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CRITICAL REVIEW

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Environmental fate behavior and risk implications of legacy and emerging PFAS across multiple media: a spatiotemporal perspective from Africa and beyond

John Kanayochukwu Nduka, *[ab] Chioma Juliet Alluwo,[b]
Chideraa Courage Offor *[a] and Ogechi Irene Eboh-Ajoku[ac]

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PFAS are a class of emerging contaminants widely used in industrial processes and consumer products, often referred to as “forever chemicals” due to their persistence in the environment. Their long-term bioaccumulation, particularly for long-chain PFAAs, is driven by their low water solubility, strong chemical stability, and resistance to degradation. In contrast, shorter-chain PFAAs, such as PFBA, PFHxA, and PFBS, tend to be more mobile and less bioaccumulative, although they still pose environmental concerns. In Nigeria and many other African countries, research on PFAS remains limited, resulting in critical gaps in baseline data, exposure pathways, and risk assessment. These gaps hinder effective scientific evaluation and policy development. This article reviews the distribution, fate, and behaviour of PFAS across environmental media, examining their persistence, mobility, transformation, and degradation processes. It also highlights ecological and human health risks associated with PFAS exposure through water, soil, and food-chain transfer, and summarizes current global trends in their occurrence. Given recent international findings, there is an urgent need for localized monitoring programs and enhanced analytical capacity to better understand PFAS behaviour under tropical environmental conditions. Addressing these knowledge gaps is essential for advancing environmental health equity and guiding evidence-based national regulation.

Environmental significance

Per- and polyfluoroalkyl substances (PFAS) are now recognized as globally distributed, persistent contaminants whose occurrence across water, soil, air, and biota reflects complex environmental transport and transformation processes. This review synthesizes spatiotemporal trends for both legacy and emerging PFAS, revealing rising burdens in regions with limited regulatory oversight; particularly across Africa, where monitoring remains sparse despite increasing industrial, agricultural, and urban emissions. By integrating environmental fate data with evolving knowledge of exposure pathways and emerging mechanistic evidence of metabolic disruption (*e.g.*, PPARα-linked effects), the review highlights the urgent need for improved analytical coverage, stronger regional monitoring capacity, and targeted risk-assessment frameworks. These insights are essential for guiding policy actions, prioritizing interventions, and preventing further accumulation of PFAS in vulnerable ecosystems and communities.

1 Introduction

Per- and polyfluoroalkyl substances (PFAS) constitute a major class of emerging contaminants that remain largely unregulated in many developing countries, despite growing evidence of their persistence, bioaccumulation, and adverse effects on human and ecosystem health. PFAS are synthetic organofluorine compounds characterized by multiple fluorine atoms bonded to an alkyl chain, conferring exceptional thermal and chemical stability.[1] These compounds encompass a wide spectrum of fluorinated organics; both polymeric and non-polymeric, with diverse physical, chemical, and biological properties, collectively comprising over 5000 known structures.[2] Following the broader definition introduced by the Organization for Economic Co-operation and Development (OECD), PFAS now include any substance containing at least one fully fluorinated methyl (–CF$_3$) or methylene (–CF$_2$–) group, expanding the potential number of PFAS-related compounds in the environment to over seven million.[3] Polymeric PFAS – including PFA, FEP, and ETFE (Table 1) – are primarily classified as

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[a] *Environmental Chemistry and Toxicology Research Unit, Pure and Industrial Chemistry Department, Nnamdi Azikiwe University, P. M. B 5025, Awka, Anambra State, Nigeria. E-mail: co.offor@unizik.edu.ng; johnnduka2000@yahoo.co.uk; Tel: +234 (0) 7061658455; +234 (0) 7039541583*

[b] *Department of Industrial Chemistry, Faculty of Science, Madonna University, Elele, Nigeria*

[c] *Department of Pure Chemistry, Kingsley Ozumba Madiwe University, Orlu, Imo State, Nigeria*

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Table 1 Common names, acronyms, carbon length and functional group of PFAS used in this study

No.	Compound name	Acronym	Carbon chain length	Functional group
1	Perfluorocarboxylic acids	PFCAs	No fixed length	Carboxylic acid
2	Perfluorobutanoic acid	PFBA	C4	Carboxylic acid
3	Perfluoropentanoic acid	PFPeA	C5	Carboxylic acid
4	Perfluorohexanoic acid	PFHxA	C6	Carboxylic acid
5	Perfluoroheptanoic acid	PFHpA	C7	Carboxylic acid
6	Perfluorooctanoic acid	PFOA	C8	Carboxylic acid
7	Perfluorononanoic acid	PFNA	C9	Carboxylic acid
8	Perfluorodecanoic acid	PFDA	C10	Carboxylic acid
9	Perfluoroundecanoic acid	PFUnDA	C11	Carboxylic acid
10	Perfluorododecanoic acid	PFDoDA	C12	Carboxylic acid
11	Perfluorotridecanoic acid	PFTrDA	C13	Carboxylic acid
12	Perfluorotetradecanoic acid	PFTeDA	C14	Carboxylic acid
13	Perfluoro (4-methylpentanoic acid)	PFMPA	C6 (branched)	Carboxylic acid
14	Perfluoro-3,7-dimethyloctanoic acid	PFDMOA	C10 (branched)	Carboxylic acid
15	5 : 3 fluorotelomer carboxylic acid	5 : 3 FTCA	C5	Carboxylic acid
16	Perfluorosulfonic acids	PFSAs	No fixed length	Sulfonic acid
17	Perfluorobutanesulfonic acid	PFBS	C4	Sulfonic acid
18	Perfluoropentanesulfonic acid	PFPeS	C5	Sulfonic acid
19	Perfluorohexanesulfonic acid	PFHxS	C6	Sulfonic acid
20	Perfluoroheptanesulfonic acid	PFHpS	C7	Sulfonic acid
21	Perfluorooctane sulfonate	PFOS	C8	Sulfonate
22	Perfluorononanesulfonic acid	PFNS	C9	Sulfonic acid
23	Perfluorodecanesulfonic acid	PFDS	C10	Sulfonic acid
24	Perfluoro-2-propoxypropanoic acid	GenX	C6 (branched)	Ether + carboxylic acid
25	Perfluoro-2-methoxyacetic acid	PFMOAA	C3	Ether + carboxylic acid
26	6 : 2 fluorotelomer sulfonic acid	6 : 2 FTS	C6 + C2 (partial F)	Sulfonic acid
27	8 : 2 fluorotelomer sulfonic acid	8 : 2 FTS	C8 + C2 (partial F)	Sulfonic acid
28	*N*-Ethyl perfluorooctane sulfonamide	N-EtFOSA	C8	Sulfonamide
29	*N*-Methyl perfluorooctane sulfonamide	N-MeFOSA	C8	Sulfonamide
30	*N*-Ethyl perfluorooctane sulfonamidoacetic acid	N-EtFOSAA	C8	Sulfonamidoacetic acid
31	*N*-Methyl perfluorooctane sulfonamidoacetic acid	N-MeFOSAA	C8	Sulfonamidoacetic acid
32	Fluorotelomer alcohol (6 : 2)	6 : 2 FTOH	C6 + C2 (partial F)	Alcohol
33	Fluorotelomer alcohol (8 : 2)	8 : 2 FTOH	C8 + C2 (partial F)	Alcohol
34	Perfluorooctanoate	PFO	C8	Alkanoates
35	Polyfluoroalkyl phosphate esters	di/PAPs	C4–C10	Phosphoric acid ester
36	Bis (6 : 2 fluorotelomer sulfonamidoethyl) phosphate	diSAMPAP	C6 + C2	Phosphate diester (–O–PO(OH)–O-linkage) with sulfonamidoethyl substituents
37	Perfluoro-3-oxanonane-1-sulfonic acid	9C L-PF$_3$ONS	C9 equivalent	Sulfonic acid (–SO$_3$H) with an ether linkage (–O–) within the fluorinated chain
38	Hexafluoropropylene oxide dimer acid	HFPO-DA	C6	Carboxylic acid

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Table 1 (*Contd.*)

No.	Compound name	Acronym	Carbon chain length	Functional group
39	Perfluoro(4-ethylcyclohexane) sulfonic acid	PFECHS	Cyclohexane ring	Sulfonic acid
40	Trifluoroacetic acid	TFA	C2	Carboxylic acid
41	8 : 2 fluorotelomer carboxylic acid	8 : 2 FTCA	C8 + C3 (partial F)	Carboxylic acid
42	7 : 3 fluorotelomer carboxylic acid	7 : 3 FTCA	C7 + C4 (partial F)	Carboxylic acid
43	Polyfluoroalkyl acids	PFAAs	No fixed length	Carboxylic acid/sulfonic acid
44	Perfluoroalkoxy alkane	PFA	C2–C4	Perfluoroalkoxy group
45	Fluorinated ethylene propylene	FEP	No fixed length	Trifluoromethyl branch
46	Ethylene tetrafluoroethylene	ETFE	No fixed length	Mixed hydrogenated (CH_2) and fluorinated (CF_2) backbone

fluoropolymers, characterized by their high molecular weight, thermal stability, and remarkable chemical inertness.[4] Among the non-polymeric PFAS, PFOA and PFOS remain the most extensively studied and regulated species. However, other congeners such as PFNA, PFHxS, 6 : 2 FTS, and PFOSA (Table 1) are increasingly detected in environmental and biological samples (Table 1). PFAS were first synthesized in 1938 for fluoropolymer production[5] and have since found widespread application in non-stick cookware, water- and stain-resistant fabrics, food packaging, firefighting foams, industrial coatings, and personal care products, owing to their remarkable resistance to heat, water, and oil. The extensive use and environmental persistence of long-chain. PFAS have raised serious concerns regarding their toxicological effects. Health outcomes associated with PFAS exposure vary with compound type, exposure duration, and individual susceptibility factors such as age, sex, and genetic predisposition.[6] Epidemiological studies have linked chronic PFOA exposure to ulcerative colitis and other immune and endocrine disorders.[6]

PFAS are highly resistant to abiotic and biotic degradation, primarily due to the strength of the carbon–fluorine bond (≈ 485 kJ mol^{-1}), one of the strongest in organic chemistry. Their amphiphilic nature; arising from functional groups such as carboxylic (–COOH), sulfonic (–SO_3H), phosphonic acids, and telomer structures, makes them both hydrophobic and lipophobic. Polarity and solubility of PFAS are largely governed by their carbon chain length and functional groups. Long-chain PFAS (*e.g.*, PFOS and PFOA) are less soluble in water and more likely to partition to air–water and soil–water interfaces, promoting accumulation in sediments and biota. In contrast, short-chain analogues exhibit higher aqueous solubility and mobility, enhancing their transport through surface and groundwater systems.[1,7] Major sources of PFAS contamination include consumer products, industrial discharges, and firefighting foams. PFAS have been ubiquitously detected in air, surface and groundwater, soils, sediments, wildlife, and human blood serum worldwide – including remote regions such as the Arctic – demonstrating their global transport potential and environmental persistence.[8–10]

Given the environmental persistence, complex chemistry, and growing evidence of toxicity, accurate quantification and characterization of PFAS in environmental matrices are essential for effective risk assessment and remediation. This review therefore aims to (i) elucidate PFAS exposure pathways, environmental behaviour and fate; (ii) evaluate ecological and endocrine-disrupting health effects; (iii) synthesize global concentration trends and associated risks across environmental compartments; and (iv) identify current research gaps and future directions. Common names and acronyms of PFAS used in this study are presented in Table 1.

2 Exposure pathways, behaviour and fate of PFAS

2.1 Exposure pathways

Exposure pathways of PFAS refer to the various routes through which these substances enter the environment and ultimately the human body. Major pathways include industrial emissions, contaminated drinking water, food, air, and household dust. Understanding these exposure routes, along with the environmental fate and behaviour of PFAS, is critical for accurate risk assessment and mitigation.

2.1.1 Industrial processes. Industrial activities are among the primary sources of PFAS released into the environment. During PFAS manufacturing, processing, and end-use applications, emissions can occur through air, water, and solid waste streams. Production processes such as electrochemical fluorination and telomerization emit both volatile PFAS precursors (*e.g.*, FTOHs) and non-volatile by-products (*e.g.*, PFOA), contributing to widespread contamination of surrounding air, soil, and water ecosystems. Industries involved in metal plating and etching have historically used PFOS-based surfactants as mist suppressants to control aerosol emissions. However, this practice has resulted in PFOS – and related PFAS by-products –

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being released into the environment through wastewater discharge and exhaust air. Untreated wastewater from surface coating and finishing operations can serve as a direct source of PFAS discharge into the environment. In addition, sludge generated from paper and pulp mills may act as a secondary source, releasing PFAS during disposal or land application. Textile and leather finishing industries also emit PFAS through the use of stain-, oil-, and water-resistant coatings. Firefighting activities involving aqueous film-forming foams (AFFF) represent another significant pathway, as these foams contain high concentrations of PFAS that readily infiltrate soils and groundwater.[11]

Field studies corroborate the industrial contribution to PFAS contamination. For instance, Schroeder *et al.*,[12] reported that soils and groundwater surrounding PFAS-emitting facilities in New York, USA, were contaminated with PFOA, with concentrations ranging from non-detectable to 3100 ng L^{-1} in groundwater and 0.62–5.6 ng g^{-1} in soils. In Sweden, Pettersson *et al.*,[13] detected ten PFAS compounds – PFBA, PFPeA, PFHxA, PFHpA, PFOA, PFBS, PFPeS, PFHxS, PFOS, and 6 : 2 FTSA – in wastewater from a waste management facility at concentrations between 440 and 17 000 ng L^{-1}. Similarly, Dauchy *et al.*,[14] observed predominant levels of PFHxA (58–156 ng L^{-1}), PFNA (13–35 ng L^{-1}), and PFOA (7–25 ng L^{-1}) in drinking water near a fluoropolymer production plant. Collectively, these findings underscore the role of industrial processes as major contributors to the environmental burden of PFAS.

2.1.2 Contaminated drinking water, food, air, and dust. Human exposure to PFAS occurs primarily through ingestion of contaminated drinking water and food, as well as inhalation of PFAS-laden air particles and indoor dust. PFAS can leach into surface and groundwater, particularly near contaminated sites such as firefighting training areas, fluorochemical manufacturing facilities, and landfills. Both long-chain (>C6) and short-chain (C4–C6). PFAS have been frequently detected in aquatic systems.[15–17] Dietary intake represents a major exposure pathway. Aquatic organisms can accumulate PFAS from contaminated water and sediments, while crops may absorb PFAS from soils treated with contaminated biosolids or irrigated with polluted water. Food-contact materials, such as grease-resistant paper packaging, can also leach PFAS into food, further contributing to dietary exposure.[18] Commonly contaminated food groups include seafood,[19,20] meat,[21,22] dairy products,[23–25] and leafy vegetables.[26] Long-chain PFAS generally exhibit higher bioaccumulation in seafood compared with short-chain homologues, although contamination from packaging materials appears variable.[27] Inhalation and ingestion of indoor air and dust also contribute to PFAS exposure. Carpets, upholstered furniture, treated clothing, and household sprays can release PFAS into indoor environments, where they may be inhaled or ingested – particularly in occupational or poorly ventilated settings.[28,29]

2.2 Fate and behaviour of PFAS in the environment

2.2.1 Persistence and stability. The presence of perfluoroalkyl moieties in vast majority of PFAS makes them resistant to common degradation processes such as microbial metabolism, hydrolysis, photolysis, and other natural attenuation mechanisms.[30] The remarkable resistance of the perfluoroalkyl moiety to degradation arises from several structural factors: (i) the carbon–fluorine (C–F) bond is the strongest single bond in organic chemistry; (ii) multiple C–F bonds on the same germinal carbon ("per fluorination"), further shorten and strengthen these bonds; (iii) the high electronegativity of fluorine and the strength of the C–F bond hinder nucleophilic attack; and (iv) the strong electron-withdrawing effect of fluorine atoms enhances the stability of adjacent carbon–carbon bonds within the molecular backbone.[30–33] Knight *et al.*[34] demonstrated that PFOA, PFOS, and PFHxS in soils showed negligible degradation or transformation after a six-month incubation period. These compounds remained chemically available for plant uptake, suggesting that these PFAS do not strongly bind to soil particles and can remain mobile within the soil matrix. Although slight increases in sorption coefficient values were observed for PFHxS and PFOA, the magnitude of change was minimal, confirming their chemical persistence and environmental stability.

2.2.2 Mobility in soils and aquatic systems. The mobility of PFAS in soils and aquatic systems is primarily governed by their molecular structure, carbon – chain length, and functional group chemistry. These physicochemical characteristics influence how PFAS interact with soil particles, organic matter, and water, ultimately determining their partitioning, transport, and persistence in the environment. Laboratory and field investigations have consistently shown that short-chain PFAS (typically ≤ C6) exhibit greater aqueous solubility and mobility than their long-chain counterparts (≥C8), resulting in more frequent detection of short-chain compounds in groundwater and surface water systems.[35,36] For instance, PFHxA and PFBS are predominantly detected in the dissolved phase of aquatic environments, allowing them to migrate downstream and infiltrate groundwater more readily than long-chain PFAS, which preferentially associate with suspended particles or sediment surfaces.[37] Molecular structure and functional group also play critical roles in mobility. Comparative studies demonstrate that sulfonate PFAS (*e.g.*, PFOS, PFHxS) generally exhibit stronger sorption affinities to soils and sediments than carboxylate analogues (*e.g.*, PFOA, PFHxA) of similar chain length. This difference arises from the greater hydrophobicity, polarizability, and acidity of the sulfonate group, which enhances electrostatic and hydrophobic interactions with soil organic matter and mineral surfaces.[38–40] As a result, carboxylate PFAS are typically more mobile in aqueous environments, whereas sulfonates exhibit higher retention in soils – though both groups remain environmentally persistent due to their strong C–F bonds and resistance to degradation. Recent evidence indicates that ultrashort-chain PFAS such as TFA and PFPrA display exceptionally high solubility and low sorption capacity. Quan *et al.*,[41] reported median concentrations of 7.40 ng L^{-1} (TFA) and 3.98 ng L^{-1} (PFPrA) in bottled water, demonstrating that these compounds can easily dissolve, migrate through water systems, and contribute to widespread human exposure potential. Similarly, a stormwater

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investigation by Gómez-Ávila *et al.*,[42] found that PFAS with chain lengths ≥C9 exhibited 35–73% particulate association, whereas ≤C8 compounds showed only 17–35%, confirming that chain length and hydrophobicity critically control PFAS partitioning in stormwater and surface waters. Beyond direct aqueous transport, volatile and semi-volatile PFAS precursors such as FTOHs also play a role in PFAS mobility. These compounds can undergo atmospheric transport and deposition onto surface waters, where they transform into stable PFAAs such as PFOA and PFNA, thereby amplifying PFAS loads in aquatic systems and increasing persistence over large spatial scales.[43]

Tang *et al.*,[44] measured emerging PFAS replacements (HFPO-DA and HFPO-TA) in both surface water and sediments of Poyang Lake and found that in certain locations, HFPO-TA concentrations surpassed those of some legacy PFAS – indicating ongoing inputs of these substitutes and their accumulation in sediments. Similarly, Bian *et al.* [45] found that longer-chain PFAS exhibited significantly greater distribution (K_d) and organic carbon-normalized partition coefficients (K_{oc}) in sediments from the Pearl River Delta, reinforcing the chain length-dependent sorption behavior ($p < 0.001$). In a more recent study, Okwuosa *et al.*,[46] observed that PFCAs were predominantly present in the dissolved phase, whereas PFSAs preferentially partitioned into sediments. This functional group-specific partitioning reflects the stronger sorption affinity of sulfonates compared with carboxylates of similar chain length. In soils, PFAS mobility is further modulated by geochemical parameters such as pH, organic matter content, and ionic composition. Long-chain PFAS like PFOS and PFOA exhibit stronger sorption affinity in soils rich in organic matter or clay minerals but can still migrate vertically under certain hydrological or chemical conditions. Xiao *et al.*,[47] observed increasing PFOS and PFOA concentrations with soil depth, suggesting vertical transport and potential contamination of underlying aquifers. Lyu *et al.*,[48] reported that PFOS adsorption was significantly higher in black soils with higher organic matter content (2.57%) than in desert soils (0.05%). Campos-Pereira *et al.*,[49] analyzed six PFAS compounds (PFOA, PFDA, PFUnDA, PFHxS, PFOS, and FOSA) across 11 temperate soils and found a strong inverse relationship between soil pH and PFAS sorption, indicating that higher pH levels (low alkalinity) reduce adsorption – especially for long-chain PFAS. Likewise, Cai *et al.*,[50] demonstrated that increasing the ionic strength of divalent cations (*e.g.*, Ca^{2+}, Mg^{2+}) enhances hydrophobic interactions and decreases soil surface charge, thereby promoting PFAS sorption. Collectively, these studies confirm that PFAS mobility and fate in environmental systems are controlled by an interplay of intrinsic molecular features (chain length, functional group, polarity) and extrinsic environmental factors (pH, ionic strength, organic content, and hydrodynamic conditions). Despite their variable sorption behaviours, both short- and long-chain PFAS exhibit remarkable persistence and the ability to migrate over long distances *via* aqueous pathways, leading to widespread global contamination of soil and water resources.

2.2.3 Air transport and atmospheric behaviour. Volatile PFAS precursors can be emitted into the atmosphere during the production, use, and thermal treatment (*e.g.*, incineration) of PFAS-containing materials. Once airborne, these compounds undergo long-range atmospheric transport and are subsequently deposited onto terrestrial and aquatic environments *via* wet and dry deposition. PFO has been detected in remote marine and Arctic environments far from known point sources,[51] demonstrating the capacity of volatile PFAS precursors to undergo global atmospheric transport before transforming into more persistent products. PFHxA and 5 : 3 FTCA are two transformative and persistent products of 6 : 2 FTOH.[52] Modeling studies further support the role of atmospheric transport in PFAS dispersion. D'Ambro *et al.*,[53] investigated emissions from a fluoropolymer manufacturing facility in North Carolina and found that approximately 5% of total PFAS and 2.5% of GenX emissions were deposited locally, while the remainder underwent long-range transport. Simulated air concentrations reached up to 8500 ng m^{-3} near the source, decreasing sharply to around 10 ng m^{-3} at a distance of 35 km downwind. The study concluded that acid-functional PFAS exhibit higher deposition rates due to greater water solubility and pH-dependent partitioning between gaseous and aqueous phases. Similarly, Zhou *et al.*,[54] detected 12 emerging and 10 legacies ionic PFAS species in fine particulate matter collected near the Chemours Fayetteville Works plant, with several compounds exceeding 1 pg m^{-3} – substantially above regional background levels. Dominant species included PFBA, PFOS, Nafion-related compounds, and other perfluorinated acids. Collectively, these findings highlight atmospheric transformation and deposition as important mechanisms contributing to the widespread distribution of PFAS, including in regions distant from direct emission sources.

2.2.4 PFAS co-contamination and synergistic toxicity. The environmental behaviour of PFAS is strongly influenced by the presence of inorganic and organic co-contaminants. Interactions with metals, microplastics, and dissolved organic matter (DOM) can alter PFAS sorption, mobility, and bioavailability in soils and aquatic environments. Cai *et al.*,[55] demonstrated that heavy metals such as Pb^{2+}, Cu^{2+}, and Zn^{2+} enhance the sorption of long-chain PFAS (*e.g.*, PFOS and PFNA) by neutralizing soil surface charges and decreasing pH; effects that can immobilize PFAS locally and increase exposure risks. Similarly, Shi *et al.*,[56] observed that virgin PVC microplastics adsorbed approximately 50% of PFOS, compared to ∼38% for weathered PVC, suggesting polymer type and aging status significantly influence PFAS partitioning. Additionally, microcystins were shown to co-adsorb to plastics, indicating multi-contaminant interactions on polymer surfaces. Biofilm formation and surface oxidation also modify PFAS behaviour. Yu *et al.*,[57] reported that oxygenated groups on aged microplastics reduce PFAS adsorption relative to virgin plastics, whereas biofilms promote binding and may facilitate limited biodegradation. Moreover, dissolved organic matter dynamically mediates PFAS interactions in soils. Qi *et al.*,[58] found that DOM can enhance PFAS desorption from plant root zones and mitigate microbial toxicity by competing for binding sites. Collectively, these findings indicate that PFAS co-contamination with metals, plastics, and organic matter can modify their transport, retention, and partitioning, complicating predictions of environmental fate.

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Beyond environmental interactions, co-occurring pollutants can amplify PFAS toxicity in organisms. In a population study from China, Su *et al.*,[59] found that co-exposure to PFHpS with arsenic (As) and strontium (Sr) was associated with a 5.4 mL min^{-1} decline in estimated glomerular filtration rate (eGFR), indicating synergistic nephrotoxicity, particularly among women. Experimental studies further demonstrate additive or synergistic effects in aquatic organisms. Soltanighias *et al.*,[60] reported that combined exposure of *Daphnia magna* to PFOS, PFOA, and microplastics increased toxicity by 41%, leading to stunted growth, delayed reproduction, and reduced survival compared to PFAS exposure alone. These findings highlight that PFAS rarely act in isolation in real environments. Co-contaminants can intensify toxicity, alter exposure pathways, and influence biological uptake, underscoring the importance of considering PFAS within complex contaminant mixtures in ecological and human health risk assessments.

3 PFAS transformation and degradation

Terminal PFAAs such as PFOA and PFOS are chemically recalcitrant, but a range of abiotic and biotic processes can transform precursors and some emerging PFAS into shorter-chain PFAAs or other fluorinated intermediates. These processes typically effect transformation (*i.e.*, conversion to other fluorinated species) rather than full defluorination (mineralization to CO_2 and F^-), which remains difficult except under severe laboratory or thermal conditions. Hori *et al.*,[61] demonstrated that UV-activated persulfate produces sulfate radicals ($SO_4^{\bullet-}$) that can oxidize perfluoroalkyl carboxylic acids *via* one-electron transfer and decarboxylation, generating perfluoroalkyl radicals that undergo HF elimination and stepwise chain-shortening. Demonstrated for PFOA and C4–C8 PFCAs, this route yields shorter PFCAs and partial fluoride release; however, PFSAs are much less susceptible, and complete mineralization requires high oxidant doses, elevated temperatures or long irradiation times.[61] Reductive systems such as UV-sulfite and VUV-sulfite generate hydrated electrons (e_{aq}^-), which drive sequential C–F bond cleavage and achieve substantial defluorination of many PFAS under controlled laboratory conditions. Liu *et al.*,[62] reported that optimized configurations – including iodide-assisted UV-sulfite and VUV irradiation – greatly enhance e_{aq}^- yield, leading to rapid PFAS degradation and high fluoride release in oxygen-free, alkaline solutions. However, the presence of dissolved oxygen, natural organic matter, and other radical scavengers in real waters severely suppresses electron availability, resulting in markedly reduced PFAS destruction efficiency. PFSAs are generally more challenging to reduce than PFCAs, although targeted studies under ideal conditions have shown progress. Anodic oxidation (*e.g.*, boron-doped diamond) and cathodic hydro defluorination have shown to decompose PFAS *via* direct electron transfer and radical generation. Recent work summarized by Yin *et al.*,[63] shows under laboratory and pilot conditions some systems have achieved high fluoride release and near-complete destruction for selected PFAS (including PFOA, PFOS, and GenX (HFPO-DA)). Nevertheless, energy intensity, electrode fouling, and formation of partially defluorinated intermediates limit routine full-scale deployment.

In contrast to terminal PFAAs, polyfluorinated precursors (*e.g.*, FTOHs, diPAPs, PAPs, sulfonamides) are frequently biotransformed by microbial communities in activated sludge, soils and sediments to yield persistent PFAAs (PFCAs/PFSAs). Laboratory and field studies (*e.g.*, FTOH and diPAP incubations) demonstrate sequential oxidative steps – analogous to β-oxidation and side-chain oxidation – producing PFAA end-products rather than complete defluorination. For instance, Dinglasan *et al.*,[64] showed that 8 : 2 FTOH biodegrades to form a suite of PFCA intermediates, including 8 : 2 FTCA and 7 : 3 FTCA, before producing PFOA as a terminal product. Liu and Liu[65] demonstrated that 6 : 2 and 8 : 2 diPAPs undergo aerobic biotransformation in sludge to yield multiple PFCAs. In another study, Butt *et al.*,[66] summarized microbial pathways converting perfluorooctane sulfonamido compounds into PFOS. As reviewed by Liu & Mejia Avendaño[67] and supported by LaFond *et al.*,[68] transformation rates and products depend strongly on redox conditions (aerobic *vs.* anaerobic), microbial consortia and matrix composition. Collectively, these pathways show that while transformation of PFAS and precursors is well documented, complete defluorination and mineralization are exceptional and typically require harsh chemical, thermal or electrochemical conditions. Consequently, many treatment and environmental processes act to redistribute or convert PFAS into other persistent fluorinated compounds; often creating secondary sources of long-lived PFAAs that sustain environmental contamination. For each method, effectiveness in real environmental matrices is constrained by matrix scavengers, energy requirements, by-product formation, and scale-up issues.

4 Ecological and health impacts of PFAS exposure

4.1 Ecological impacts

One of the major environmental concerns with PFAS is their bioaccumulation potential. Unlike many organic pollutants that primarily accumulate in fatty tissues, PFAS bind strongly to proteins in blood, muscle, skin, and liver.[69] For example, Shi *et al.*,[70] reported that PFOS predominantly accumulated in the blood, followed by the liver, brain, and muscles of freshwater fish from Beijing; demonstrating its strong affinity for serum proteins rather than lipids. Similarly, Valdersnes *et al.*,[71] reported geographical trends in PFOS concentrations in cod livers sampled along the Norwegian coast: livers from eastern fjords and harbors had significantly higher PFOS than those from the western and northern regions, reflecting spatial differences in PFAS pollution. PFAS have been detected across multiple trophic levels, significantly elevating risks to aquatic wildlife. In Lake Michigan, PFAS were found in all analyzed fish species, with PFOS being the dominant compound.[72] Bioaccumulation

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levels in aquatic organisms depend on species, habitat, trophic level, and physiology, with benthic fish showing higher accumulation compared to pelagic species.[72,73] Beyond fish, PFAS contamination has also been detected in marine mammals such as seals, whales, and dolphins, with high concentrations reported in muscle and liver tissues. These elevated levels have been associated with immune system suppression or dysregulation, reducing the animals' ability to fight infections, as well as renal (kidney) and hepatic (liver) dysfunction, indicating significant physiological stress and potential population-level health risks.[74–76] Increasing evidence also points to PFAS exposure in amphibians and reptiles, particularly in serum, eggs, tissues, and hatchlings.[77–79] Although amphibians and reptiles are increasingly recognized as being vulnerable to PFAS exposure, the mechanisms of accumulation differ fundamentally from those of lipophilic pollutants such as PCBs or dioxins. PFAS are amphiphilic compounds that preferentially bind to proteins rather than lipids, leading to bioaccumulation in blood, liver, and kidney rather than fatty tissues. In mammals, this process is mediated by organic anion transporters (OATs) in the kidney, which misidentify PFAS as endogenous fatty acids, promoting reabsorption and reducing excretion.[80,81] Similar protein-binding and renal transport mechanisms may contribute to PFAS retention in amphibians, whose permeable skin and aquatic habitats further enhance direct uptake from contaminated water. This protein-mediated mechanism explains the widespread detection of PFAS in amphibian serum, eggs, and tissues, even at low environmental concentrations.

Terrestrial ecosystems are similarly affected. An eight-year investigation of PFAS in an urban terrestrial system (soil, earthworms, bird eggs, and livers from red fox and brown rat) revealed PFOS concentrations significantly above background levels.[82] Elevated PFAS concentrations were also found in polar bears and Arctic foxes, increasing with trophic level as marine prey replaced terrestrial food sources due to climate change.[83] Such widespread contamination raises concern due to the potential immune-toxic and endocrine-disrupting effects in wildlife. In zebrafish, PFOA exposure was linked to impaired fertilization and hatching success.[84] PFAS-induced oxidative stress in mammals has also been associated with cellular and molecular damage, reduced antioxidant defenses, and organ dysfunction.[85] Further, PFAS toxic effects on plant have been documented. PFAS bioaccumulates in plants at levels ranging from ng g^{-1} to μg g^{-1}, depending on species and soil contamination. Li *et al.*,[86] reported that while the "50% inhibition concentration" (IC_{50}) for plant growth is often much higher than environmentally relevant concentrations, PFAS still disrupt biochemical functions: they generate reactive oxygen species (ROS), impair organelle function, and perturb photosynthesis, nitrogen/carbon metabolism, and gene expression. Xu *et al.*,[87] found that PFAS are primarily taken up by plant roots from soil. Long-chain PFAS (*e.g.*, PFCAs with ≥ C7 and PFSAs ≥ C6) tend to remain largely in the roots, while short-chain PFAS distribute more into above-ground tissues (stems, leaves). The authors report variable bioaccumulation factors (BAFs) across species and organs, with the order: leaf vegetables > root vegetables > flower vegetables > shoot vegetables, indicating differing PFAS risks by crop type, with PFOA often dominating in plant tissue because of its high-water solubility and mobility. In another study, PFAS uptake, accumulation, and even partial metabolism (conversion/transformation) have been documented in higher plants.[88] The rate of uptake and accumulation depends strongly on PFAS chemical properties (chain length, functional group), plant species, environmental conditions, and competing ions in soil. Metabolism in plants (*e.g.*, partial dealkylation or transformation) is still poorly understood, creating gap for more research, especially on emerging PFAS. Further, as a means of environmental remediation, some aquatic plants (*e.g.*, macrophytes) can uptake PFAS and may be used for phytoremediation, but efficiency depends on species, PFAS type, and environmental factors.[89] Factors that influence uptake include root morphology, transpiration rate, PFAS chain length, and plant compartment (root *vs.* shoot). However, PFAS exposure can have phytotoxic effects in these plants (reduced growth, metabolic disruption), which complicates remediation efforts. There is a need for more mechanistic studies using omics, long-term field experiments, and a better understanding of PFAS metabolism in plants.

4.2 Human impacts

4.2.1 Endocrine-disrupting effects. PFAS-contaminated drinking water has been linked to endocrine disruption, affecting thyroid function, sex hormones, puberty timing, and cancer risk. Xie *et al.*,[90] demonstrated significant associations between serum PFOS, PFHxS, and PFOA with altered testosterone (TT), estradiol (E), and sex hormone-binding globulin (SHBG) levels, with age- and sex-dependent effects. Similarly, Du *et al.*,[91] reported reduced thyroid-stimulating hormone (TSH) in children and increased free thyroxine (FT4) and triiodothyronine (T3) in adults exposed to PFAS. In Belgium, van Larebeke *et al.*,[92] observed altered thyroid and sex hormone levels in adolescents living within 5 km of a PFAS-contaminated site. High PFAS concentrations in drinking water have been linked to elevated cholesterol, immune toxicity, osteoporotic fractures, reproductive disorders (*e.g.*, PCOS, endometriosis), type 2 diabetes, cancers, and foetal exposure.[93–99] In Kallinge, Sweden, firefighting foam contamination of groundwater was associated with elevated PFAS levels in residents' blood, particularly in children, correlating with increased risks of cancer, diabetes, and immune disorders.[100] Similarly, in Merrimack, New Hampshire, PFOA contamination from an industrial source was linked to elevated thyroid, colon, and prostate cancer rates compared to control communities.[101]

While epidemiological data remain correlational, emerging experimental studies are beginning to reveal mechanisms. For example, in mice, PFOA increased serum and hepatic cholesterol in a PPARα-dependent manner.[102] Moreover, recent reviews identify PFAS-mediated activation of nuclear receptors (PPARα/γ, CAR, PXR), disruption of bile-acid/lipid homeostasis, oxidative stress and epigenetic modifications as plausible mechanistic underpinnings.[103]

4.2.2 PFAS structural mimicry and fatty acid-mediated toxicity. Many PFAS, such as PFOA and PFOS, structurally

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Table 2 Comparison of natural fatty acid binding sites and PFAS interference mechanism[a]

Binding site/target	Role in fatty acid metabolism	Natural fatty acid interaction	PFAS interference mechanism	Representative PFAS/evidence
PPAR-α/PPAR-β (nuclear receptors)	Regulate lipid oxidation, storage, and inflammatory signaling	Long-chain fatty acids and eicosanoids act as natural ligands, activating transcription of lipid metabolism genes	PFAS mimic fatty acids and bind to PPARs, causing aberrant activation or suppression of lipid and immune pathways	PFOA significantly increased mouse and human PPAR-α, and mouse PPAR-β activity in transfected cells relative to control relative to control[105]
Liver fatty acid binding protein (L-FABP1)	Transports fatty acids to organelles for β-oxidation and esterification	Binds hydrophobic tails of C16–C20 fatty acids *via* internal lipid site	PFAS competitively bind to L-FABP1, displacing natural fatty acids and altering intracellular lipid transport	PFOS and PFOA occupied the fatty acid binding site on L-FABP, blocking the normal binding of natural fatty acids (represented by 11-(5-dimethylaminonapthalenesulphonyl)-undecanoic acid (DAUDA))[106]
Serum albumin	Main plasma carrier of free fatty acids	Binds fatty acids at multiple hydrophobic sites to maintain plasma solubility	PFAS bind strongly to albumin, prolonging blood half-life and reducing fatty acid transport efficiency	When PFOA and PFNA bind to bovine serum albumin, the fluorine signals in their NMR spectra shift and broaden, showing that the PFAS molecules interact closely with the protein surface and form a stable complex[107]
Acyl-CoA synthetase (ACS)	Activates fatty acids for oxidation or lipid synthesis	Binds fatty acids at active site, forming acyl-CoA derivatives	PFAS inhibit ACS activity, reducing β-oxidation and promoting lipid accumulation	Early biochemical studies show that certain perfluorinated carboxylates (*e.g.*, PFOA/PFNA/PFDA) inhibit long-chain acyl-CoA synthetase activity in isolated hepatocytes and mitochondria, which would be expected to reduce β-oxidation and contribute to lipid accumulation. However, direct measurements of ACS inhibition in contemporary mammalian models are limited, and PFAS-induced steatosis likely reflects multiple converging mechanisms (*e.g.*, PPAR signaling, mitochondrial/peroxisomal effects, and membrane perturbation)[108]
Cell membrane phospholipids	Structural component maintaining membrane integrity and signaling	Fatty acids modulate membrane fluidity and receptor clustering	PFAS integrate into lipid bilayers or alter surface charge, affecting receptor function and immune signaling	PFOS has been shown to perturb the architecture of cholesterol- and sphingolipid-rich plasma-membrane microdomains (lipid rafts) in immune cells, thereby impairing cell-surface receptor clustering and downstream signalling[109,110]

[a] PPARs = peroxisome proliferator-activated receptors.

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resemble long-chain fatty acids (*e.g.*, palmitic or oleic acid) due to their hydrophobic alkyl tails and polar functional groups (–COOH or –SO_3H). This similarity allows PFAS to compete with natural fatty acids for binding to transport proteins, enzymes, and nuclear receptors; including peroxisome proliferator-activated receptors (PPARs) – that regulate lipid metabolism, immune function, and energy homeostasis.[104] These competitive interactions can disrupt normal lipid signaling and metabolism, contributing to hepatic steatosis, immune suppression, and endocrine dysregulation. A detailed comparison of natural fatty acid binding sites and PFAS interference mechanisms is presented in Table 2.

5 Global trends in PFAS contamination (2018–2025): a spatiotemporal and risk-based study across biological and non-biological matrices

5.1 Africa

Research on PFAS contamination across African environments remains limited due to inadequate monitoring capacity, analytical constraints, and shortage of trained personnel. One major challenge is that current analytical methods can detect only a few dozen well-characterized PFAS, while over 7 million PFAS compounds (including precursors and derivatives) are registered globally. This analytical gap contributes significantly to incomplete environmental monitoring and underestimation of total PFAS burdens.

In the Apies River, Pretoria (South Africa), surface water PFAS concentrations exceeded the EU drinking water guideline (4.4 ng L^{-1}), while sediment PFOS levels surpassed 13.5 ng g^{-1}. PFCAs dominated the water phase, whereas PFSAs were more abundant in sediments.[46] However, the study focused on dry-season sampling, limiting its spatial and temporal coverage. The authors recommended implementing upstream pollution control to reduce inputs. In Asejire and Eleyele Lakes (Southwestern Nigeria), Adeogun *et al.*,[111] detected novel PFAS (di-SAMPAP and 9C L-PF_3ONS) and observed biomagnification of PFOS, PFUnA, PFDA, and EtFOSE in fish. Although the study duration (8 months) and site coverage were limited, the findings underscored urbanization as a primary driver of PFAS contamination in tropical freshwater systems. Similarly, in the Vaal River (South Africa), PFOS was the dominant PFAS detected in biota, reaching concentrations up to 289 ng g^{-1} in fish liver.[112] Although these values indicate substantial bioaccumulation, the associated human health risk is likely limited, as fish liver is not commonly consumed in Nigeria or most African regions. Consequently, evaluating PFAS levels in edible fillets or whole-fish tissues would yield a more accurate assessment of dietary exposure. For example, marine seafood from the Gulf of Guinea contained ΣPFAS in muscle tissues (reported in pg per g wet weight), with PFOS and long-chain PFCAs being the predominant compounds. Estimated daily intakes and hazard ratios for PFOS in croaker muscle were below the European Food Safety Authority (EFSA) tolerable weekly intake (TWI) threshold of 4.4 ng per kg per bw per week, suggesting no immediate exceedance of current safety limits.[113] However, these findings underscore the importance of continued monitoring to capture potential long-term exposure risks. In the Nairobi River Basin (Kenya), Chirikona *et al.*,[114] reported elevated PFUnDA (39.2 ng L^{-1} in water) and PFOA (39.62 ng g^{-1} in sediment; 29.33 ng g^{-1} in plants). Sediments and water exhibited higher long-chain PFAS concentrations than vegetation, confirming chain-length-dependent partitioning. In the aMatikulu and uMvoti estuaries (South Africa), fifteen PFASs were analyzed across water, sediment, invertebrates, and fish.[115] Thirteen were detected in all matrices, with PFOA dominant – ranging from 171–258 ng L^{-1} (aMatikulu) and 711–788 ng L^{-1} (uMvoti). The uMvoti estuary reported the highest PFOA levels in South Africa. Although *Oreochromis mossambicus* accumulated PFAS in tissues, the concentrations remained below minimum risk levels (MRLs). Similarly, in Lake Hawassa (Ethiopia), Melake *et al.*,[116] found higher PFAS concentrations in fish liver than in muscle tissues. PFOA dominated in both water (6.93 ng L^{-1}) and sediment (0.23 ng per g dw). Although current exposure levels posed low immediate risk, prolonged fish tissue consumption could be linked to potential health effects. In Lake Victoria (Uganda), Arinaitwe *et al.*,[117] reported low-to-moderate PFAS exposure in drinking water, with mean concentrations ranging from 1.00 ng L^{-1} (atmospheric deposition) to 23.8 ng L^{-1} (Nakivubo Channel). PFHxA and PFOS showed the highest influx and retention, suggesting recycling of PFAS from urban runoff into the drinking water system. In southern Nigeria, Ololade *et al.*,[118] detected PFOS and PFOA in sediment, pore water, and surface water, with PFOS concentrations higher in aqueous media (3.9–10.1 ng per L surface water; 10.9–20.4 ng per L pore water; 5.1–10.4 ng per g sediment). The study found that increasing organic matter enhanced PFOS enrichment in sediments, highlighting the role of natural carbon in PFAS sorption.

Mwapasa *et al.*,[119] investigated PFAS exposure among 605 pregnant women (mean age: 24.8 years) and their infants in Southern Malawi. Serum analysis revealed significantly higher concentrations of PFOA, PFNA, and ΣPFOS in urban participants compared to rural counterparts. The study further showed that ΣPFOS was inversely associated with gestational age, while PFOA, PFNA, and ΣPFHxS were negatively correlated with birth length and birth weight, indicating potential developmental toxicity linked to urban PFAS exposure.

5.2 Asia

In Asia, PFAS contamination has been widely detected across aquatic and terrestrial ecosystems. Li *et al.*,[120] analyzed 10 PFAS in groundwater from the North China Plain (NCP) and reported total concentrations up to 13.34 ng L^{-1}, dominated by PFOA and PFHxA (detected in 98.39% and 95.16% of samples, respectively). Although the levels were relatively low, they reflect the influence of industrial emissions in the region. Similarly, Li *et al.*,[121] identified untreated industrial effluents and wastewater as the major sources of PFAS in NCP groundwater, with PFOA

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and PFHpS being the most prevalent compounds. While overall human health risks were low, children were found to be the most sensitive population. In South Korea, 16 PFAS were detected in shallow groundwater with a mean ΣPFAS of 45.2 ng L^{-1}, dominated by short-chain compounds (C < 8). The hazard quotients (HQ < 1) for PFOA and PFOS *via* multi-exposure routes indicated low potential health risk.[122] Surface waters near major airports in Beijing, Shanghai, and Guangzhou contained elevated PFAS levels, with PFOA concentrations reaching 342 ng L^{-1} at Shanghai Pudong International Airport. Total PFAS concentrations varied regionally – Beijing (19.0–62.8 ng L^{-1}), Shanghai (25.6–342 ng L^{-1}), and Guangzhou (7.35–72.7 ng L^{-1}) – with long-chain PFAS preferentially bound to particulate phases.[123] Lin *et al.*,[124] reported, for the first time, the occurrence of HFPO-DA (<0.086–21.5 pg m^{-3}) in atmospheric particulate matter across nine Asian cities. While PFOA and PFOS dominated in China, short-chain and emerging PFAS (*e.g.*, chlorinated polyfluorinated ether sulfonate (F-53B)) were more prevalent in South Korea, India, and Japan. Most PFAS were associated with fine particles, though PFOS and its alternatives tended to bind to coarser fractions.

In aquatic biota, edible bullfrog tissues from southern China contained ΣPFAS ranging from 3.36 to 84.07 ng per g dw, with the stomach and intestine showing the highest concentrations – lower than corresponding water (50.26–364.25 ng L^{-1}) and sediment (2.89–162.26 ng per g dw) levels.[125] The study also detected F-53B, a PFOS alternative, across all habitat samples, exhibiting a higher bioaccumulation factor. Despite detection, hazard ratios for most PFAS were <1, suggesting minimal non-cancer health risk. A subsequent study found PFOS and F-53B prevalent in marine algae (*Porphyra haitanensis*) and fish (*Siganus fuscescens*), indicating widespread distribution in aquaculture zones of southern China.[126] Similarly, Diao *et al.*,[127] reported significant bioaccumulation of PFOS, PFNA, and PFBA in the southern China Sea food web, underscoring their persistence and stability in aquatic ecosystems.

Sharma *et al.*,[128] examined PFBS and PFOS toxicity in human kidney epithelial (HEK293T) cell lines exposed to contaminated drinking water from Assam, India, and found PFBS to be more cytotoxic than PFOS. PFBS significantly disrupted lipid metabolism, particularly glycerophospholipid pathways, affecting cellular function and integrity. In Malaysia, serum analysis of 219 individuals revealed detectable PFAAs, with PFOS as the dominant compound (up to 32.57 ng mL^{-1}). Regression modeling showed that PFAS concentrations were higher in men than women and positively correlated with age.[129] In Papua New Guinea, pooled serum samples contained 11 PFAS, including PFAA and 9Cl-F53B – a PFOS substitute – marking the first detection of this compound in the region. The PFAS profile resembled those reported in Europe and Malaysia, suggesting local industrial contributions.[130]

5.3 Europe

Long-chain PFAS are consistently detected across European aquatic ecosystems. Stecconi *et al.*,[131] found PFOS and long-chain PFCAs in fish liver (3.1–10 ng g^{-1}) at higher levels than muscle (0.032–1.7 ng g^{-1}) from Lake Trasimeno (Italy), with concentrations below EU safety thresholds but indicative of environmental persistence. In the Belgian North Sea, PFOS dominated across fish liver (up to 107 ng per g ww), muscle (up to 24 ng per g ww), and crustaceans (0.29–5.6 ng per g ww), with elevated PFTrDA near estuaries. While trophic transfer was limited, some species posed potential human consumption risks.[132]

Grønnestad *et al.*,[133] found widespread long-chain PFAS in soils of Norwegian ski areas (≥70%), with PFOS biomagnifying from worms to voles (biomagnification factor >1). In Germany, Rupp *et al.*,[134] detected elevated PFAS in wild boar liver from contaminated sites, with PFOS concentrations ranging from 82 ng g^{-1} (background) to 426 ng g^{-1} (CPSS). PFOA dominated industrial emission sites (650 ng g^{-1} ww). In the UK, O'Rourke *et al.*,[135] analyzed 50 otters and detected multiple PFCAs, PFSAs, and PFOS in livers, highlighting widespread PFAS contamination in British freshwater ecosystems. In Barcelona, Spain, PFHpA concentrations in tap water were positively correlated ($r = 0.20$) with plasma levels among third-trimester pregnant women, suggesting even low-level exposure contributes to bodily PFAS accumulation.[136] Similarly, Pitter *et al.*,[27] found significantly elevated serum PFOS, PFOA, and PFHxS in adolescents and young adults exposed to PFAS-contaminated drinking water compared with reference populations. Multiple regression analysis confirmed drinking water as the primary exposure route. A large-scale survey of 1957 European adolescents (12–18 years old) reported geometric mean serum concentrations of 2.13 μg L^{-1} (PFOS), 0.97 μg L^{-1} (PFOA), 0.30 μg L^{-1} (PFNA), and 0.41 μg L^{-1} (PFHxS), with higher exposure levels in Northern and Western Europe. Frequent consumption of seafood and eggs was strongly associated with PFOS and PFNA serum concentrations.[137]

5.4 Americas

National-scale assessments consistently show that PFAS occur widely across U.S. drinking-water systems, typically at low ng L^{-1} concentrations but with substantial spatial variability. Recent syntheses estimate that roughly 45% of U.S. drinking-water samples contain at least one detectable PFAS, with higher detection frequencies in urban areas and locations influenced by industrial or military activities.[138] While these surveys document widespread exposure potential, they also highlight the uneven distribution of PFAS contamination across the country. A prominent contamination case is the lower Cape Fear River (North Carolina), where elevated concentrations of HFPO-DA (GenX) and other emerging PFAS have been documented. To evaluate biological impacts, Guillette *et al.*,[139] measured 23 PFAS in Striped Bass (*Morone saxatilis*) collected from the river ($n = 58$) and compared them with a reference population raised in a well-water aquaculture facility ($n = 29$). PFOS dominated the PFAS profile in wild Striped Bass and was detected in every individual, reaching serum concentrations of 977 ng mL^{-1}. PFNA and PFDA were also ubiquitous, and PFHxS occurred in >98% of samples. Emerging PFAS such as HFPO-DA (0.24–5.85 ng mL^{-1}) and Nafion byproduct 2 (0.2–1.03 ng mL^{-1})

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were detected in 48% and 78% of wild fish, respectively. In contrast, Striped Bass from the reference facility exhibited ~40-fold lower total PFAS, with HFPO-DA detected in only 10% of fish and Nafion byproduct 2 entirely absent. These elevated PFAS burdens in Cape Fear River fish were significantly associated with biomarkers of liver dysfunction and altered immune responses, indicating ecologically relevant sublethal effects. Beyond biota, PFAS contamination of freshwater fish is a growing public health concern. A recent national evaluation reported that fish fillets from U.S. rivers and streams contained a median total PFAS concentration of 9500 ng kg^{-1}, with even higher median levels in the Great Lakes (11 800 ng kg^{-1}). PFOS accounted for approximately 74% of total PFAS. Notably, PFAS concentrations in freshwater fish were 278 times higher than those measured in commercially available fish analysed by the U.S. Food and Drug Administration (2019–2022). Exposure modelling suggests that consuming one serving of freshwater fish per year at these median concentrations would substantially increase human serum PFOS levels. Because subsistence and culturally dependent fishing communities disproportionately rely on local catch, these contamination patterns represent a clear instance of environmental injustice in the United States.[140] PFAS contamination in wastewater solids is another important pathway. A multi-facility study of 12 U.S. water resource recovery facilities (WRRFs) revealed significant analytical and treatment-related challenges in quantifying PFAS in sludge and biosolids.[141] Cross-laboratory comparisons showed large discrepancies in measured concentrations: for example, 5 : 3 FTCA in the same post-stabilized sludge sample ranged from 21 ng g^{-1} to 151 ng g^{-1} depending on the laboratory. Across all WRRFs, PFAS concentrations were consistently higher in post-stabilized biosolids compared with pre-stabilized sludges, even after accounting for solids reduction during stabilization. These findings suggest both analytical uncertainty and apparent PFAS enrichment during sludge treatment, underscoring the need for standardized methods, improved mass-balance studies, and clearer regulatory guidance on biosolids land application. Further south, PFAS contamination of wastewater systems has also been documented in Mexico. Rodríguez-Varela *et al.*,[142] conducted monthly monitoring of wastewater in Mexico City, quantifying five PFCAs (PFBA, PFHxA, PFHpA, PFOA, PFUnA). Total PFCA concentrations averaged 419.4 ± 24.3 ng L^{-1} in the underground sewer system and 591.1 ± 39 ng L^{-1} in the open-air canal, indicating substantial PFCA loading in surface wastewater channels. Approximately 60 km downstream, where canal water is used for irrigation, short-chain PFCAs were most abundant – PFBA: 176.9 ng L^{-1}, PFHxA: 133.4 ng L^{-1}, PFHpA: 116.6 ng L^{-1} – while long-chain PFOA and PFUnA persisted at 133.1 ± 3.5 ng L^{-1} and 23.5 ± 6.5 ng L^{-1}, respectively. These findings highlight the predominance and mobility of short-chain PFAS in the region and indicate sustained environmental inputs with downstream transport into agricultural areas. A basin-wide survey of 26 sites in the Piracicaba, Capivari, and Jundiaí (PCJ) rivers, São Paulo, showed that both agriculture and urbanization significantly shape contaminant patterns, with pharmaceuticals such as caffeine (up to 13 500 ng L^{-1}) and carbamazepine (1240 ng L^{-1}) dominating urban-impacted sites, while pesticides like atrazine (1930 ng L^{-1}) were highest in agricultural zones. Hormones (estrone up to 14 ng L^{-1}) and PFAS (PFOA up to 92 ng L^{-1}) were also widely detected, contributing to multiple ecological risk "hotspots." Overall, the mixture of pollutants; often 30+ compounds per site, highlights chronic contamination and the combined pressures of wastewater discharge and agricultural runoff in the PCJ basin.[143]

5.5 Oceania

Similar to American studies, research in Oceania, particularly in Australia and New Zealand, have increasingly reported PFAS contamination in wastewater treatment plants, surface waters, sediments, and drinking water sources, with both legacy PFAS (*e.g.*, PFOA, PFOS) and emerging compounds (*e.g.*, HFPO-DA, 6 : 2 FTS) being detected. Lenka *et al.*,[144] analyzed 38 PFAS species – including long-chain, short-chain, ultrashort, and precursor compounds – in two urban wastewater treatment plants (WWTPs), their effluents, downstream receiving waters, and the drinking water treatment system in New Zealand. $\sum$PFAS ranged from 0.1 to 13 ng L^{-1} across wastewater samples. Following secondary biological treatment, PFHxA (a short-chain PFCA) increased by 41.6%, while 6 : 2 FTS (a precursor) decreased by 49.7%, suggesting in-plant transformation of 6 : 2 FTS into PFHxA. The linear isomer of PFOS (PFOS-L) declined by ~48% in effluent, likely due to partitioning into sludge. PFPrA was detected at 5.5 ng L^{-1} in treated drinking water, though its source was uncertain. The study highlights a circulating PFAS cycle in urban water systems characterized by partial transformation, incomplete removal, and re-release. Coggan *et al.*,[145] measured 21 PFAS in both aqueous and solid phases of 19 WWTPs across Australia. Total PFAS in wastewater effluent ranged from 9.3 to 520 ng L^{-1}, with a median of 80 ng L^{-1} and a mean of 110 ng L^{-1}. PFAS levels in sludge ranged from 2.0 to 130 ng per g dry weight, with a median of 12 ng g^{-1}. Long-chain PFAS preferentially partitioned to solids, with partition coefficients from 0.05 to 1.22 log units, illustrating that WWTPs act as a concentrating source of long-chain PFAS in sludge, which may pose environmental risks if biosolids are reused or disposed. Paige *et al.*,[146] surveyed 33 PFAS across 65 sites spanning residential, industrial, municipal, and rural land uses in Melbourne area, Australia. At least one PFAS species was detected in 98% of water samples, but only 8% of sediment samples. Median total PFAS (Σ33 PFAS) in surface water, excluding a high airport point-source, was 63.5 ng L^{-1} (average 78.6 ng L^{-1}, range < detection limit to 526 ng L^{-1}). The PFAS with the highest median concentrations were: PFBA 11.3 ng L^{-1}, PFHxA 9.2 ng L^{-1}, PFOA 8.3 ng L^{-1}, PFOS 8.0 ng L^{-1}, PFPeA 7.5 ng L^{-1}, PFHpA 3.2 ng L^{-1}, PFHxS 2.9 ng L^{-1}. One site near an airport exhibited extremely elevated PFAS levels (Σ33 PFAS = 4261 ng L^{-1}), identifying a strong point-source input. Page *et al.*,[147] modeled long-term transport of PFAS, particularly PFOS, under managed aquifer recharge (MAR) schemes using treated wastewater and stormwater. Using sorption isotherms, advection-dispersion, and decay models over 100 years, the simulation predicted that PFOS can persist and migrate long

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distances in the subsurface. This highlights a mechanistic risk: MAR may inadvertently transport PFAS into drinking water supplies, emphasizing the need for careful system design and monitoring. Mahinroosta & Senevirathna,[148] applied numerical modeling to simulate PFOS fate in a contaminated site in New South Wales. Their model incorporated advection, dispersion, adsorption, and decay, calibrated with LC/MS soil data. Predictions matched measured PFOS concentrations in monitoring wells and indicated that, without intervention, PFOS levels in groundwater would exceed drinking-water guideline values over time. Scenario testing demonstrated that active remediation strategies, such as source removal or pump-and-treat, are necessary to prevent PFOS migration, whereas passive approaches alone are insufficient.

5.6 Polar/Arctic regions

Per- and polyfluoroalkyl substances enter the Arctic mostly through long-range transport and local pollution.

Ahrens *et al.*,[149] measured 43 PFAS in water (meltwater, freshwater, seawater), snow, soil, and sediments across Svalbard archipelago, Norwegian Arctic. Mean $\sum$PFAS concentrations were 6.5 ± 1.3 ng L^{-1} in meltwater, 2.5 ± 1.7 ng L^{-1} in surface snow, 2.3 ± 1.1 ng L^{-1} in fresh water, and 1.05 ± 0.64 ng L^{-1} in seawater. In lake sediments, they found 0.084 ± 0.038 ng per g (dw), and in marine sediments, median levels were as low as 0.015 ng per g (dw). The PFAS composition suggested that local sources (*e.g.*, airport firefighting site, landfill) contributed PFSA and 6 : 2 FTSA, while long-range atmospheric transport of volatile precursors contributed PFCAs in remote areas. They also detected PFBA as a dominant short-chained PFAS in freshwater, suggesting replacement of longer chain PFAS. Importantly, meltwater from snow and ice during Arctic Spring contributes to PFAS mobilization, which could intensify under climate warming. Hartz *et al.*,[150] investigated 36 PFAS in surface snow around Spitsbergen (Arctic) from January–August 2019. The authors found very high diversity and concentrations, particularly in locations with local emissions (*e.g.*, Longyearbyen), where PFAS in snow were up to 54× higher than at remote high-elevation ice cap sites. Deposition fluxes of C_2–C_{11} PFCAs, PFOS, and HFPO-DA (GenX) were strongly correlated with daylight (solar flux), suggesting photochemical formation from volatile precursors is seasonally driven. Lin *et al.*,[151] studied 9 PFAS in a sediment core (1945–2014) + 29 surface sediment sites from the Bering Sea to the western Arctic. Surface sediment concentrations ranged from 0.06 to 1.73 ng per g dry weight, dominated by PFOS, PFNA, and PFBS. Using positive matrix factorization, the authors attributed 45% of PFAS in the core to atmospheric oxidation of precursor chemicals, ~43% to oceanic transport of PFNA-related sources, and ~30% current/local input from firefighting foam (PFOS, PFBS). Their data suggest sediments act as both sinks and secondary sources, potentially making PFAS available to benthic food webs.

PFAS in Arctic Terrestrial and Marine have also been documented. A recent study measured 20 PFAS in soil, plants, sediment, and benthic marine organisms from Svalbard.[152] Concentrations ranged: soil: 0.12–4.84 ng per g dw, sediment: 0.15–0.93 ng per g dw, plant tissue: 0.11–16.6 ng per g dw, benthic marine biota: 0.049–26.2 ng per g dw. PFCAs dominated in most matrices except for amphipods, where PFOS accounted for 80% of PFAS. Plant species like mountain avens had biota-soil bioaccumulation factors (BSAF) 12.1 ($\sum$PFAS), amphipods 44.9 – indicating significant uptake. Their findings support that atmospheric precursors are major PFAS sources, but also show evidence for trophic magnification of PFOS and other sulfonates in benthic food webs. Cross-cutting review by Lohmann *et al.*,[9] summarizes PFAS exposure and effects across Arctic wildlife and humans. PFOS and PFNA dominate in wildlife (polar bears, seals, seabirds), and time-trend data show rising PFAS levels in recent decades. Modeled and observed PFAS levels in polar bear and ringed seal livers exceed thresholds linked with immunotoxicity or endocrine disruption in lab species; humans in Arctic communities (who rely on marine mammal diets) may also face elevated risk. The authors argue for more non-target screening, use of sum-parameter metrics, and long-term monitoring to clarify sources, exposure routes, and toxicological risks in the Arctic.

6 Summary, research gaps, and recommendations

PFAS are synthetic chemicals widely used in industrial and consumer products. Their strong carbon–fluorine (C–F) bonds confer resistance to thermal, chemical, and biological degradation, driving persistence in environmental matrices. Long-chain PFAS, such as PFOA and PFOS, preferentially adsorb to soil and sediment, whereas short-chain PFAS remain more soluble in water. The ecological risk posed by PFAS is largely associated with their tendency to bioaccumulate within organisms, induces oxidative stress, leading to cellular and organ damage. Evidence suggests that differences in organic anion transporter (OAT) activity in kidneys influence the recirculation and excretion of PFAS – low systemic concentrations may be retained and recirculated, while higher concentrations are more efficiently excreted. Consequently, PFAS accumulation patterns can vary between species and may not always lead to consistent increases in whole-body concentrations at higher trophic levels. In humans, PFAS exposure is associated with increased cholesterol, thyroid dysfunction, and liver injury, with pregnant women and infants being particularly vulnerable. PFAS predominantly accumulate in blood and liver, binding to proteins more than fats. Across Africa, recent studies have detected PFAS in water, sediments, and fish, with concentrations often exceeding international safety thresholds, signaling ecological and human health concerns. Most studies, however, have focused on legacy PFAS, with limited temporal and spatial coverage, and few directly assess human exposure or health outcomes. In Asia, both legacy and emerging PFAS are widespread in wildlife, with compounds such as F-53B and HFPO-DA/GenX highlighting a shifting exposure profile.

Future research should prioritize expanding environmental surveillance to include both legacy PFAS and emerging

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substitutes – such as GenX and perfluorinated betaines (PFBs) – with particular emphasis on African regions where regulatory oversight and monitoring remain limited. Moreso, Nigeria is scantly represented going by share size and massive oil and mineral exploitation inherent in its Niger Delta basin. Rapid growth of Nigeria industrial sector requires that adequate attention should be paid to both legacy and emerging PFAS contaminants, as a way of early prevention. Strengthening regional laboratory capacity is also essential to improve analytical coverage and ensure reliable detection across diverse environmental matrices. In addition, advancing mechanistic toxicology research across a broader range of PFAS structures is critical for understanding potential health effects, especially given the limited toxicity data currently available. Complementary use of non-targeted analytical techniques, including total organofluorine (TOF) and extractable organofluorine (EOF) measurements, will help quantify the large fraction of unidentified PFAS that escape conventional targeted methods. Improving species-specific toxicokinetic models; particularly those capturing interspecies differences in absorption, distribution, metabolism, and excretion, will be key for refining ecological and human health risk assessments. Finally, as a regulatory control, policy makers especially in the 3rd world countries like Nigeria with inadequate technology should ensure emerging industries with potential release of legacy and emerging PFAS contaminants into the environment, should include monitoring and preventive mechanism as condition for approval.

Author contributions

The study conception and design were performed by John Kanayochukwu Nduka. Main writer is Chideraa Courage Offor. Literature search, material preparations, and data analysis were performed by Chideraa Courage Offor, Chioma Juliet Alluwo and Ogechi Irene Eboh-Ajoku. The first draft of the manuscript was written by Ogechi Irene Eboh-Ajoku and Chideraa Courage Offor while John Kanayochukwu Nduka revised and edited it.

Conflicts of interest

The authors have no relevant financial or non-financial interests to disclose.

Data availability

No new data was created or analyzed during this study. Data sharing does not apply to this article.

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