-13	BR-14	BR-15	BR-16	BR-17	BR-18	B19	BR-20	BR-21	BR-22
Paraxanthine	2	<2	<2	<2	<2	7.57	7.00	7.02	10.47	5.15	6.72	5.46	3.41	3.55	2.55	2.37	2.75
Atenolol	1	<1	<1	<1	<1	6.69	4.15	2.45	3.81	1.27	1.89	1.63	<1	<1	<1	<1	<1
Atorvastatin	1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1
Carbamazepine	1	<1	<1	<1	<1	68.23	46.42	31.44	41.49	18.06	22.21	18.54	10.07	11.58	8.66	6.93	5.31
Citalopram	1	<1	<1	<1	<1	<1	<1	<1	1.33	1.23	1.24	1.95	<1	1.16	<1	1.41	1.69
Codeine	2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2
N-Desmethylcitalopram	1	<1	<1	<1	<1	1.55	1.29	1.81	2.11	1.54	1.46	1.85	1.10	<1	1.00	1.25	1.25
N-Desmethyldiazepam	2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2
Fluoxetine	1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1
Gabapentin	2	<2	<2	<2	<2	51.85	32.18	22.39	35.97	14.29	19.53	15.80	8.81	12.50	9.51	9.07	7.82
Iopromide	2	<2	<2	<2	<2	19.55	13.26	10.44	16.19	8.83	10.48	9.75	4.50	4.47	5.41	6.29	3.89
Naproxen	1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1
Paracetamol	2	<2	<2	<2	<2	<2	<2	<2	4.03	2.63	4.44	4.50	2.45	3.16	2.14	<2	5.98
Temazepam	1	<1	<1	<1	<1	25.68	19.25	13.62	14.93	7.05	8.15	7.62	4.04	4.48	3.39	3.03	2.46
Tramadol	1	<1	<1	<1	<1	30.87	21.10	18.40	26.34	13.35	12.61	12.88	4.54	4.89	3.95	4.34	3.01
Venlafaxine	1	<1	<1	<1	<1	36.46	28.00	19.82	20.01	12.75	11.03	11.70	4.51	6.31	4.75	6.33	4.97
Sildenafil	1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1
Verapamil	1	<1	<1	<1	<1	<1	<1	<1	<LOR	<1	<1	<1	<1	<1	<1	<1	<1
Hydroxycotinine	10	<10	<10	<10	<10	<10	<10	<10	<10	<10	<10	<10	<10	<10	<10	<10	<10
Ibuprofen	2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2
Furosemide	1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1
Caffeine	1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1
Cotinine	1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1

Nicotine	1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1
Hydrochlorthiazide	1	<1	<1	<1	<1	6.16	2.64	1.42	2.14	<1	<1	<1	<1	1.23	1.04	1.33	1.33
DEET	1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1
Triclosan	1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1
Salicylic acid	1	62.09	35.07	31.96	67.24	<1	<1	<1	<1	<1	37.51	<1	<1	<1	76.92	64.97	78.43

Table C-6.2: Raw data of current-use pesticides and the limits of quantitation (LOQ). All values are reported in ng/L.
Results for sampling sites BR-1 to BR-10 are presented in sub-table **(A)** while results for sampling sites BR-11 to BR-22 are presented in sub-table **(B)**

(A) Analyte	LOQ	Pre sample blank	Post sample blank	Trip Blank	Lab Blank	BR-1	BR-2	BR-3	BR-3 repeat	BR-4	BR-5	BR-6	BR-7	BR-8	BR-9	BR-10	BR-10 repeat
Tebuconazole	1	<1	<1	<1	<1	1.46	2.21	3.16	2.72	4.51	5.52	6.74	9.47	11.28	14.65	16.38	16.62
Fluroxypyr	5	<5	<5	<5	<5	<5	<5	<5	<5	<5	<5	<5	11.09	10.03	13.59	15.40	21.13
Pendimethalin	2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2
Fluazifop	1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1
Propazine	1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1
3,4 DiCl Aniline	2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2
Ametryn	1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1
Asulam	1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1
Atrazine	1	<1	<1	<1	<1	38.99	32.64	21.83	20.33	23.09	21.77	21.44	24.53	25.52	28.82	33.66	31.11
Bromacil	2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2
Carbofuran	1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1
Chlorpyriphos	2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2
Clopyralid	2	<2	<2	<2	<2	6.84	9.81	8.72	9.37	14.17	15.60	16.87	19.13	15.35	16.78	17.61	19.48

Desethyl Atrazine	1	<1	<1	<1	<1	5.07	4.32	2.74	2.59	3.45	3.41	3.27	2.91	2.55	2.45	2.77	2.27
Desisopropyl Atrazine	1	<1	<1	<1	<1	5.45	5.10	3.54	3.30	4.91	5.37	4.99	4.72	3.80	3.41	4.15	3.63
Diazinon	10	<10	<10	<10	<10	<10	<10	<10	<10	<10	<10	<10	<10	<10	<10	<10	<10
Dichlorvos	50	<50	<50	<50	<50	<50	<50	<50	<50	<50	<50	<50	<50	<50	<50	<50	<50
Diuron	1	<1	<1	<1	<1	6.28	8.73	9.43	9.65	17.40	15.07	18.29	21.00	23.63	28.35	33.30	34.14
Fenamiphos	1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1
Flumeturon	1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1
Hexazinone	1	<1	<1	<1	<1	3.14	4.35	4.38	3.91	6.88	7.72	9.26	10.06	9.36	9.71	10.82	9.82
Imazapic	1	<1	<1	<1	<1	<1	1.73	2.31	2.18	5.61	4.80	5.10	6.86	5.50	7.74	8.60	9.31
Imazethapyr	5	<5	<5	<5	<5	<5	<5	<5	<5	<5	<5	<5	<5	<5	<5	<5	<5
Imidacloprid	1	<1	<1	<1	<1	11.64	17.35	16.71	16.23	31.82	28.05	33.19	37.41	38.18	41.07	46.12	43.36
Malathion	10	<10	<10	<10	<10	<10	<10	<10	<10	<10	<10	<10	<10	<10	<10	<10	<10
Methomyl	2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2
Metolachlor	1	<1	<1	<1	<1	128.19	125.64	85.21	83.46	80.44	73.96	60.63	56.59	58.06	59.74	64.02	62.22
Metribuzin	2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2
Metsulfuron-Methyl	5	<5	<5	<5	<5	5.91	5.90	7.30	2.96	9.03	10.39	8.69	11.56	8.30	12.29	12.16	8.08
Picloram	5	<5	<5	<5	<5	22.35	22.39	17.04	17.60	23.74	24.21	25.28	25.60	22.26	24.12	25.27	31.40
Prometryn	1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1
Propiconazole	2	<2	<2	<2	<2	<2	2.31	2.86	2.51	4.08	4.05	4.11	4.80	5.30	6.34	6.72	7.08
Propoxur	5	<5	<5	<5	<5	<5	<5	<5	<5	<5	<5	<5	<5	<5	<5	<5	<5
Simazine	1	<1	<1	<1	<1	21.90	28.13	24.40	25.02	34.15	34.06	31.33	27.44	22.78	20.74	21.83	21.72
Tebuthiuron	1	<1	<1	<1	<1	<1	<1	<1	<1	<LOR	<1	<1	<1	<1	<LOR	<1	1.00
Terbuthylazine	10	<10	<10	<10	<10	<10	<10	<10	7.47	12.16	14.06	15.58	21.03	23.21	28.06	31.53	32.11
Terbuthylazine-desethyl	1	<1	<1	<1	<1	<1	<1	<LOR	<1	<1	<1	<1	<LOR	<1	<1	<1	<1
Terbutryn	2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2
Simazine hydroxy	1	<1	<1	<1	<1	4.01	4.98	4.28	4.09	5.21	5.43	6.07	5.68	6.30	6.09	6.62	7.62
DCPU	2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2
DCPMU	1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	1.36

Ametryn hydroxy	1	<1	<1	<1	<1	10.74	10.92	9.22	9.35	9.90	10.05	9.99	10.08	10.96	10.95	11.62	13.58
Metalaxyl	1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	1.17	1.21	1.53
Pyrimethanil	1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1
Mecoprop	1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1
Dicamba	10	<10	<10	<10	<10	<10	<10	<10	<10	<10	<10	<10	<10	<10	<10	<10	<10
2,4,5-T	1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1
Bromoxynil	1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1
MCPA	5	<5	<5	<5	<5	68.2	54.9	33.1	36	64.5	40.5	28.7	20.4	16.3	15.9	17.2	16.8
2,4 D	1	2.63	2.31	2.50	2.54	51.95	53.13	37.96	36.68	46.97	50.42	48.34	54.59	55.65	58.38	62.67	59.59
Triclopyr	50	<50	<50	<50	<50	<50	<50	<50	<50	<50	<50	59.69	139.35	111.40	188.30	213.98	130.43
Haloxyfop	1	<1	<1	<1	<1	1.91	2.48	<1	<1	4.33	5.39	6.36	8.12	9.69	11.07	12.00	7.62
Diketonitrile	1	<1	<1	<1	<1	<LOR	<1	<1	<1	<1	<1	1.28	1.24	1.14	1.27	1.19	<1

(B) Analyte	LOR	Pre sample blank	Post sample blank	Trip Blank	Lab Blank	BR-11	BR-12	BR-13	BR-14	BR-15	BR-16	BR-17	BR-18	BR-19	BR-10	BR-21	BR-22
Tebuconazole	1	<1	<1	<1	<1	14.27	12.81	10.85	9.00	5.86	5.60	4.77	3.44	2.38	2.15	1.36	0.88
Fluroxypyr	5	<5	<5	<5	<5	21.22	15.68	11.00	15.40	6.58	8.44	7.74	<5	<5	<5	<5	<5
Pendimethalin	2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2
Fluazifop	1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1
Propazine	1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1
3,4 DiCl Aniline	2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2
Ametryn	1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1
Asulam	1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1
Atrazine	1	<1	<1	<1	<1	34.88	30.25	24.79	27.47	13.90	14.52	12.19	6.80	6.13	4.42	3.30	1.74
Bromacil	2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2
Carbofuran	1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1
Chlorpyriphos	2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2
Clopyralid	2	<2	<2	<2	<2	18.88	12.80	9.06	14.00	5.40	7.40	<2	<2	<2	<2	<2	<2
Desethyl	1	<1	<1	<1	<1	2.61	1.69	1.28	1.99	<1	1.12	<1	<1	<1	<1	<1	<1

Atrazine																	
Desisopropyl Atrazine	1	<1	<1	<1	<1	3.79	2.55	1.80	3.11	<1	1.59	1.03	<1	<1	<1	<1	<1
Diazinon	10	<10	<10	<10	<10	<10	<10	<10	<10	<10	<10	<10	<10	<10	<10	<10	<10
Dichlorvos	50	<50	<50	<50	<50	<50	<50	<50	<50	<50	<50	<50	<50	<50	<50	<50	<50
Diuron	1	<1	<1	<1	<1	40.81	40.53	34.84	56.84	35.06	46.94	45.27	33.32	37.95	29.55	20.64	13.08
Fenamiphos	1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1
Flumeturon	1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1
Hexazinone	1	<1	<1	<1	<1	10.72	7.84	6.13	9.68	4.21	5.59	4.60	2.46	2.82	1.91	1.67	<1
Imazapic	1	<1	<1	<1	<1	7.86	5.65	3.04	5.64	1.58	2.18	<1	<1	<1	<1	<1	<1
Imazethapyr	5	<5	<5	<5	<5	<5	<5	<5	<5	<5	<5	<5	<5	<5	<5	<5	<5
Imidacloprid	1	<1	<1	<1	<1	45.64	30.06	20.46	29.58	10.04	13.23	10.09	4.74	4.44	2.84	1.68	<1
Malathion	10	<10	<10	<10	<10	<10	<10	<10	<10	<10	<10	<10	<10	<10	<10	<10	<10
Methomyl	2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2
Metolachlor	1	<1	<1	<1	<1	61.67	54.19	43.35	40.77	23.42	21.53	19.82	12.50	9.57	8.20	5.97	4.08
Metribuzin	2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2
Metsulfuron-Methyl	5	<5	<5	<5	<5	8.84	8.86	7.15	10.35	3.37	6.64	4.80	2.70	<5	<5	<5	<5
Picloram	5	<5	<5	<5	<5	25.40	14.67	10.30	16.71	6.20	10.01	6.75	<5	<5	<5	<5	<5
Prometryn	1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1
Propiconazole	2	<2	<2	<2	<2	6.20	5.96	5.06	3.83	2.33	2.27	<2	<2	<2	<2	<2	<2
Propoxur	5	<5	<5	<5	<5	<5	<5	<5	<5	<5	<5	<5	<5	<5	<5	<5	<5
Simazine	1	<1	<1	<1	<1	21.12	16.48	11.87	16.58	7.51	9.34	8.08	4.12	4.76	3.88	2.28	1.46
Tebuthiuron	1	<1	<1	<1	<1	1.05	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1
Terbuthylazine	10	<10	<10	<10	<10	27.27	26.38	24.31	19.01	13.26	13.01	10.38	<10	<10	<10	<10	<10
Terbuthylazine-desethyl	1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1
Terbutryn	2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2
Simazine hydroxy	1	<1	<1	<1	<1	7.95	5.98	4.89	6.96	3.45	4.34	3.57	1.92	1.90	1.68	1.21	<1
DCPU	2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2
DCPMU	1	<1	<1	<1	<1	1.91	2.26	<1	2.52	<1	2.03	1.77	<1	<1	<1	<1	<1

Ametryn hydroxy	1	<1	<1	<1	<1	12.83	10.51	8.80	10.18	5.40	5.81	5.25	2.68	2.70	2.18	1.52	<1
Metalaxyl	1	<1	<1	<1	<1	1.16	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1
Pyrimethanil	1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1
Mecoprop	1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1
Dicamba	10	<10	<10	<10	<10	<10	<10	<10	<10	<10	<10	<10	<10	<10	<10	<10	<10
2,4,5-T	1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1
Bromoxynil	1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1
MCPA	5	<5	<5	<5	<5	16.5	11.3	7.5	12	5.4	5.9	6.3	<5	<5	<5	<5	<5
2,4 D	1	2.63	2.31	2.50	2.54	54.27	32.76	24.26	31.95	16.55	22.62	19.12	13.28	17.98	14.07	10.56	6.74
Triclopyr	50	<50	<50	<50	<50	226.38	151.48	84.46	139.60	<50	<50	<50	<50	<50	<50	<50	<50
Haloxyfop	1	<1	<1	<1	<1	9.02	7.93	5.18	4.84	2.24	2.38	<1	<1	<1	<1	<1	<1
Diketonitrile	1	<1	<1	<1	<1	1.45	1.15	<LOR	1.06	<1	<1	<1	<1	<1	<1	<1	<1

Table C-6.3: List of target compounds that were below limits of quantitation. Sub-tables (A), (B) and (C) present the list for pharmaceuticals, pesticides and personal care products respectively.

	(A) Pharmaceuticals		
analyte	***Use***	***quantitation frequency(%)***	***min-max(ng/L)***
Atorvastatin	Controls cholesterol and decrease risk for heart attack and stroke	-	<1
Fluoxetine	Treats depression	-	<2
Naproxen	Reduces swelling and pain	-	<10
Sildenafil	Treats erectile dysfunction	-	<1
Verapamil	Treats high blood pressure	-	<1
Hydroxycotinine	Main nicotine metabolite in smokers urine	-	<1
Ibuprofen	Anti-inflammatory	-	<10
Furosemide	Reduces extra fluid in the body: liver, heart failure, kidney diseases	-	<2
Caffeine	Stimulant (psycho active)	-	<1
	(B) Pesticide (herbicide, fungicide, insecticide)		
Analyte	***Use***	***Quantitation frequency (%)***	***min-max(ng/L)***
Pendimethalin	Herbicide (grasses and some broadleaf weeds)	-	<2
Fluazifop	Herbicide (annual and perennial grasses)	-	<1
Propazine	Herbicide (pre-planting: carrots, celery, fennel)	-	<1
3,4 Dichloroaniline	Diuron metabolite	-	<2

Ametryn	Herbicide (broadleaf weeds and grasses)	-	<1
Asulam	Herbicide (bracken control in horticulture and agriculture)	-	<1
Bromacil	Herbicide (grasses and broadleaf weeds)	-	<2
Carbofuran	Insecticide(corn, potatoes, soybean fields)	-	<1
Chlorpyriphos	Insecticide (termites, mosquitoes, roundworm)	-	<2
Diazinon	Insecticide (cockroaches, ants in residential buildings)	-	<10
Fenamiphos	Insecticide	-	<1
Flumeturon	Herbicide	-	<1
Imazethapyr	Herbicide (preplanting application)	-	<5
Malathion	Insecticide (mosquito and fruit fly eradication)	-	<10
Methomyl	Insecticide (control ticks, foliage and soil borne insects on food and feed crops)	-	<2
Metribuzin	Herbicide (weed control - cereals, pastures, vegetables)	-	<2
Prometryn	Herbicide	-	<1
Propoxur	Herbicide	-	<5
Tebuthiuron	Herbicide	-	<1
Terbuthylazine-desethyl	Herbicide (degradation product of tertbuthylazine)	-	<1
Terbutryn	Herbicide(broadleaf weeds in wheat, barley)	-	<2
DCPU	Degradation product of diuron	-	<2
Metalaxyl	Fungicide (control phytophthora heart and root rots in pineapples, peaches)	-	<1
Pyrimethanil	Fungicide (applied to seeds)	-	<1

Mecoprop	Herbicide (household weed killers)	-	<1
2,4,5-T	Herbicide (weed control: on home lawns, in rice farms, also destroys marijuana plants)	-	<1
Bromoxynil	Herbicide (control weeds in wheats, barley, oats)	-	<1
Diketonitrile	Herbicide (derivative of Isoxaflutole)	-	<1
	(C) Personal care products (PCP) and food additive		
Analyte	***Use***	***Quantitation frequency %***	***min-max(ng/L)***
DEET	Skin insect repellent	-	<1
Salicylic acid	Skin care agent (acne)	-	<1

APPENDIX D

Distribution and transport of PFASs in core soils impacted by aqueous film-forming foams

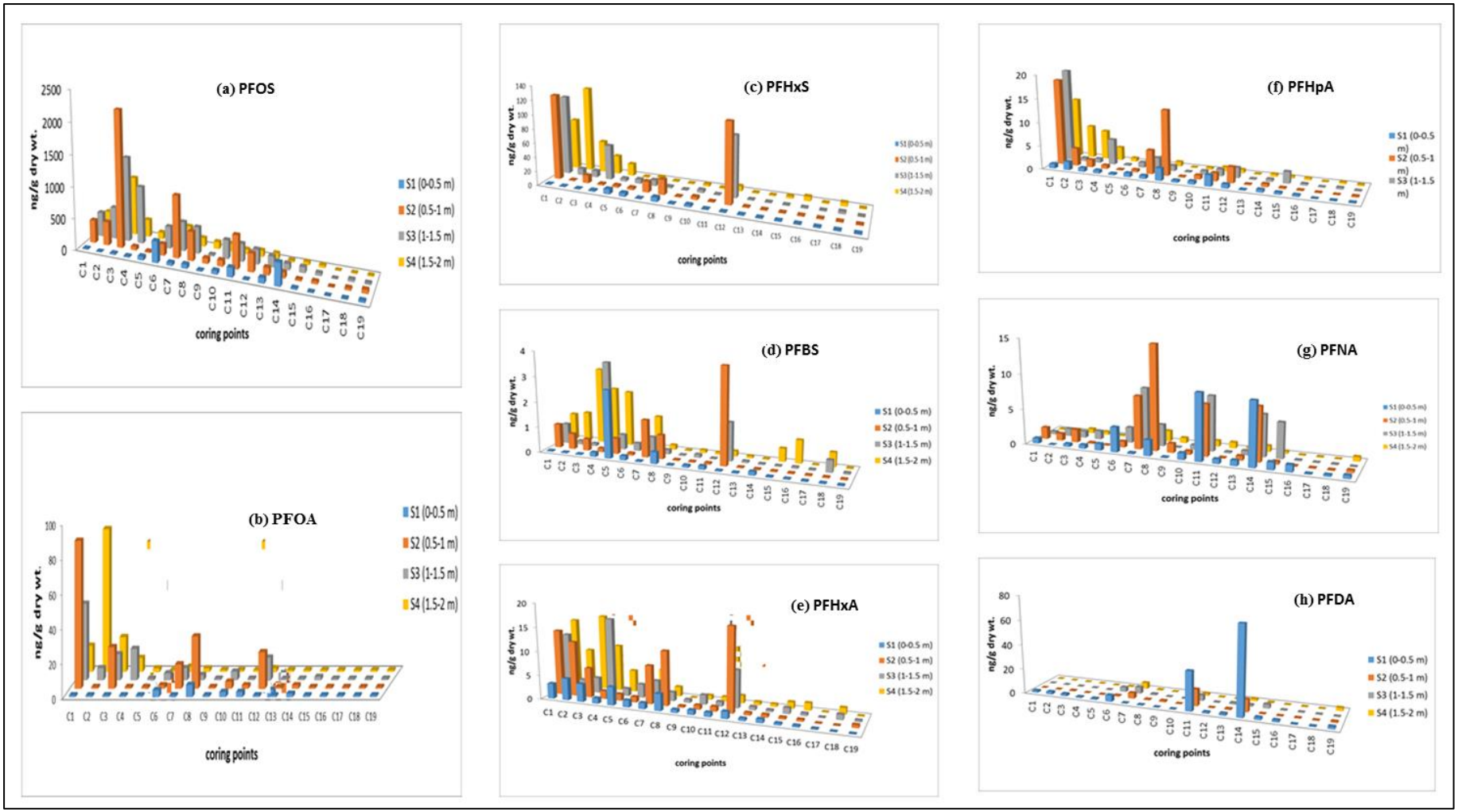

Fig. D-7.1: Spatial distribution of PFASs; (a) PFOS, (b) PFOA, (c) PFHxS, (d) PFBS, (e) PFHxA, (f) PFHpA, (g) PFNA and (h) PFDA in the soils at the respective coring depths.

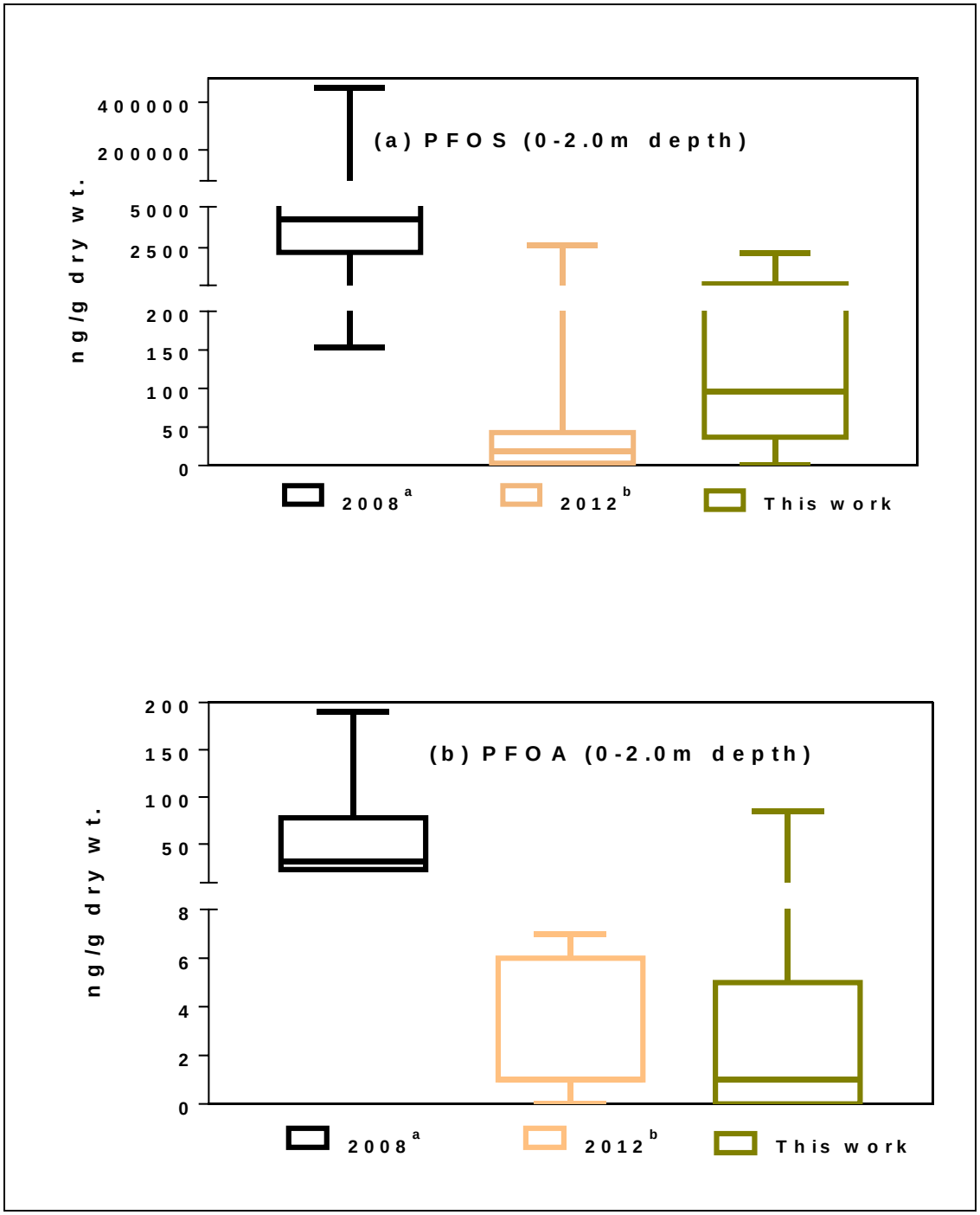

Fig. D-7.2: Comparison of (a) PFOS and (b) PFOA concentrations from respective studies across soil depth of 0-2 m. [a][45], [b][289].

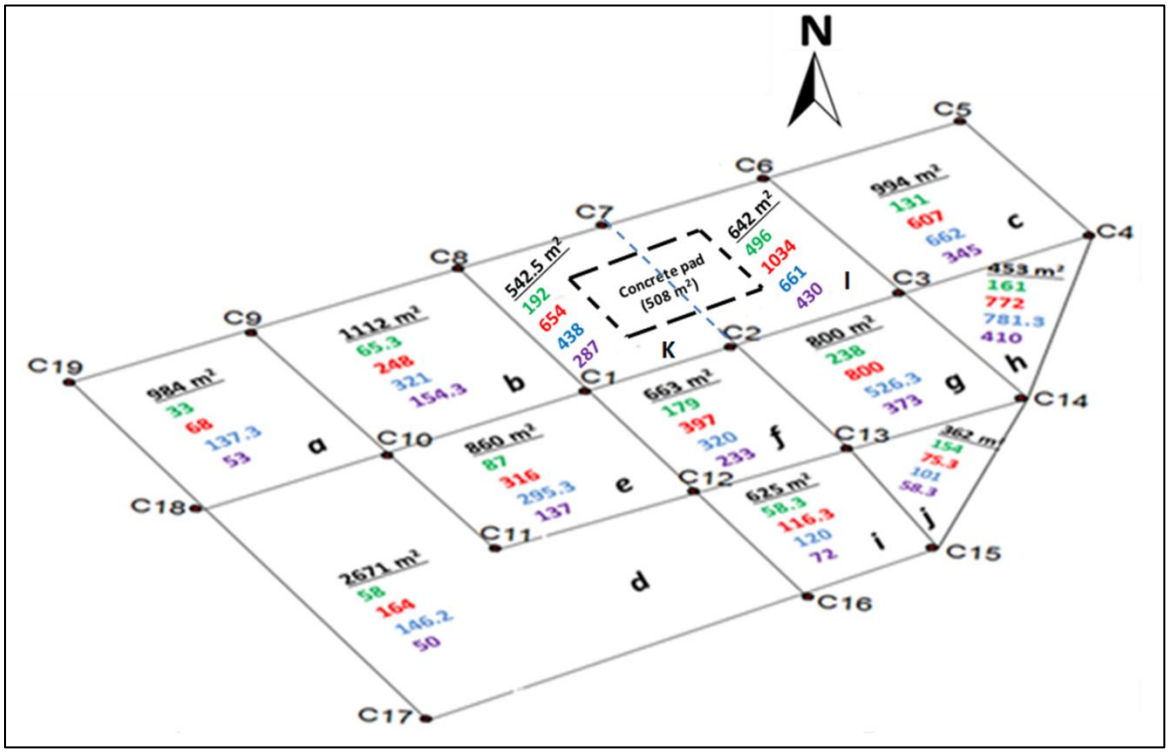

Fig. D-7.3: A diagram showing the sampling grids (***a***-l), bounded area per grid in **black** and mean concentration of PFOS (ng/g dry weight) in coloured codes for the respective sampling depths: **green (0-0.5 m), red (0.5-1 m), blue (1-1.5 m) and purple (1.5-2 m).** Area of the individual core C7 was calculated based on the cylindrical shape of the PVC sampling tube with radius (0.038 m), height (0.5 m).

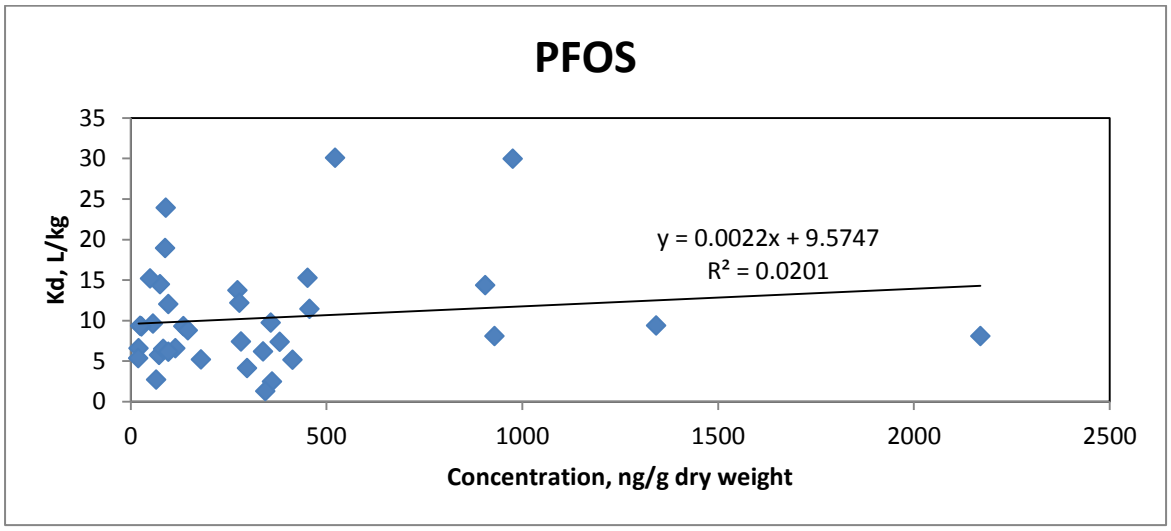

Fig. D-7.4: Scatter plot showing non-correlation between K_d and concentration for PFOS

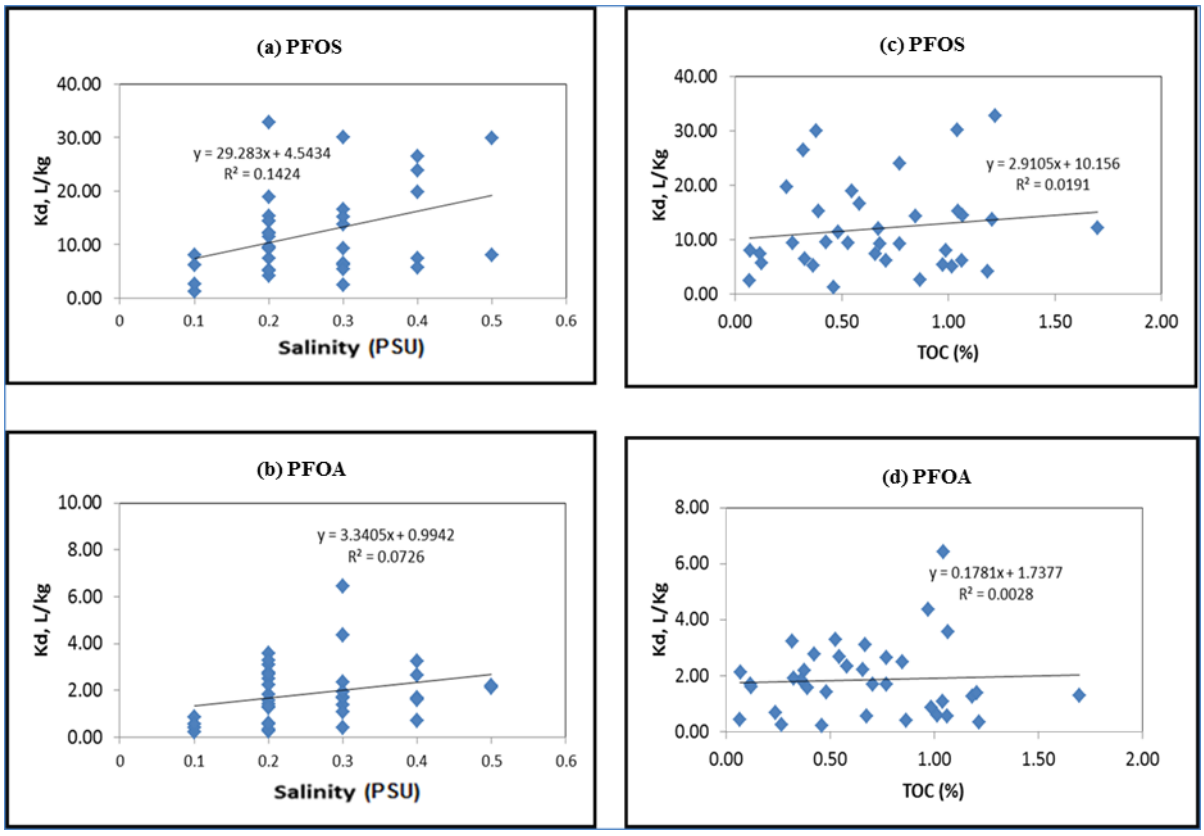

Fig. D-7.5: Correlation plots of: salinity with K_d for PFOS and PFOA presented as (a) and (b) respectively; TOC with K_d for PFOS and PFOA presented as (c) and (d) respectively.

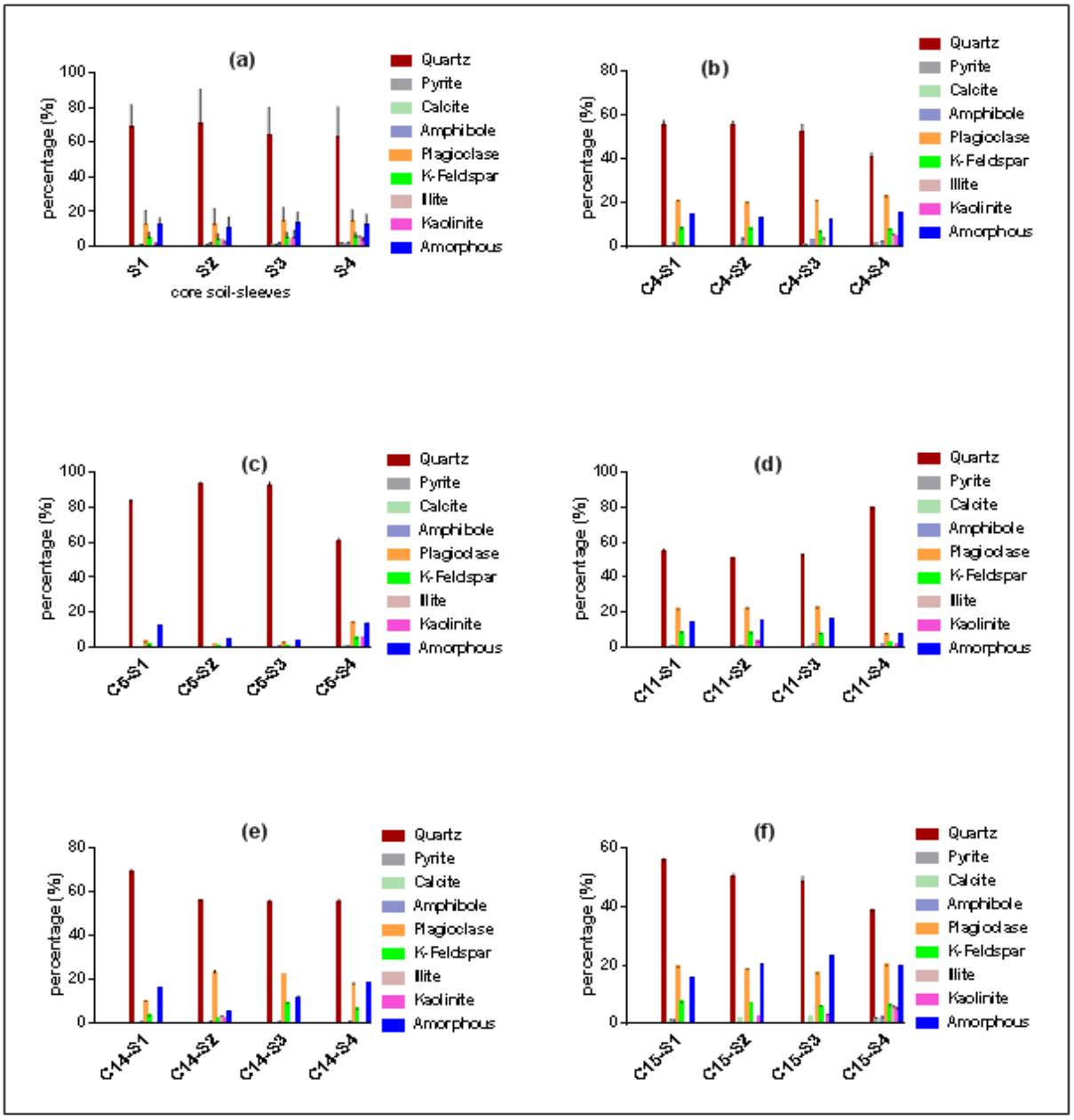

Fig.D-7.6: Distribution of percentage mineralogy composition along the depths (S1-S4) for all core-soils analysed (a) and depth mineralogy profile for some cores; C 4 (b), C6 (c), C11 (d), 14 (e) and C15 (f).

Table D-7.1: Estimated mass loads for PFOS in each of the four sleeves (S1, S2, S3 and S4) at the FTG. Data used for calculating the mass loads in each sleeve (depth) is presented as (A), (B), (C) and (D) for S1, S2, S3 and S4 respectively. The mass load per sleeve excludes the area (508 m^2) of the concrete pad.

(A) S1 (0-0.5 m)						**(B)** S2 (0.5-1 m)					
Grid/soil core	Mean Concentration ng/g dry wt. $x10^{-9}$	Area m^2	Depth (d), m	Soil density, g/m^3 $x10^6$	Mass, g	Grid/soil core	Mean Concentration ng/g dry wt. $x10^{-9}$	Area m^2	Depth (d), m	Soil density, g/m^3 $x10^6$	Mass, g
a	33	984	0.5	1.12	18.18	a	68	984	0.5	1.23	41.15
b	65	1112	0.5	1.15	41.56	b	248	1112	0.5	1.21	166.84
c	131	994	0.5	1.23	80.08	c	607	994	0.5	1.26	380.12
d	58	2671	0.5	1.14	88.30	d	164	2671	0.5	1.2	262.83
e	87	860	0.5	1.13	42.27	e	316	860	0.5	1.21	164.41
f	179	663	0.5	1.16	68.83	f	397	663	0.5	1.2	157.93
g	238	800	0.5	1.17	111.38	g	800	800	0.5	1.15	368.00
h	161	453	0.5	1.18	43.03	h	772	453	0.5	1.22	213.33
i	58	625	0.5	1.20	21.75	i	116	625	0.5	1.25	45.31
j	154	362	0.5	1.20	33.45	j	75	362	0.5	1.21	16.43
k	192	543	0.5	1.20	62.55	K	654	543	0.5	1.20	213.07
l	496	642	0.5	1.20	191.06	l	1034	642	0.5	1.21	401.62
					Ms1 = 802.44						**Ms2= 2431.04**
(C) S3 (1-1.5 m)						**(D)** S4 (1.5-2 m)					
Grid/soil core	Mean Concentratio	Area	Depth (d), m	Soil density,	Mass, g	Grid/soil core	Mean Concentratio	Area	Depth (d),	Soil density,	Mass, g

	n ng/g dry wt. $x10^{-9}$	m^2		g/m^3 $x10^6$			n ng/g dry wt. $x10^{-9}$	m^2	m	g/m^3 $x10^6$	
a	137	984	0.5	1.18	79.54	a	53	984	0.5	1.21	31.55
b	321	1112	0.5	0.87	155.27	b	154	1112	0.5	1.18	101.04
c	662	994	0.5	1.15	378.37	c	345	994	0.5	1.26	216.05
d	146	2671	0.5	1.2	233.98	d	50	2671	0.5	1.16	77.46
e	295	860	0.5	1.21	153.49	e	137	860	0.5	1.15	67.75
f	320	663	0.5	1.21	128.36	f	233	663	0.5	1.17	90.37
g	526	800	0.5	1.21	254.58	g	373	800	0.5	1.18	176.06
h	781	453	0.5	1.19	210.51	h	410	453	0.5	1.17	108.65
i	120	625	0.5	1.17	43.88	i	72	625	0.5	1.24	27.90
j	101	362	0.5	1.18	21.57	j	58	362	0.5	1.23	12.91
k	438	543	0.5	1.16	137.69	K	287	543	0.5	1.18	91.95
l	661	642	0.5	1.18	250.37	l	430	642	0.5	1.20	165.64
					Ms3 = 2047.61						**Ms4= 1167.33**

Table D-7.2: Physicochemical parameters and partitioning coefficient (K_d) for soil core samples

					K_d in L/kg						
ID	Depth, (m)	%TOC	pH	Salinity (PSU)	PFHxA	PFHpA	PFOA	PFNA	PFDA	PFBS	PFHxS	PFOS
	0.5-1		7.1	0.3								
SC1-S2		0.07			0.32	0.32	0.42	0.92	NA	0.32	0.33	2.5
SC1-S2R*	0.5-1	0.06	7.2	0.3	0.33	0.34	0.41	0.75	NA	0.36	0.32	2.2
SC1-S3	1-1.5	0.12	7.7	0.4	1.4	1.4	1.7	2.4	NA	1.9	1.7	7.4
SC1-S4	1.5-2	1.2	7.8	0.2	1.1	1.1	1.3	1.9	NA	0.97	1.1	4.1
SC3-S2	0.5-1	0.07	7.9	0.2	2.1	0.37	2.1	0.67	NA	NA	0.56	8.1
SC3-S2R*	0.5-1	0.07	8.0	0.2	2.1	0.33	2.0	0.60	NA	0.61	0.05	8.0
SC3-S3	1-1.5	0.27	7.8	0.2	0.23	0.24	0.26	0.37	1.5	0.24	0.24	9.4
SC3-S4	1.5-2	0.99	7.8	0.1	0.58	0.62	0.87	1.8	NA	0.57	0.73	8.1
SC4-S1	0-0.5	0.58	8.0	0.3	1.3	1.3	2.3	4.7	NA	1.8	2.3	6.6
SC4-S2	0.5-1	1.2	7.7	0.2	2.0	2.2	2.8	4.4	NA	1.4	2.2	9.6
SC4-S3	1-1.5	0.85	7.8	0.2	1.6	2.0	2.5	5.9	NA	1.4	2.1	14.4
SC4-S4	1.5-2	0.43	6.2	0.3	0.59	0.76	1.4	5.4	NA	0.55	1.2	13.7
SC6-S1	0-0.5	0.71	7.6	0.3	1.9	1.3	1.7	2.4	7.5	NA	1.9	6.2
SC6-S2	0.5-1	0.36	7.8	0.2	2.4	2.5	1.8	2.8	NA	NA	2.2	5.2
SC6-S3	1-1.5	0.46	7.5	0.1	0.21	0.20	0.23	0.27	1.1	0.38	0.24	1.3
SC6-S4	1.5-2	0.12	7.5	0.4	1.3	1.5	1.6	2.1	NA	1.5	1.7	5.8
SC7-S2	0.5-1	0.38	7.9	0.5	1.6	1.8	2.2	4.8	NA	1.6	2.3	29.9
SC7-S3	1-1.5	0.48	7.1	0.2	2.8	1.4	1.4	3.3	12	1.4	1.7	11.4
SC7-S4	1.5-2	0.66	8.8	0.2	2.1	2.4	2.2	2.5	4.9	2.1	2.6	7.4
SC8-S1	0-0.5	0.87	7.9	0.1	0.36	0.36	0.41	0.61	NA	0.72	0.50	2.7
SC8-S2	0.5-1	0.39	7.7	0.2	1.3	1.4	1.6	2.2	12	1.1	1.4	15.3
SC8-S3	1-1.5	1.2	7.8	0.2	0.40	0.48	0.60	1.1	3.9	0.41	0.82	5.1

SC8-S4	1.5-2	0.77	7.4	0.3	1.1	1.3	1.7	2.8	NA	1.1	1.7	9.3
SC11-S1	0-0.5	1.2	8.0	0.2	0.39	0.24	0.33	1.7	2.6	1.4	0.81	8.8
SC11-S2	0.5-1	1.0	7.9	0.3	0.52	0.46	1.1	3.3	20	0.48	0.88	30
SC11-S2R*	0.5-1	1.1	7.8	0.2	0.52	0.46	1.0	3.4	12	0.57	0.85	29
SC11-S3	1-1.5	1.7	8.0	0.2	1.1	1.2	1.3	1.3	7.6	0.90	6.1	12
SC11-S4	1.5-2	1.1	7.7	0.2	2.4	2.4	3.6	5.3	10	NA	3.2	14
SC13-S1	0-0.5	0.32	7.5	0.4	3.1	2.6	3.2	7.2	2.2	4.4	4.9	6.5
SC13-S2	0.5-1	0.33	6.9	0.3	NA	3.5	1.9	3.1	6.2	NA	2.3	6.6
SC13-S3	1-1.5	0.32	7.2	0.7	0.18	1.2	0.6	1.1	NA	NA	3.1	5.4
SC13-S4	1.5-2	0.67	7.9	0.2	1.8	2.0	3.1	4.1	0.22	NA	2.2	12
SC14-S1	0-0.5	0.24	7.9	0.4	0.68	0.57	0.7	4.2	6.3	NA	1.04	9.8
SC14-S2	0.5-1	0.55	7.5	0.2	2.5	2.4	2.7	11	9.9	NA	2.6	19
SC14-S3	1-1.5	1.1	7.7	0.1	0.47	0.55	0.55	1.6	7.6	NA	0.66	6.2
SC14-S4	1.5-2	0.68	7.5	0.2	0.42	0.43	0.57	2.2	9.4	NA	0.72	9.3
SC15-S1	0-0.5	0.97	7.5	0.3	1.9	2.9	4.4	18	NA	NA	NA	5.4
SC15-S2	0.5-1	0.77	7.9	0.2	1.4	2.1	3.3	NA	NA	NA	2.1	9.3
SC15-S3	1-1.5	0.53	8.0	0.4	1.4	1.0	2.6	12	7.2	NA	3.4	24
SC15-S4	1.5-2	1.1	8.2	0.3	4.9	5.2	6.4	NA	NA	3.9	5.7	15

*Results showing test of repeatability for respective samples. NB: soil core-sleeves (SC-S) with respective core and sleeve numbers as samples codes.

Table D-7.3: Mineralogical composition of the core soil samples

ID	Quartz	Pyrite	Calcite	Amphibole	Diopside	Plagioclase (0 < Ca < 0.5)	K-Feldspar	Gypsum	Illite	Kaolinite	Amorphous
C1-S2	94.65	-	-	-	-	1.5	1.1	-	-	-	2.75
C1-S3	88.96	-	-			3.4	0.99	-	-	-	6.65
C1-S4	55.88	-	0.28	1.54	-	21.12	8.21	-	-	-	12.97
C3-S2	93.75	-	-	-	-	1.89	0.54	-	-	-	3.83
C3-S3	76.26	-	0.07	-	-	12.57	5.33	-	-	1.72	4.05
C3-S4	53.05	-	0.37	1.29	-	19.37	7.63	-	-	-	18.29
C4-S1	55.24	-	0.1	1.33	-	20.91	8.1	-	-	-	14.31
C4-S2	55.47	-	0.4	3.37	-	20	8	-	-	-	12.75
C4-S3	52.57	0.74	0.24	3.16	-	21.05	6.66	-	3.53	-	12.05
C4-S4	40.86	1.48	-	2.28	-	22.61	7.89	2.02	5.38	4.3	13.2
C6-S1	83.16	-	-	-	-	3.15	1.18	-	-	-	12.51
C6-S2	93.4	-	-	-	-	1.55	0.81	-	-	-	4.25
C6-S3	92.25	-	0.04	0.34		2.73	0.62	-	-	-	4.02
C6-S4	60.72	-	0.22	1.07	-	14.17	5.23	-	-	5.22	13.36
C6-S4R	59.2	-	0.20	1.3	-	14.53	4.62	-	-	5.84	14.31
C7-S2	90.98	-	0.04	0.47	-	2.92	0.82	-	-	1.15	3.63
C7-S3	62.52	-	-	-	-	7.34	2.09	-	-	9.91	18.15
C7-S4	76.21	-	0.13	3.73	-	9.08	2.16	-	-	1.81	5.69
C8-S1	82.84	-	0.04	0.49	-	3.44	0.94	-	-	1.28	10.98
C8-S2	64.06	-	0.07	1.13	-	14.84	5.11	-	-	-	14.79
C8-S3	55.92	-	0.04	1.78	-	18.51	6.04	-	-	-	17.7
C8-S4	69.12	-	0.61	2.47	-	10.67	4.2	-	-	2.64	10.3
C11-S1	54.77	-	0.25	0.89	-	21.59	8.39	-	-	-	14.1
C11-S2	50.77	-	0.59	1.04	-	21.9	8.04	-	-	3.08	14.6
C11-S2R	51.35	-	0.71	0.84	-	19.78	8.62	-	-	2.76	15.94

C11-S3	52.1	-	0.51	1.35	-	22.3	7.81	-	-	-	15.96
C11-S4	79.22	-	0.1	1.44	0.65	7.6	2.79	-	-	1.61	6.61
C13-S1	77.38	-	0.23	-	-	11.3	3.8	-	-	1.47	5.79
C13-S2	53.43	-	0.89	1.25	-	18.5	7.3	-	-	-	18.68
C13-S4	95.67	-	-	-	-	3.4	0.95	-	-	-	0.04
C14-S1	69.09	-	0.17	0.55	-	10.2	3.58	-	-	-	16.46
C14-S2	56.07	-	0.24	0.92	-	23.2	2.02	-	3.44	2.02	5.41
C14-S3	55.53	-	0.32	0.58	-	22.5	9.3	-	-	-	11.78
C14-S4	55.75	-	0.25	0.79	-	17.8	6.57	-	-	-	18.85
C15-S1	55.69	-	1.2	1.05	-	19.3	7.3	-	-	-	15.43
C15-S2	50.25	-	1.71	-	-	18.6	6.67	-	-	2.54	20.28
C15-S3	48.48	-	2.41	-	-	17.2	5.95	-	-	2.88	23.07
C15-S4	38.32	1.88	1.37	2.01		19.9	6.14		5.63	5.04	19.76

Table D-7.4: Multi-linear regression results which informed the likely independent soil physical factors for elimination to assess the dependence PFASs soil-water partitioning at the study site. Results for PFOA and PFOS are presented separately as (A) and (B) respecttively.

(A) PFOA Dependence	Coefficient	Std. Error	t	P	VIF
Constant	0.00657	0.0464	0.142	0.888	
pH	-0.0328	0.126	-0.26	0.797	1.246
Salinity	2.19	0.524	4.182	<0.001	1.376
TOC	-0.496	0.168	-2.952	0.007	1.919
Calcite	0.175	0.12	1.464	0.155	1.828
Plagioclase	-0.0513	0.045	-1.141	0.264	54.566
Amorphous	-0.0552	0.0255	-2.162	0.04	10.664
Quartz	-0.0444	0.0254	-1.749	0.092	84.079
K-feldspar	-0.0457	0.0449	-1.016	0.319	7.782
Kaolinite	-0.0362	0.0406	-0.893	0.38	3.309

(B) PFOS Dependence	Coefficient	Std. Error	t	P	VIF
Constant	0.00421	0.0421	0.1	0.921	
pH	0.0422	0.115	0.369	0.715	1.246
Salinity	2.784	0.475	5.857	<0.001	1.376
TOC	-0.382	0.152	-2.508	0.019	1.919
Calcite	-0.121	0.109	-1.11	0.277	1.828
Plagioclase	0.0499	0.0408	1.222	0.233	54.566
Amorphous	0.00409	0.0232	0.177	0.861	10.664
Quartz	0.015	0.023	0.651	0.521	84.079
K-feldspar	-0.024	0.0408	-0.589	0.561	7.782
Kaolinite	0.0508	0.0368	1.381	0.179	3.309

Table D-7.5: Estimated distance (m) of PFASs migration within 10 years for nth percentiles

PFASs	Distance (m) of migration within 10 years				
	10^{th} percentile	25^{th} percentile	50^{th}percentile	75^{th} percentile	90^{th} percentile
PFHxA	9.1	6.7	3.2	2.2	1.9
PFHpA	9.1	6.7	3.2	2.1	1.8
PFOA	7.7	5.3	2.6	1.8	1.4
PFNA	6.0	3.7	1.8	1.0	0.6
PFDA	3.2	1.3	0.7	0.5	0.4
PFBS	17	7.7	6.0	3.0	2.3
PFHxS	9.1	5.3	2.6	2.0	1.4
PFOS	1.5	0.80	0.60	0.40	0.30