REVIEW ARTICLE

Occupational exposure to per- and polyfluoroalkyl substances: a scope review of the literature from 1980–2021

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BACKGROUND: Per- and polyfluoroalkyl substances (PFAS) comprise a large group of chemicals that have been integrated into a wide variety of industrial processes and consumer products since the 1950s. Due to their profuse usage and high persistence in human serum, understanding workplace exposures to PFAS is critical.
OBJECTIVE: We aimed to characterize the PFAS exposure profiles of relevant occupational populations, elucidate trends in the PFAS exposure characterization process, and identify major research gaps that remain within the occupational PFAS exposure literature.
METHODS: A systematic search of four literature databases for peer-reviewed articles published between 1980 and 2021 on PFAS exposure in occupational settings was conducted.
RESULTS: Of the 2574 articles identified, 92 met the inclusion criteria. Fluorochemical workers were the target population in most early exposure assessment research; however, studies conducted within the last 10 years have evaluated a wider range of occupational populations and settings. The highest exposures were reported in fluorochemical workers, but, in comparison to reference populations, one or more PFAS were elevated in most workers and in most workplaces that were assessed. PFAS was most frequently assessed in worker serum using a discrete analytical panel of PFAS, with earlier studies restricted to a few long-alkyl chain PFAS while more recent studies have included more expansive panels due to more robust methods.
SIGNIFICANCE: Characterization of occupational exposure to PFAS is limited but expanding. Current analytical methods are not robust enough to fully capture the potential range of PFAS present across different workers and workplaces. While exposures to PFAS for certain occupational groups have been studied in detail, exposure information for other occupational groups with high potential for exposure are limited. This review highlights substantial findings and major research gaps within the occupational literature.

Keywords: PFAS; Inhalation exposure; Dermal exposure; Workplace exposure; Environmental monitoring; Biomonitoring

Journal of Exposure Science & Environmental Epidemiology (2023) 33:673–686; https://doi.org/10.1038/s41370-023-00536-y

INTRODUCTION

Per- and polyfluoroalkyl substances (PFAS) comprise over 12,000 compounds [1]. Because of their widespread use as industrial processing aids and in consumer products, and their persistence in environmental matrices (e.g., water and food) and the human body following exposure, PFAS have been detected in over 98% of the US population [2, 3]. Researchers who conduct in vitro, in vivo, and epidemiological studies have reported associations between PFAS exposure and a range of dose-dependent adverse effects, including changes in lipid and glucose metabolism; changes in thyroid hormone levels; immunomodulatory effects; and increased tumorigenesis [4]. Workers that produce, integrate into production, or handle PFAS in high quantities may experience higher exposure compared to other workers and the general population, and thus greater risks of dose-dependent effects from PFAS exposure.

Currently, recommended occupational exposure limits (OELs), which are established to protect workers from health risks associated with workplace exposures, exist for three PFAS in the US and four PFAS globally (Table S1). In the absence of recommended OELs for most PFAS and regulatory OELs for all PFAS, U.S. workers are often reliant on the broader protections afforded under the Occupational Safety and Health Administration's General Duty Clause [5, 6]. While there are no Biological Exposure Indices for PFAS, in 2022 the National Academies of Sciences, Engineering, and Medicine published Guidance on PFAS Testing and Health Outcomes that includes recommendations for exposure reduction and clinical follow-up using serum-based thresholds [7].

Unlike most persistent organic chemicals, PFAS partition preferentially to serum proteins rather than fatty tissue. Perfluoroalkyl substances, which are defined by having fully fluorinated

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Received: 7 October 2022 Revised: 13 March 2023 Accepted: 13 March 2023
Published online: 28 March 2023

carbon chains and are commonly referred to as terminal PFAS—such as the perfluorinated carboxylic acids (PFCAs), perfluorobutanoic acid (PFBA), perfluorohexanoic acid (PFHxA), and perfluorooctanoic acid (PFOA); and perfluorinated sulfonic acids (PFSAs), perfluorobutane sulfonic acid (PFBS), perfluorohexane sulfonic acid (PFHxS), and perfluorooctane sulfonic acid (PFOS) [8]—do not metabolize in the body and bind efficiently to serum proteins [9]. Polyfluoroalkyl substance, which are defined by having partially fluorinated carbon chains and are commonly referred to as precursor PFAS—such as fluorotelomer alcohols (FTOHs) and perfluoroalkane sulfonamides—readily metabolize into terminal PFAS in the body [8]. Terminal PFCAs and PFSAs can be further categorized as "long-alkyl chain" or "short-alkyl chain" PFAS based on their carbon chain length and functional group attachment. In general, PFSAs bind more efficiently to serum than PFCAs of equal carbon chain length due to their sulfonation group attachment. Therefore, while PFCAs containing a carbon chain consisting of at least 8 carbons are considered "long-alkyl chain", PFSAs only require a carbon chain consisting of 6 or more carbons to meet the "long-alkyl chain" definition. Elimination half-lives of PFAS vary from days to months for short-alkyl chain PFAS to several years for long-alkyl chain PFAS (Table S2); however, the precise distribution and elimination pathways for short- and long-alkyl chain PFAS are not sufficiently understood [9].

Unlike community settings, where diet and contaminated drinking water are considered the major drivers of PFAS exposure, occupational exposures are generally attributed to inhalation of PFAS through aerosols and vapors or ingestion of PFAS in dust [10, 11]. In certain occupational settings, absorption of PFAS through the skin may also contribute to exposure [12–14]; however, dermal exposure data for PFAS are limited.

Fluorochemical producers in the United States began voluntarily phasing out long-alkyl chain PFAS, such as PFOA and PFOS, from production in 2000 [15]. This shift in production led to emission reductions of PFOA and PFOS down to less than 10% of 2001 baseline levels by 2015 [16]. While not proportional to emission reductions, serum PFOA and PFOS concentrations in the general US population have also steadily diminished [17]; however, the impacts of voluntary phaseout efforts on environmental and serum levels of short-alkyl chain PFAS and other alternatives are less clear. Regulations and guidance initiatives at the federal and state level have further contributed to a changing pattern in PFAS usage by US manufacturers. As industries have continued to substitute alternative PFAS that are perceived to be less persistent or harmful, the number of PFAS in use have grown exponentially. Due to resource constraints, including limitations in method capabilities and the timing of sample collection, background surveillance efforts for capturing the range of PFAS present in the environment and in the general population are currently impractical. However, for certain occupational populations, where a variety of PFAS are routinely present in the workplace, capturing a wide range of PFAS across different biological and environmental matrices is more feasible.

The objective of this review was to characterize the occupational PFAS exposure literature, including sample collection and analytical methods employed in occupational settings, PFAS exposure profiles of workers by industry over time, and potential determinants of exposure in different industries. Critical gaps in the occupational exposure literature were also defined and suggestions provided for future occupational exposure assessment and epidemiological studies.

MATERIALS AND METHODS

We performed a targeted review of the peer-reviewed literature for manuscripts published between 1980–2021 on occupational exposures to PFAS. Using search terms selected by the authors, articles were extracted by the CDC Thacker Library on September 8th, 2021 from Medline, Scopus, Embase, and Environmental Science Collection (Table S3). Extracted citations were managed using a citation manager software (Clarivate EndNote 20). An industrial hygienist manually reviewed the full results of the literature search using the exclusion criteria outlined in Fig. 1 by screening article titles, abstracts, and keywords. A second industrial hygienist also manually reviewed the articles that met the criteria for this literature review and provided input to fill-in the gaps where insufficient data extraction was observed.

We defined PFAS as all aliphatic substances that contain a fluorinated carbon chain ($n \geq 2$) [18]. Studies published in non-peer-reviewed sources, such as company reports and trade journals, and articles not published in English were excluded from this review. To characterize the literature documenting and describing exposures in occupational settings, we excluded studies that did not include a quantitative, semi-quantitative, or qualitative assessment of PFAS exposure in the workplace. We defined occupational settings as environments where individuals perform work-related tasks; however, we excluded residential settings from this definition. Additionally, studies that only collected environmental matrices with indirect application to occupational exposures such as wastewater, drinking water, and soil were excluded from this review.

Literature review results were summarized descriptively by occupational population, PFAS analyzed, collection method, sampling and analytical methodologies, data analysis approach, study design, publication year, sample collection year, and study findings. These topics were established during the initial review process because of their relevance toward the focus of this review. Additionally, several non-occupational studies were referenced to place existing exposure results from occupational studies into perspective.

RESULTS

The literature search returned 2574 peer-reviewed articles, of which, 91 met the review criteria (Fig. 1). In reviewing the references within individual articles, we identified 1 additional article that deserved inclusion in this review paper ($n = 92$). While this literature review includes articles published in the 1980s and 1990s, ~90% of the included literature was published after 1999 (Fig. S1).

Most studies measured PFAS in blood, but the proportion of studies that measured blood, air, dust, or urine differed by industry (Table S4). Long-alkyl chain PFAS, specifically PFOA and PFOS, were the most frequently investigated PFAS; however, studies conducted within the last 15 years have increasingly measured short-alkyl chain, precursor, and alternative PFAS (Fig. 2).

Air monitoring studies frequently measured a more diverse panel of PFAS than biomonitoring exposure studies, often including measurements of both terminal and precursor PFAS (Table 1a, b). Commonly used long-alkyl chain substitutes, chlorinated polyfluorinated polyether sulfonic acids or polyfluoroalkyl phosphoric acid diesters, were also measured in a small proportion of studies (Table 1a).

While most articles captured PFAS using solely quantitative sampling approaches (75%), some studies relied on purely qualitative (~4%) or semi-quantitative approaches (~21%) to approximate PFAS exposure (Table S4). Purely qualitative methods based on industrial hygiene expertise and worker history records were utilized in a small proportion of fluorochemical production studies (3 of 41) and one case study involving a plastic machinist worker [19]. Semi-quantitative approaches combining quantitative (partial serum data, annual usage data, annual emission data, person-years of exposure) and qualitative data (worker history records and IH expertise) were used in several fluorochemical production studies (17 of 41) and one study of ski wax technicians.

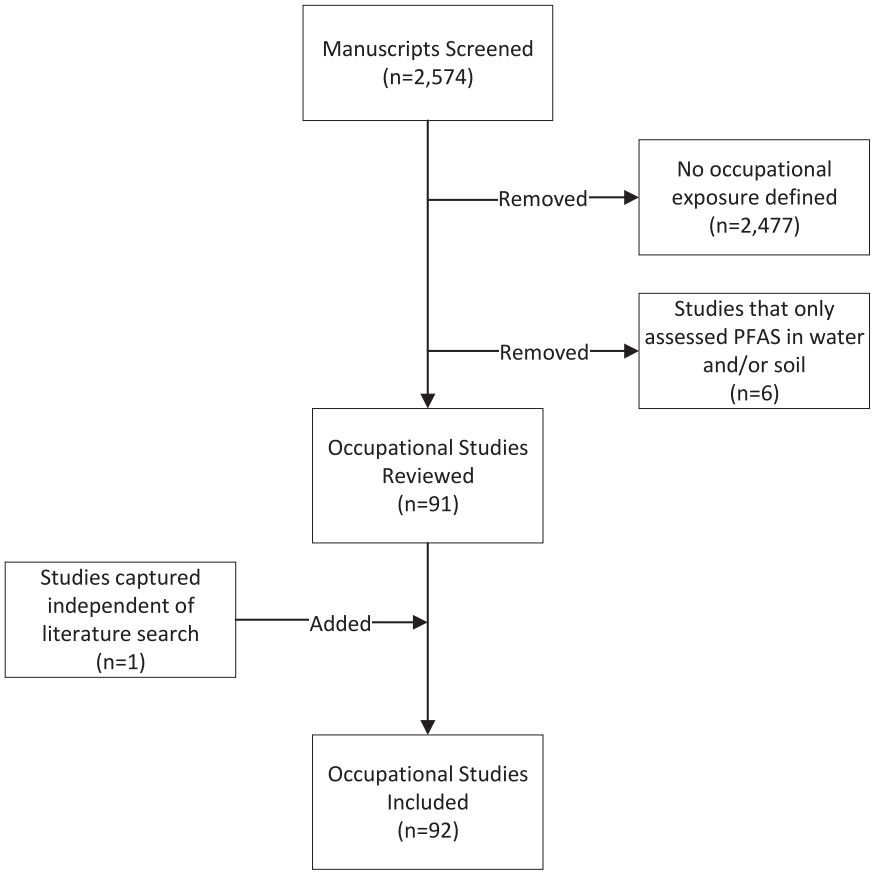


Fig. 1 Flow diagram of articles captured from literature search and selected for literature review.

Most articles (~60%) evaluated PFAS exposure in fluorochemical production workers or first responders. Most of the fluorochemical production studies were conducted at ammonium perfluorooctanoate or PFOS production facilities, but separate studies evaluated exposure at a perfluorononanoic acid production facility and a polytetrafluoroethylene production facility [20, 21]. Studies of firefighters included municipal, airport, and volunteer firefighter populations from a range of geographic locations and firefighting environments (e.g., fire response, training, and fire stations). All studies of ski wax technicians were conducted in Northern Europe. Among the textile manufacturing research, two out of three studies specified the types of materials produced, including a facility that produces turnout gear for firefighters and a facility that produced a range of heavyweight and lightweight garments [22, 23]. Other manufacturing environments that were represented in this review include: a metal plating workshop, a powder coating shop, a metalworking shop, a plastic production facility, and a pesticide packing plant (Table S5 provides information about specific manufacturing industries associated with PFAS exposure).

A small, but substantial fraction of studies evaluated PFAS exposure in non-manufacturing settings. Multiple studies specified the evaluation of PFAS levels at a store(s) selling outdoor wear while the remaining clothing retail studies only specified the selling of textiles or clothing. Details pertaining to the types of office spaces evaluated were not consistently provided across studies evaluating PFAS exposure in office settings. Jahnke et al. and Heydebreck et al. specified that the presence of textiles may have impacted the exposure profile of sampled office areas [22, 24]; whereas, Besis et al. suggested that the volume of paper products may impact PFAS exposure at a newspaper office [25]. Additional non-manufacturing workplace environments that were represented in this review include: college lecture halls, school laboratories, computer rooms, primary/secondary classrooms, furniture shops, printing shops, autobody shops a mechanical shop, an electrotechnical shop, carpet shops, a car selling store, electronic stores, a sports equipment shop, coffee shops, internet cafes, restaurants, libraries, movie theaters, and hotels. Multiple studies also evaluated PFAS exposure in fishermen.

Sample collection methodology

Over the last several decades, researchers have quantified PFAS in workers' biological matrices (serum, blood, plasma, urine, seminal fluid) and at worksites across multiple environmental matrices (air, dust, aerosols, surfaces) utilizing a variety of sampling methods (Tables S6 and S7).

Researchers have predominately measured PFAS in serum rather than other biological matrices (Table S4) [26–28]. Medical surveillance programs conducted at fluorochemical facilities prior to the 1980s measured PFAS levels in whole blood [29]; however, independent of early surveillance efforts and ski wax technician studies, no occupational studies have measured PFAS in whole blood. Of the non-serum biological matrices that have been evaluated, researchers have most frequently measured PFAS levels in urine [26, 27, 30]. Research efforts associated with measuring PFAS levels in urine have primarily focused on understanding the partitioning, distribution, or elimination characteristics of specific PFAS rather than the magnitude of exposure. Separate studies have also investigated PFAS levels in plasma or seminal fluid [31, 32].

Outside of conducting biological sampling to evaluate PFAS exposure in workers, researchers have most frequently measured PFAS in workplace air to assess exposure in workers. Methods for assessing PFAS levels in workplace air have varied by method

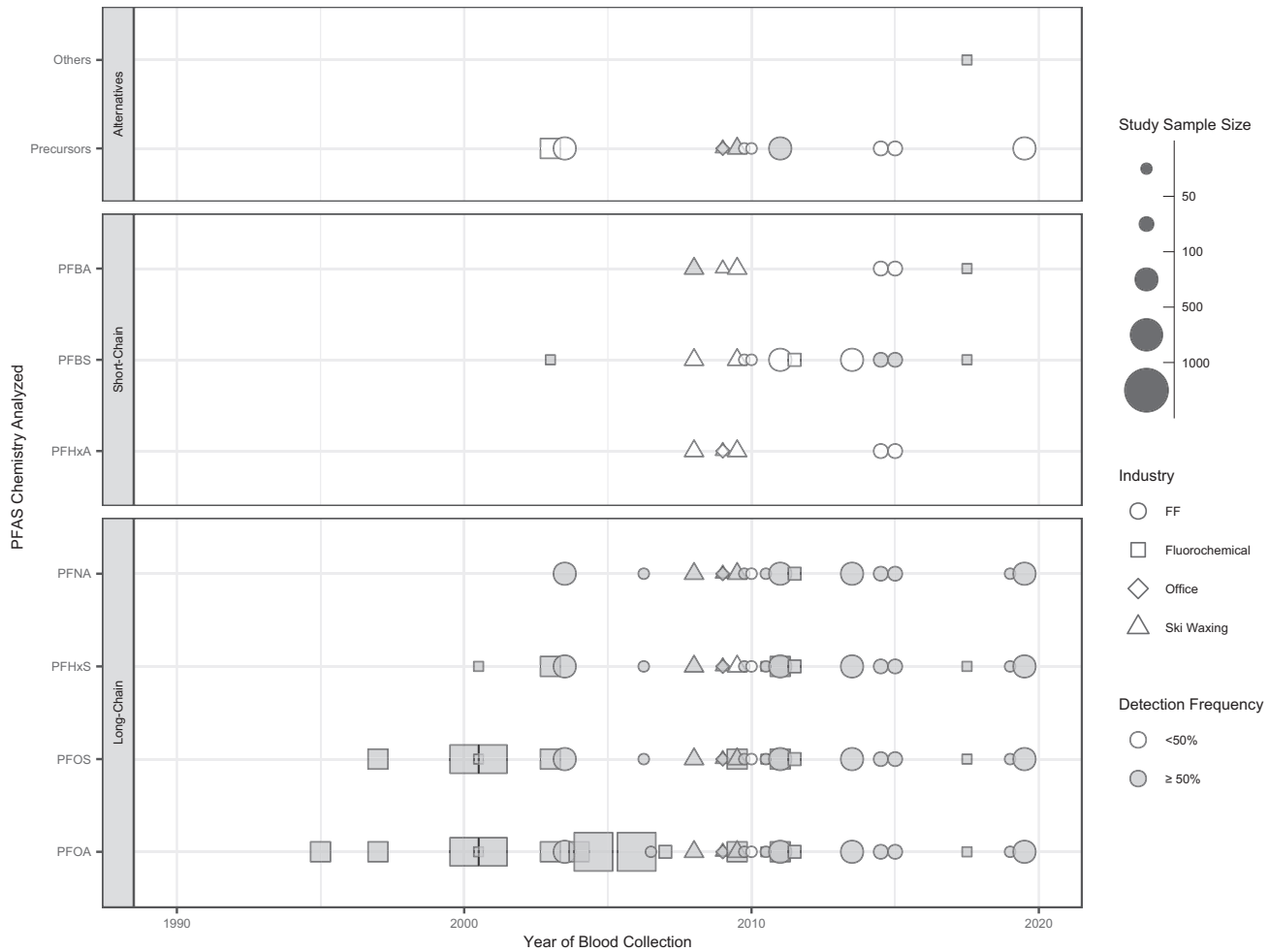


Fig. 2 Bubble plot of PFAS chemistries analyzed in worker blood across individual studies that sampled whole blood, plasma, or serum. Sample data are stratified by year of collection (midpoint), industry type (shape), sample size (shape size), and detection frequency (shaded gray).

constraints and by workplace characteristics, which dictate what PFAS are present and the gas-particle partitioning behavior of PFAS. Several studies have employed methods capable of detecting both the aerosol and gas-phase fractions simultaneously to best approximate total air concentrations within the work environment. These studies have often employed multi-layered instruments capable of capturing the desired aerosol fraction while simultaneously collecting gas-phase PFAS [22, 24, 33–36]. Methods employed by researchers to collect both phases simultaneously include the Occupational Safety and Health Versatile Sampler (quartz fiber filter + XAD-2 sorbent), an ISOLUTE + sorbent cartridge with a total aerosol membrane filter, and a glass fiber filter with sorbent-impregnated polyurethane foam [35, 37–39].

For collecting PFAS-laden aerosols in occupational settings, researchers have deployed various instruments, including cyclones, open-faced cassettes, and particle impactors. Glass fiber or quartz fiber material have frequently been used as filter medium for the entrapment of aerosol particles. Studies of ski waxing tasks have employed multiple aerosol collection instruments to determine the relative abundance of PFAS across multiple aerosol size fractions during ski waxing operations [35, 40, 41]. In several studies, this data was collected alongside direct reading instrumentation to obtain time-integrated data on aerosol concentrations across multiple size bins [40–44]. Few studies captured the gas-phase exclusively. Studies that captured the vapor or gas-phases separate from the particulate phases have employed low-volume samplers such as charcoal tubes or midget impingers [41, 43, 45]. Studies that captured precursor PFAS have often utilized sorbent-impregnated polyurethane foam with XAD-2 or XAD-4 resin or Isolute ENV+ solid-phase extraction cartridges [37, 46–48]. Yao et al. deployed a solid-phase extraction cartridge that included both Isolute ENV+ and weak anion exchange sorbent to capture the precursor and terminal PFAS in the gas-phase simultaneously [49].

Few studies have assessed PFAS exposure in the breathing zone of workers [35, 40, 43, 45, 50]. For most manufacturing settings, large scale area sampling was conducted near processes and worker tasks anticipated to be associated with elevated PFAS levels. In non-manufacturing settings, where the sources of exposure are harder to define, researchers have typically collected a small number of samples. In contrast to non-occupationally focused research efforts, which have frequently deployed passive monitors to measure PFAS levels in the air, only one study employed passive monitoring to evaluate PFAS levels in an occupational setting [37]. Concerns with how efficiently passive monitors capture different classes of PFAS, especially when PFAS levels are high (low sorption capacity) and their performance under atypical sampling conditions such as elevated relative humidity and short sampling-time duration have likely contributed toward decisions to utilize active monitoring instead.

Table 1. Summary of median serum and air PFAS levels stratified by industry.

PFAS groups	PFAS	<LOD	LOD–<1	1–10	11–100	101–1000	1001–10,000	>10,000
a. Summary of median serum PFAS levels in workers stratified by industry (ng/ml)								
PFCAs	PFOA		W (84[b])	F (32[d], 70–73, 74[a], 75[b], 76[b], 77, 79), O (85, 106[b]), OF (38[b], 74[a]), T (85[a])	F (78), P (65[a]), S (44), W (83)	P (26, 27, 58[a], 61[b], 65[a], 69), S (42[c], 80[c])	P (56[a], 58[a], 59[a], 60, 62, 66)	P (60)
	PFNA	P (68)	F (32[d], 70, 73, 74[a], 76[b], 77), O (31, 85), OF (74[a]), W (84[b])	F (71–72, 78, 79), OF (38[b]), P (66), T (85[a]), W (83)	S (42[c], 81[c])			
	PFHxA	F (73, 74[a], 78, 81[c]), OF (38[b]), P (68), S (44, 81[c]), W (83)	O (31), S (42[c], 44)					
	PFBA	F (73, 74[a]), S (44)	S (42[c], 44, 81[c])	W (83)	P (68)			
	Other	F (72, 73, 75[b], 76[b], 77, 78), O (31), OF (38[b]), P (68), S (81[c]), W (83)	F (71–73, 76[b], 77), O (85), OF (38[b]), P (66), S (42[c], 44), T (85[a]) W (83, 84)	F (71), P (66), S (42[c], 81[c]), W (83)	W (83)			
PFSAs	PFOS		O (31)	F (70, 71, 74[a], 76[b]), O (31, 85), OF (38[b], 74[a]), T (85[a])	F (32[d], 72, 75[b], 77–79), O (106[b]), P (65[a], 66), S (42[c], 44, 81[c]), W (84[b])	P (58[a], 59[a], 61[b], 65[a], 68)	P (26–27, 55[a], 58[a], 59[a])	W (83)
	PFHxS		O (31, 85), P (66), T (85[a]), W (84[b])	F (32[d], 70–73, 74[a], 75[b], 76[b], 78–79), OF (38[b, 74[a]), S (42[c], 44, 81[c])	F (77)	P (26, 27, 61[b], 63, 67), W (83)		
	PFBS	F (75[b], 77), S (81[c])	F (73, 74[a]), OF (74[a]), P (66), S (42[c])		P (67), W (83)	P (85)		
	Other	F (72, 78), P (69), S (42[c])	S (42[c])					
Precursor PFAS		F (73, 74[a], 75[b], 76[b]), OF (38[b]), P (68), S (42[c], 44)	F (72, 75[b]), S (42[c])	S (42[c]), W (83)	P (61[b])			
Other PFAS		S (42[c])		P (68[a])	M (30[a])	M (30[a])		

PFAS groups	PFAS	<LOD	LOD–<1	1–10	11–100	101–1000	1001–10,000	10,001–100,000	>10,000
b. Summary of median air PFAS levels in work environments stratified by industry (ng/m^3)									
PFCAs	PFOA		P (26)		S (44)	S (35[a], 50)	P (39[e]), T (22)	P (39[e]), S (35[a])	S (35[a])
	PFNA				S (35[a], 50)	T (22)	S (35[a])		
	PFHxA						S (35[a], 50), T (22)	S (35[a])	
	PFBA					T (22)			
	Other	T (22)			S (44, 50), T (22)	S (50), T (22)			
PFSAs	PFOS	S (44), T (22)	P (26)						
	PFHxS	S (44)	P (26)						
	PFBS								
	Other				S (35[a])	S (35[a])	S (35[a])	S (35[a])	
Precursor PFAS	FTOHs		O (46[e], 47[a])	C (47[a], 48), M (37[e]), O (34[a], 37[a]), OF (24[e], 46[a], 47[a]), R (49)	O (37[a], 46[e]), OF (24[e], 37[e], 38), R (46[a], 49)	O (37[a]), R (37[a], 46[a])		S (50), T (22)	S (35[a])
	FOSEs	C (48)	O (33[e], 34[a]), OF (38)	O (33[e]), OF (24[e], 37[e])					
	Other	OF (24[e])	C (48), M (37[e]), O (37[e]), OF (24[e], 34[a], 38), R (49)	C (48), M (37[e]), O (37[e]), OF (37)	O (37[a]), R (37[a])	O (37[e])			

F firefighters and first responders, *FS* Fire stations, *M* metal plating, *OF* office, *P* fluorochemical production, *S* ski waxing, *T* textile manufacturing, *W* wranglers/fishermen, *C* classroom/lectures/school, *H* hotels, *O* other.

[a]Reported mean value only.

[b]Reported geometric mean value only.

[c]Whole blood analyzed (whole blood values multiplied by 1.8).

[d]Plasma fraction analyzed.

[e]Only one sample collected.

Surface dust levels have predominately been measured in work environments where the potential for rapid volatilization of PFAS-laden materials is low (e.g., absence of a point source of heat, anionic or deprotonated form); however, researchers have also assessed surface dust levels in manufacturing settings, where resuspended dust concentrations may contribute toward inhalation exposure [22, 51]. Most commonly, researchers have captured PFAS surface dust using a vacuum with an attachment containing sorbent media for PFAS [36]; however, pre-cleaned brushes, air conditioning filters, and glass fiber filters have also been utilized to collect dust in occupational settings [22, 25, 26].

Currently, no occupational exposure studies have directly measured dermal PFAS levels (e.g., wipe samples of skin); however, two separate studies measured PFAS exposure near the skin of workers by quantifying PFAS levels on worker uniforms [13, 52].

Analytical methods

Analytical techniques utilized to measure PFAS have varied by year of sample collection and type of PFAS analyzed (e.g., precursor vs. terminal). Studies utilizing targeted approaches mostly relied on liquid chromatography-mass spectrometry to analyze semi-volatile (e.g., short- and long-alkyl chain) PFAS and gas chromatography-mass spectrometry to analyze volatile (e.g., precursor) PFAS (Table S4). Several studies also employed semi- or non-targeted analytical methods such as the total oxidizable precursor assay and particle-induced gamma emission spectroscopy to more comprehensively capture the fluorinated compounds present in a workplace. Analyte extraction methods also differed based on instrument availability or analytes of interest as different extraction methods are better at capturing different PFAS chemistries (e.g., base vs. methanol).

Quantitative assessment of workplace exposures

While qualitative and semi-quantitative approaches are useful proxies for assessing exposure risk, particularly in large worker cohorts with a diverse range of worker job titles and tasks, purely quantitative approaches that employ environmental or biological sampling offer a greater level of specificity and detail, when feasible.

Trends in biological PFAS exposure across industries. The magnitude of PFAS in worker blood have varied by occupation and collection period. Fluorochemical workers have consistently exhibited the highest blood concentrations of PFOA and PFOS compared to other occupational groups, overexposed community populations, and background populations (Figs. 3 and 4). Irrespective of occupation or collection period, the detection frequencies of long-alkyl chain PFAS, PFOA, PFOS, PFHxS, and PFNA, in the blood of workers were consistently between 65–100% apart from two studies; whereas, for short-alkyl chain PFAS, PFBS, PFBA, and PFHxA, detection frequencies varied considerably by occupation and collection period. Prior to voluntary phaseout efforts, PFOA and PFOS were frequently reported at parts per million (ppm) levels in fluorochemical workers [45, 53–59]. Woskie et al. estimated that PFOA emissions peaked in 2001 then decreased steadily after phaseout efforts began [29]. Even before long-alkyl chain PFAS production and usage peaked, several studies reported declining serum levels in fluorochemical production workers [45, 55, 60–63]. Several researchers suggested that pre-phaseout reductions resulted, in partial, from an increased emphasis on exposure reduction practices [21, 29, 39].

Because few researchers collected PFAS exposure biomarker data for non-fluorochemical workers prior to the late 2000s, it is difficult to determine how phaseout efforts have affected serum levels in non-fluorochemical workers. Serum levels have declined over time in some firefighters; however, their exposure profiles are generally within an order of magnitude of reference populations making it increasingly difficult to determine whether observed decreases in serum levels are attributable to reductions in occupational or community exposure sources (Table 1a). For other occupational groups, biological PFAS exposure data is too limited to evaluate temporal trends in exposure intensity; however, analytical efforts have grown increasingly robust with studies conducted in the last 15 years more frequently measuring a wider panel of PFAS, including short-alkyl chain PFAS, precursor PFAS, and alternative PFAS such as Cl-PFESAs (Fig. 2).

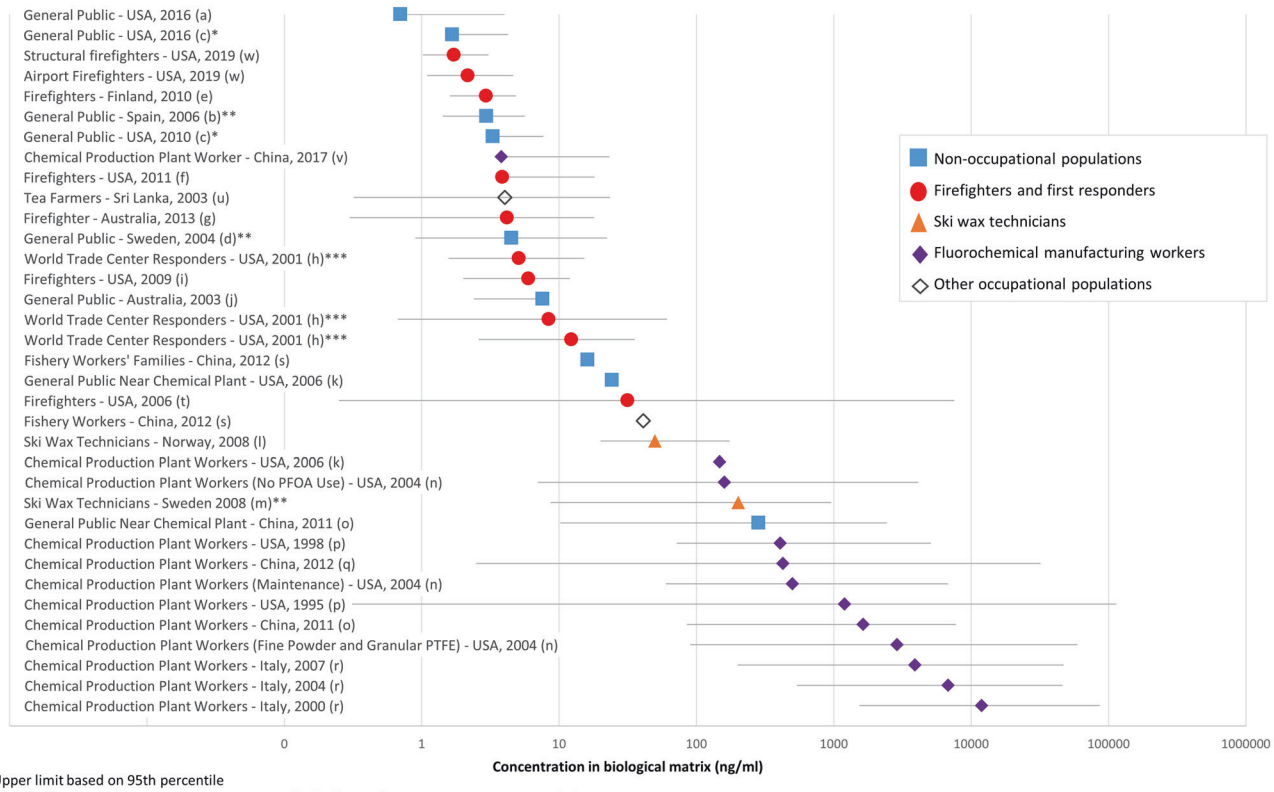
Biomonitoring—fluorochemical workers. At the plant level, job title, department, and assignment have been cited as determinants of serum PFAS levels in fluorochemical workers. Multiple studies reported substantial variation in serum PFAS across job titles. Olsen et al. reported that among fluorochemical workers cell and chemical operators exhibited the highest serum levels of PFOA, PFOS and PFHxS followed by waste operators and maintenance workers [61]. Results from Fu et al. indicated that individuals working in the electrochemical fluorination department for an extended period (>6 months) exhibited elevated PFOA levels; whereas individuals working in the sulfonation department exhibited elevated PFOS levels [27]. Only a subset of fluorochemical worker studies evaluated the influence of different worker tasks on serum PFAS levels. Of the studies that have, grinding and refining PFAS-laden materials, measuring powdered or granulated PFAS, drying PFAS-infused slurry, and decommissioning a fluorochemical facility were linked to elevated PFAS exposure [29, 45, 64, 65]. Consistently higher serum PFAS levels were reported in fluorochemical workers when compared to nearby residents and other types of workers [66–69].

Biomonitoring—firefighters. First responders have frequently experienced elevated serum PFAS levels in comparison to reference populations, but not as consistently or to the same extent as fluorochemical workers. With research spanning three continents, PFOS, PFHxS, and perfluorononanoic acid have been the most consistently elevated PFAS chemistries in firefighters; however, substantial variability in the exposure profiles of firefighters have been reported across firefighter studies.

Firefighter type (e.g., airport, municipal, etc.) is an important predictor of PFAS serum levels among firefighters. Leary et al. reported that serum PFOS and PFHxS levels were approximately two times as high in aircraft rescue firefighting firefighters as suburban firefighters [70]. Across five separate municipal, career firefighter cohorts, serum PFOS varied by sex (with concentrations in females lower than males) [71–74], and while serum PFOS levels from two all-male municipal cohorts were comparable to levels reported previously for the aircraft rescue firefighter cohort, serum PFHxS levels across all five cohorts were consistently well below levels reported for the aircraft rescue firefighter cohort [70–75]. Across the career cohorts, serum PFOS was consistently higher than serum PFOS reported in a cohort study of volunteer firefighters [76].

Beyond firefighter type, several determinants of PFAS exposure have been identified for first responders. Employment duration [77], aqueous film forming foam (AFFF) usage [70, 72, 73, 78, 79], routine handling of contaminated PPE [72], routine handling of hazardous materials [72], and conducting overhaul procedures during response efforts [73] were positively associated with PFAS serum levels in firefighters. Previously unidentified PFESAs were captured utilizing quadrupole-time-of-flight mass spectrometry in the serum of firefighters with prior AFFF exposure [80]. Among a cohort of World Trade Center collapse first responders, smoke inhalation and dust exposure (qualitatively defined) were positively associated with plasma PFOA and PFHxS levels [32].

Biomonitoring—ski wax technicians. While levels of PFSAs in the blood of ski wax technicians mostly approximate background



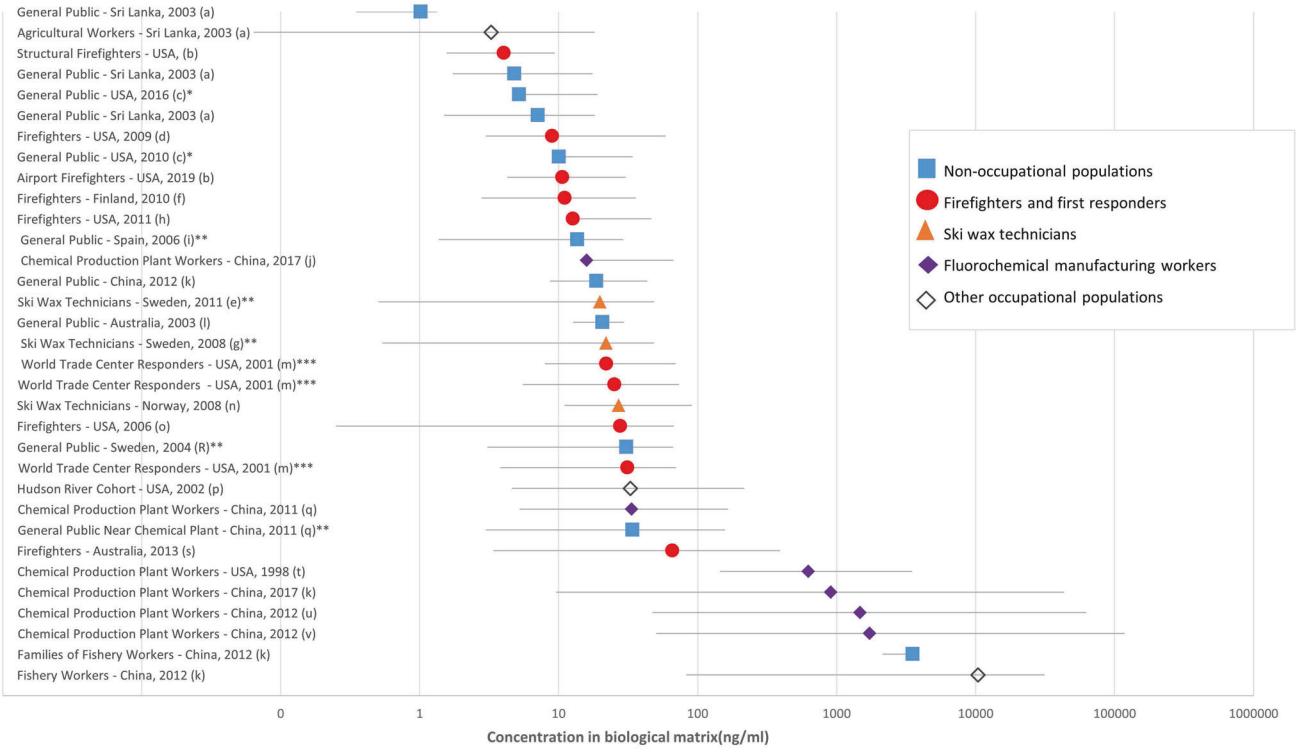
*Upper limit based on 95th percentile
**Whole blood sample. Serum concentration multipled by 1.8 for comparison to serum and plasma
***Plasma sample
(a) Kato et al., 2018[102]; (b) Ericson et al., 2007[103]; (c) CDC, 2019[17]; (d) Karrman et al., 2006b[105]; (e) Laitinen et al., 2014[79]; (f) Dobraca et al., 2015[72]; (g) Rotander et al., 2015[77]; (h) Tao et al., 2008[32]; (i) Shaw et al., 2013[71]; (j) Karrman et al., 2006a[104]; (k) Steenland et al., 2009[101]; (l) Freberg et al., 2010[44]; (m) Nilsson et al., 2010[81]; (n) Woskie et al., 2012[29]; (o) Wang et al., 2012[66]; (p) Olsen et al., 2007[63]; (q) Fu et al., 2016[27]; (r) Costa et al., 2009[62]; (s) Zhou et al., 2014[83]; (t) Jin et al., 2011[78]; (u) Guruge et al., 2005[31]; (v) Lu et al., 2019[68]; (w) Leary et al., 2019[70]

Fig. 3 PFOA in serum, plasma, or whole blood* by population, geographic region, and year of most recent sample collection. Statistics include median, minimum, and maximum concentrations (ng/ml) [17, 27, 29, 31, 32, 44, 62, 63, 66, 68, 70–72, 77–79, 81, 83, 101–105].

levels, PFCA levels have consistently been elevated in comparison to reference populations. Freberg et al. reported that average serum levels of several PFCAs in a cohort of ski wax technicians were between 10–40 times higher than levels reported for the reference population [44]. This may be attributable to precursor PFAS such as FTOHs, which are frequently found in high quantities within the ski wax material utilized to condition performance skis [42, 81]. One study estimated that up to 45% of PFOA levels in the blood of ski wax technicians could be attributed to inhalation of 8:2 FTOH [82].

Biomonitoring—other occupational populations. Other occupational populations in which researchers have conducted biological sampling to assess exposure include office workers, fishermen, textile mill workers, barbers, and metal plating workers. Within a small office worker cohort, serum levels of PFOS, PFOA, PFHxS, and perfluorodecanoic acid were 2–3 times higher than levels reported in the background US population [38]. Air FTOHs accounted for ~36% of the variation in PFOA serum levels within the office worker cohort [38]; whereas, dust was not determined to be a significant predictor of serum PFOA within the cohort [36]. In a small metal plating cohort, workers exhibited elevated serum levels of Cl-PFESAs [30]. Individuals that had worked at the metal plating facility for more than a year exhibited median serum levels of 8:2 Cl-PFESA two orders of magnitude higher than the background Chinese population [30]. Within the same cohort, fishery employees located near the metal plating facility exhibited median serum levels of Cl-PFESAs an order of magnitude higher than the background population, but lower than the metal plating workers [30]. A separate study reported PFOS, PFHxS and PFOA in fishermen between 1–3 orders of magnitude higher than the background Chinese population [83]; whereas serum PFAS levels reported from a New York angler cohort study were only slightly elevated compared to the general US population [84]. All three of these studies indicated that dietary consumption of fish was the predominate pathway for exposure among fishermen; however, differences in exposure intensity were likely dependent on the extent of contamination of the body of water they fished [84]. Another study comparing serum PFAS levels in barbers and textile mill workers reported that textile mill workers exhibited serum levels of PFOS, PFOA, PFHxS, and perfluorodecanoic acid approximately two times as high as barbers [85].

Biomonitoring—urinary PFAS. While PFAS levels are frequently detectable in the urine of workers, in studies that have measured PFAS in both urine and serum, urine levels were consistently 3–4 orders of magnitude lower for long-alkyl chain PFAS and 1–2 orders of magnitude lower for short-alkyl chain PFAS [26–28, 83, 86]. Several studies have measured PFAS in urine to elucidate toxicokinetic differences between PFAS. Findings from these studies indicate that (1) short-alkyl chain PFAS are removed through the urinary excretion pathway 2–3 orders of magnitude faster than long-alkyl chain PFAS [83], (2) branched PFAS isomers are removed through the urine faster than their linear counterparts [26], and (3) renal clearance rates of long-alkyl chain PFAS are independent of the frequency and magnitude of exposure [27]. Fu et al. also reported that previous estimations of the elimination half-lives of long-alkyl chain PFAS likely underestimated renal clearance, which could have led to an overestimation of their half-lives [27].



*Upper limit based on 95th percentile
**Whole blood sample. Serum concentration multipled by 1.8 for comparison to serum and plasma
***Plasma sample
(a) Guruge et al., 2005[31]; (b) Leary et al., 2019[70]; (c) CDC, 2019[17]; (d) Shaw et al., 2013[71]; (e) Nilsson et al., 2013[42]; (f) Laitinen et al., 2014[79]; (g) Nilsson et al., 2010[81]; (h) Dobraca et al., 2015[72]; (i) Ericson et al., 2007[103]; (j) Lu et al., 2019[68]; (k) Zhou et al., 2014[83]; (l) Karrman et al., 2006a[104]; (m) Tao et al., 2008[32]; (n) Freberg et al., 2010[44]; (o) Jin et al., 2011[78]; (p) Tanner et al., 2018[106]; (q) Wang et al., 2012[66]; (r) Karrman et al., 2006b[105]; (s) Rotander et al., 2015[77]; (t) Olsen et al., 2007[63]; (u) Gao et al. 2015[26]; (v) Fu et al., 2016[27]

Fig. 4 PFOS in serum, plasma, or whole blood* by population, geographic region, and year of most recent test. Statistics include median, minimum, and maximum concentrations (ng/ml) [17, 26, 27, 31, 32, 42, 44, 63, 66, 68, 70–72, 77–79, 81, 83, 103–106].

Exposure pathway—workplace air. A relatively small fraction of selected studies (19/92) assessed levels of PFAS in the air. These studies investigated a range of occupational environments representing the manufacturing and service sectors. Air concentrations of PFAS reported from studies that evaluated manufacturing settings such as fluorochemical production, textile manufacturing, and ski waxing facilities were frequently higher than levels reported in non-manufacturing settings such as offices, retail shops, schools, and residential settings. However, large variations in reported PFAS levels were found across studies that investigated similar occupational environments (Table 1b). These differences can largely be explained by changes in work practices over time and differences in work practices across geographic regions. The evolution of methods likely also impacted sampling results over time; however, differences in sample results attributable to collection or analytical methods are difficult to quantify.

Ambient concentrations of PFAS varied by multiple orders of magnitude across manufacturing industries, including fluorochemical and ski waxing workplaces, and to a much less extent, the metal plating (1 sample) and textile industries. Across fluorochemical studies, PFOA levels at fluorochemical facilities have varied by up to five orders of magnitude [26, 39]; however, differences in processing conditions, the low number of samples collected, and the central tendency measure reported (median vs. mean) have likely played a role in the degree of perceived variation. Substantial variation in PFCA levels, including PFOA levels, was also present across multiple ski waxing air monitoring campaigns (up to four orders of magnitude for PFOA), but differences in PFCA levels at the ski wax facilities were likely mediated by the type of material processed at the facilities (solid block vs. powder wax) rather than processing conditions or sample size [35, 41, 43]. Few studies of manufacturing workers have evaluated ambient levels of precursor PFAS; however, of the studies that have, FTOH levels were consistently the highest among PFAS classes that were measured [22, 35, 50].

Precursor PFAS levels reported in non-manufacturing settings such as office buildings, schools, hotels, laboratories, and libraries have frequently been less than or approximate to levels reported in residential settings [33, 34, 37, 48, 49]. Sha et al. evaluated precursor PFAS levels in several offices, lecture halls, laboratories, and homes and reported that levels were higher in homes than occupational environments [47]. Differences in indoor levels were likely driven by differences in ventilation reported across indoor environments as forced ventilation systems were more frequently employed in occupational settings than in homes [47]. PFAS concentrations in non-manufacturing, indoor settings are also influenced by geographic location and age of the facility. FTOH concentrations reported from a cluster of US office buildings were comparable to levels reported in office buildings from a Swedish study but were an order of magnitude higher than levels reported in three separate studies of German office buildings [24, 37, 38, 46, 47]. Levels reported from the US office study varied significantly by building age and renovation status with buildings that were constructed within the last year exhibiting higher levels of FTOHs than the older, unrenovated buildings [38].

Precursor PFAS levels in stores that sell textile products such as outdoor clothing or carpet have consistently been elevated in comparison to residential settings [23, 37, 46, 49]. Separate studies that measured FTOH levels across a range of indoor environments reported that FTOH levels were 1–3 orders of magnitude higher in stores that sell textiles than in other indoor environments such as hotels, movie theaters, offices, and

residential settings [46, 49]. In a separate study, ambient FTOH levels at two outdoor equipment shops were an order of magnitude higher than FTOH levels at a carpet shop [37]; FTOH levels at the carpet shop were still substantially higher than levels in homes and in offices [37].

Because air monitoring studies of non-manufacturing settings have primarily focused on evaluating precursor PFAS levels, levels of short- and long-alkyl chain PFAS in these environments are poorly understood. Yao et al. reported that ambient levels of short- and long-alkyl chain PFAS were an order of magnitude higher in stores selling textiles than hotels, but significantly lower than levels reported across multiple movie theaters [49].

Exposure pathway—surface dust. A total of 12 studies have evaluated surface dust for PFAS in occupational settings. Findings from these studies suggest that surface dust levels are highly variable across both manufacturing and non-manufacturing settings. The lone study that evaluated PFAS surface dust levels at a fluorochemical plant estimated that surface dust was the most important exposure pathway for PFOA and PFHxS among fluorochemical workers; however, a small proportion of samples skewed the data heavily to the right [26]. Long-alkyl chain PFAS surface dust levels at a textile factory that produced firefighter turnout gear were much lower than levels reported in the fluorochemical study, but short-alkyl chain PFAS levels were considerably higher [23, 26]. This could partially be explained by experimental evidence indicating that turnout gear produced post-2012 contain high levels of short-alkyl chain PFAS (>1000 ppm), which over time can desorb from textile gear and deposit on the wearer or the floor [23].

Young et al. evaluated PFAS surface dust in firefighting stations and reported that PFAS levels were highest in turnout gear storage rooms [52]. AFFF type, the frequency of AFFF usage, and the volume of emergency calls at the station were important factors in determining PFAS surface dust levels across firefighting stations [52]. A separate study reported that the size of the station and the presence and volume of carpeting were also determinants of PFAS surface dust levels at firefighting stations [87]. Similar to the textile factory, precursor PFAS were the predominant congeners found across firefighting stations; however, in contrast, long-alkyl chain PFCAs and PFSAs were higher than short-alkyl chain PFAS at the firefighting stations [87].

While several studies have evaluated PFAS surface dust in office settings, major differences in which congeners were evaluated has made comparisons across studies difficult. Fraser et al. reported that FTOH levels were an order of magnitude higher than all other congener groups; however, no other office study has measured FTOH levels [36]. Moreover, levels of PFCAs and PFSAs in Fraser et al. were 1–2 orders of magnitude lower than levels reported in two other studies of office environments [25, 88, 89]. Because researchers have not measured FTOH levels in office environments where PFCAs or PFSAs are elevated, it is difficult to ascertain whether FTOH levels are consistently higher than PFCAs or PFSAs in office environments or if the distribution of PFAS in surface dust is subject to significant variability across office environments. Across these studies, levels of PFAS surface dust were consistently higher in offices than residential settings but lower than levels reported in classroom settings [88]. PFAS surface dust levels reported at a clothing shop were comparable to two separate office studies that were on the lower end of levels reported in office settings [28, 36, 89]. Yao et al. reported comparable levels of PFAS surface dust in hotels and residential settings; however, PFAS surface dust levels varied considerably from hotel to hotel, especially across precursor PFAS congeners [49]. Building age, renovation status, presence of carpeting, and extent of ventilation were deemed determinants of PFAS surface dust levels across several hotels [49].

Exposure pathway—skin contact. Few researchers have quantitatively evaluated PFAS levels near the skin of workers. N-ethyl perfluorooctane sulfonamido acetic acid was analyzed in clothing samples obtained from multiple body locations of 3 pesticide packing workers [13]. Estimated total body exposure for an 8- and 10-h workday were 359.1 ug/day and 448.6 ug/day, respectively [13]. These levels fell well below the margin of safety derived from toxicological studies that had been conducted previously [13]. In a separate study, PFAS were analyzed in wipe samples collected from turnout gear obtained from several firefighting stations [52]. Total PFAS levels obtained from 6 wipe samples varied widely from 1.2–84.5 ug [52].

Qualitative and semi-quantitative approaches for characterizing workplace exposure

In place of, or complementary to, collecting exposure data through environmental or biological sampling, several studies have utilized some combination of facility emission reports, annual usage data, worker history records, and activity pattern data to characterize exposure to workers in the fluorochemical industry.

Categorizing exposures into similar exposure groups. For assessing PFAS exposure in the fluorochemical industry, several studies have incorporated iterations of job exposure matrices to categorize workers into similar exposure groups. This approach to characterizing PFAS exposure has ranged from stratifying the workforces into binary exposure categories: exposed and non-exposed [53, 67, 90, 91], to more complex categorization using three or more categories to evaluate health indicators and outcomes in fluorochemical workers across an exposure gradient [20, 67, 92]. Most frequently, researchers have utilized a combination of worker history records and available serum data to classify workers into exposure groups; however, several researchers have also utilized historic usage, emission, or air monitoring data to characterize worker exposures [29, 91, 93–95]. From worker history records, researchers have primarily extracted job title and employment duration-related information to categorize exposures [91, 92]. Raleigh et al. categorized exposures based on the proportion of the workday spent doing exposure-related tasks and non-exposure-related tasks, in and outside of the processing area [95].

Semi-quantitative approaches for modeling exposures. Qualitative approaches for assigning exposure intensity are subject to worker misclassification and lack the resolution to explore temporal trends in exposure [29, 53, 90]. Using historical serum data, several researchers have estimated cumulative exposure in fluorochemical workers [64, 93, 94], and to a lesser extent, annual serum levels [96, 97]. Sleeuwenhoek et al. used semi-structured interviews and walkthroughs to gather activity pattern data over time at several fluorochemical plants [21]. Woskie et al. also estimated exposure over time but used emission and historical serum data instead [29]. Raleigh et al. estimated air exposure to PFAS for both production and non-production workers using available air monitoring, activity pattern, and annual usage data [95].

Additionally, several studies have modeled background exposures utilizing a combination of fate and transport modeling of affected waterways, water consumption rates, and annual emission rates from the nearby fluorochemical plant to delineate between exposures occurring at and away from the worksite [29, 64, 93].

DISCUSSION

This review adds to the occupational PFAS exposure literature by providing a summary of the peer-reviewed research. While previous reviews have summarized the many uses of PFAS,

contributions of PFAS exposure in community settings, and the relationship between PFAS exposure and health, this review is the first to comprehensively evaluate PFAS exposure data in occupational settings. The growing volume of publications over the last 25 years is indicative of the high level of interest in understanding PFAS exposure in the workplace, with an apparent shift toward a more diverse range of occupations of interest and approach to characterizing exposure (e.g., media and analytes) around 2010 that may reflect trends in exposure characterization for non-occupational populations. Articles identified in this review reported a range of PFAS concentrations, with substantial advancements in the capacity of analytical methods to capture a broader array of PFAS over the last decade leading to more robust PFAS analyses in more recent years.

Information on how different workplace factors contribute to PFAS exposure has also greatly expanded for both manufacturing and non-manufacturing settings. For manufacturing settings, processing conditions such a high temperature, low pH, and PFAS in dry powder form have been linked to elevated PFAS exposure. For textile manufacturing facilities, limited bulk and dust monitoring data suggest that textile manufacturing workers that produce flame retardant materials are at an elevated risk of PFAS exposure. For non-manufacturing industries, factors such as building age, presence of carpeting, and the extent of ventilation were heavily linked to PFAS exposure.

Efforts to quantify the impact of control implementation on PFAS exposure are documented for ski waxing and fluorochemical production facilities. For both industries, the utilization of local exhaust ventilation near the exposure source was linked to reductions in PFAS exposure. Other measures that led to reductions include maintaining pH levels above 7 to reduce volatilization potential and wetting PFAS-containing dry powders at fluorochemical production facilities and replacing powder wax with block wax at ski waxing facilities.

Research gaps

There are several major research gaps within the occupational PFAS exposure literature that deserve consideration prior to or when conducting future exposure assessments in occupational settings. Some of the most pressing research gaps are outlined below.

First, analytical capabilities for PFAS are rapidly improving and opportunities exist to utilize broader panels of targeted PFAS, as well as semi- and non-targeted analytical methods. Because of the limited biomonitoring data for short-alkyl chain PFAS and PFAS alternatives such as chlorinated PFESAs, it is unclear whether decreases in long-alkyl chain PFAS have been offset by increases in short-alkyl chain and alternative PFAS and if so, to what extent. Additionally, researchers have reported major differences in the physiochemical and bioaccumulation properties of PFAS across different isomeric forms, which can impact toxicity [98]. Researchers aiming to characterize biological PFAS exposure in working populations may consider evaluating a broader panel of PFAS that includes short-alkyl chain PFAS and PFAS alternatives as well as different isomeric forms.

Non-targeted analytical methods such as total oxidizable precursor assay and particle-induced gamma emission spectroscopy have previously been used, albeit to a limited extent, to measure total organic fluorine levels in dust and bulk samples collected from work environments; however, to our knowledge, researchers have yet to utilize these methods to evaluate total organic fluorine content in serum, air, or urine from occupational settings. Future exposure assessors may consider employing a non-targeted method in conjunction with targeted methods when evaluating PFAS levels in serum, urine, or air to place the results of the targeted PFAS into perspective.

Second, guidance on exposure reducing strategies and interventions is quite limited. Researchers have evaluated the effectiveness of various controls in reducing long-alkyl chain PFAS in fluorochemical production facilities and precursor PFAS in ski wax huts [21, 29, 39]. Utilizing more robust analytical methods, researchers could assess the effectiveness of these controls at reducing exposure to a wider array of PFAS in fluorochemical production and ski wax facility settings. Additionally, utilizing similar study design concepts, researchers could evaluate the effectiveness of different controls in reducing PFAS exposure in other workplace settings where exposures are potentially high.

Third, PFAS exposure biomarkers have only been quantified for a limited number of occupational groups. An increased emphasis on conducting biological PFAS exposure assessments for manufacturing and non-manufacturing occupations that are not currently well characterized could inform which industries deserve prioritization for future exposure intervention or epidemiological studies. Specific manufacturing-related occupations where the potential for PFAS exposure is high and the PFAS exposure biomarker data is limited or nonexistent, include textile workers, metal plating workers, paper mill workers, incineration workers, AFFF mixing workers, personal care product manufacturing workers, and contract workers involved in decommissioning PFAS manufacturing facilities [99, 100].

Fourth, research utilizing traditional industrial hygiene monitoring methods are limited. While several studies have included air monitoring of PFAS, only a fraction have conducted personal breath zone air sampling. For workers that perform multiple job tasks throughout the workday, area monitoring may not provide adequate resolution for evaluating inhalation exposure in these individuals. For more accurate assessments of PFAS exposure in workers, future air monitoring efforts may consider conducting PBZ monitoring in conjunction with area or task-based sampling.

For many of the air sampling studies that have been conducted in non-manufacturing work settings, researchers have only collected a very limited number [1, 2] of air samples per work environment. Researchers should take caution when interpreting the results of these studies given their small sample sizes. Furthermore, future exposure assessment efforts at non-manufacturing settings may consider collecting more samples to increase the power of their data.

Fifth, research characterizing toxicokinetic factors and occupationally relevant exposure pathways and routes is limited. Future studies of occupational exposures would benefit from improved characterization of absorption, distribution, metabolism, and elimination to guide the timing of biological sample collection for peek concentrations following an exposure or periods of continuous exposure. Few studies have conducted air and biological exposure monitoring simultaneously for workers. More efforts to assess the correlation between air monitoring and biological exposure monitoring data would benefit researchers attempting to better understand the relationships between ambient PFAS levels in the work environment and biological exposure levels. Quantifying these relationships across a wide range of ambient concentrations could contribute toward the development of biological exposure indices, which would further strengthen the value in conducting biological exposure assessment studies.

Given the limited number of studies that have included sampling near the skin of workers and the absence of studies sampling directly from the skin, it remains unclear how important of a contributor the dermal route is across different workplaces settings. In select manufacturing settings, such as fluorochemical production, textile manufacturing, and metal plating facilities, the dermal route may contribute substantially toward PFAS exposure. Future research efforts may consider addressing the dermal route when evaluating PFAS exposure in manufacturing settings. Additionally, more experimental studies aiming to assess the permeation efficiency of different PFAS under various, relevant occupational conditions are warranted to place future dermal exposure results into perspective.

Lastly, given how PFAS comprise a large group of chemicals and there exists a wide range of occupational environments with the potential for exposure, future researchers may consider including comprehensive descriptions of their sample collection and analytical methods for stronger comparisons across PFAS exposure studies moving forward. Additionally, information describing the workplace conditions of the sampling environment would also enhance the comparability of studies, as certain conditions, such as temperature, relative humidity, ventilation performance, and pH [39, 41], can impact PFAS levels in the work environment, partitioning behavior of individual PFAS (Table S2), and the collection efficiency of different sampling instruments, thus affecting the interpretation of sampling results.

Strengths and limitations of review

This examination of the peer-reviewed occupational PFAS exposure literature adds a much needed assessment to the existing body of knowledge. Collection and analytical methods, sampling results, exposure determinants for different occupational groups, exposure estimation strategies, and important research gaps were summarized; whereas health effects and health outcomes related to PFAS exposure were outside of the scope of this review and were therefore not discussed.

There were a few limitations pertaining to the methodology of the review process. Due to variations and changes in PFAS terminology over the last four decades, including sufficiently inclusive search terms was challenging and resulted in one relevant article from 1980 not being captured during the formal literature search. The presence of study selection bias may have affected the studies included in the review. While some aspects of the PRISMA standard were not integrated into the approach, the review was conducted based on PRISMA principles, with the assistance of a librarian, a formal set of exclusion criteria, and two reviewers to minimize this risk.

Because collection methods were not consistently defined in the literature, it was difficult to elucidate how different sampling methodologies may have influenced the reported results for each study. Several industries represented in this review included very limited exposure data. Details presented in this review regarding those industries should be interpreted in that context. Due to differences in partitioning, direct comparisons between studies that reported PFAS levels in plasma or whole blood and studies that reported PFAS in serum should be interpreted with caution. Descriptive statistics were not consistently reported in the literature making it difficult to compare results.

CONCLUSIONS

Research characterizing occupational exposures to PFAS consists of 92 studies published over 41 years spanning a wide array of manufacturing and non-manufacturing work environments and data collected from biological and traditional industrial hygiene sources. Overall, findings from this review indicate that an increased emphasis on conducting robust PFAS exposure assessment efforts in non-fluorochemical manufacturing settings may be beneficial. As regulatory oversight and commercial trends continue to shift production away from long-alkyl chain PFAS, an increased focus would be helpful to identify classes of PFAS that are replacing long-alkyl chain PFAS. An improved understanding of occupational exposure levels across various environmental and biological matrices would inform future research aiming to better understand the relationship between PFAS exposure and health outcomes. Epidemiological studies conducted in occupational environments would be especially valuable in informing threshold-based recommendations, including whether current occupational exposure limits are sufficiently protective of workers as well as whether additional exposure limits are worth considering for different PFAS.

DISCLAIMER

The findings and conclusions in this report are those of the author(s) and do not necessarily represent the official position of the National Institute for Occupational Safety and Health (NIOSH), Centers for Disease Control and Prevention (CDC).

DATA AVAILABILITY

See the Supplementary Information section for information on where the selected articles for this review were extracted from.

REFERENCES

1. EPA. Comptox chemicals dashboard: master list of PFAS substances. United States Environmental Protection Agency; 2021.
2. Buck RC, Franklin J, Berger U, Conder JM, Cousins IT, de Voogt P, et al. Perfluoroalkyl and polyfluoroalkyl substances in the environment: terminology, classification, and origins. Integr Environ Assess Manag. 2011;7:513–41.
3. Calafat AM, Kato K, Hubbard K, Jia T, Botelho JC, Wong LY. Legacy and alternative per- and polyfluoroalkyl substances in the U.S. general population: paired serum-urine data from the 2013–2014 National Health and Nutrition Examination Survey. Environ Int. 2019;131:105048.
4. Fenton SE, Ducatman A, Boobis A, DeWitt JC, Lau C, Ng C, et al. Per- and polyfluoroalkyl substance toxicity and human health review: current state of knowledge and strategies for informing future research. Environ Toxicol Chem. 2021;40:606–30.
5. OSHA. General Duty Clause. United States Occupational Safety and Health Administration; 1970.
6. OSHA. Standard interpretations: elements necessary for a violation of the General Duty Clause. United States Occupational Safety and Health Administration; 2003.
7. National Academies of Sciences Engineering and Medicine. Guidance on PFAS exposure, testing, and clinical follow-up. Washington DC: The National Academies Press; 2022.
8. Interstate Technology Regulatory Council (ITRC). Naming conventions and physical and chemical properties. Washington, D.C.: Interstate Technology Regulatory Council (ITRC); 2018.
9. Gomis MI, Vestergren R, Borg D, Cousins IT. Comparing the toxic potency in vivo of long-chain perfluoroalkyl acids and fluorinated alternatives. Environ Int. 2018;113:1–9.
10. Bjorklund JA, Thuresson K, De Wit CA. Perfluoroalkyl compounds (PFCs) in indoor dust: concentrations, human exposure estimates, and sources. Environ Sci Technol. 2009;43:2276–81.
11. Su H, Lu Y, Wang P, Shi Y, Li Q, Zhou Y, et al. Perfluoroalkyl acids (PFAAs) in indoor and outdoor dusts around a mega fluorochemical industrial park in China: implications for human exposure. Environ Int. 2016;94:667–73.
12. Poothong S, Padilla-Sanchez JA, Papadopoulou E, Giovanoulis G, Thomsen C, Haug LS, et al. Wipes: a useful tool for assessing human exposure to poly- and perfluoroalkyl substances (PFASs) through hand-to-mouth and dermal contacts. Environ Sci Technol. 2019;53:1985–93.
13. Machado-Neto JG, Queiroz ME, Carvalho D, Bassini AJ. Risk of intoxication with sulfluramid in a packing plant of Mirex-S. Bull Environ Contam Toxicol. 1999;62:515–9.
14. Franko J, Meade BJ, Frasch HF, Barbero AM, Anderson SE. Dermal penetration potential of perfluorooctanoic acid (PFOA) in human and mouse skin. J Toxicol Environ Health A 2012;75:50–62.
15. Interstate Technology Regulatory Council (ITRC). History and use of per- and polyfluoroalkyl substances (PFAS). Washington, D.C.: Interstate Technology Regulatory Council (ITRC); 2017.
16. EPA. 2010/2015 PFOA Stewardship Program—2014 annual progress reports. United States Environmental Protection Agency; 2016.
17. CDC. Fourth national report on human exposure to environmental chemicals: updated tables. United States Centers for Disease Control and Prevention; 2019.
18. Interstate Technology Regulatory Council (ITRC). Chemistry, terminology, and acronyms. Washington, D.C.: Interstate Technology Regulatory Council (ITRC); 2021.
19. Silver MJ, Young DK. Acute noncardiogenic pulmonary edema due to polymer fume fever. Clevel Clin J Med. 1993;60:479–82.
20. Mundt DJ, Mundt KA, Luippold RS, Schmidt MD, Farr CH. Clinical epidemiological study of employees exposed to surfactant blend containing perfluorononanoic acid. Occup Environ Med. 2007;64:589–94.
21. Sleeuwenhoek A, Cherrie JW. Exposure assessment of tetrafluoroethylene and ammonium perfluorooctanoate 1951–2002. J Environ Monit. 2012;14:775–81.

22. Heydebreck F, Tang J, Xie Z, Ebinghaus R. Emissions of per- and polyfluoroalkyl substances in a textile manufacturing plant in China and their relevance for workers' exposure. Environ Sci Technol. 2016;50:10386–96.
23. Peaslee GF, Wilkinson JT, McGuinness SR, Tighe M, Caterisano N, Lee S, et al. Another pathway for firefighter exposure to per- and polyfluoroalkyl substances: firefighter textiles. Environ Sci Technol Lett. 2020;7:594–9.
24. Jahnke A, Huber S, Temme C, Kylin H, Berger U. Development and application of a simplified sampling method for volatile polyfluorinated alkyl substances in indoor and environmental air. J Chromatogr A. 2007;1164:1–9.
25. Besis A, Botsaropoulou E, Samara C, Katsoyiannis A, Hanssen L, Huber S. Perfluoroalkyl substances (PFASs) in air-conditioner filter dust of indoor microenvironments in Greece: implications for exposure. Ecotoxicol Environ Saf. 2019;183:109559.
26. Gao Y, Fu J, Cao H, Wang Y, Zhang A, Liang Y, et al. Differential accumulation and elimination behavior of perfluoroalkyl acid isomers in occupational workers in a manufactory in China. Environ Sci Technol. 2015;49:6953–62.
27. Fu J, Gao Y, Cui L, Wang T, Liang Y, Qu G, et al. Occurrence, temporal trends, and half-lives of perfluoroalkyl acids (PFAAs) in occupational workers in China. Sci Rep. 2016;6:38039.
28. Wu N, Cai D, Guo M, Li M, Li X. Per- and polyfluorinated compounds in saleswomen's urine linked to indoor dust in clothing shops. Sci Total Environ. 2019;667:594–600.
29. Woskie SR, Gore R, Steenland K. Retrospective exposure assessment of perfluorooctanoic acid serum concentrations at a fluoropolymer manufacturing plant. Ann Occup Hyg. 2012;56:1025–37.
30. Shi Y, Vestergren R, Xu L, Zhou Z, Li C, Liang Y, et al. Human exposure and elimination kinetics of chlorinated polyfluoroalkyl ether sulfonic acids (Cl-PFESAs). Environ Sci Technol. 2016;50:2396–404.
31. Guruge KS, Taniyasu S, Yamashita N, Wijeratna S, Mohotti KM, Seneviratne HR, et al. Perfluorinated organic compounds in human blood serum and seminal plasma: a study of urban and rural tea worker populations in Sri Lanka. J Environ Monit. 2005;7:371–7.
32. Tao L, Kannan K, Aldous KM, Mauer MP, Eadon GA. Biomonitoring of perfluorochemicals in plasma of New York State personnel responding to the World Trade Center disaster. Environ Sci Technol. 2008;42:3472–8.
33. Shoeib M, Harner T, Ikonomou M, Kannan K. Indoor and outdoor air concentrations and phase partitioning of perfluoroalkyl sulfonamides and polybrominated diphenyl ethers. Environ Sci Technol. 2004;38:1313–20.
34. Shoeib M, Harner T, Lee SC, Lane D, Zhu J. Sorbent-impregnated polyurethane foam disk for passive air sampling of volatile fluorinated chemicals. Anal Chem. 2008;80:675–82.
35. Nilsson H, Karrman A, Rotander A, van Bavel B, Lindstrom G, Westberg H. Professional ski waxers' exposure to PFAS and aerosol concentrations in gas phase and different particle size fractions. Environ Sci Process Impacts. 2013;15:814–22.
36. Fraser AJ, Webster TF, Watkins DJ, Strynar MJ, Kato K, Calafat AM, et al. Polyfluorinated compounds in dust from homes, offices, and vehicles as predictors of concentrations in office workers' serum. Environ Int. 2013;60:128–36.
37. Langer V, Dreyer A, Ebinghaus R. Polyfluorinated compounds in residential and nonresidential indoor air. Environ Sci Technol. 2010;44:8075–81.
38. Fraser AJ, Webster TF, Watkins DJ, Nelson JW, Stapleton HM, Calafat AM, et al. Polyfluorinated compounds in serum linked to indoor air in office environments. Environ Sci Technol. 2012;46:1209–15.
39. Kaiser MA, Dawson BJ, Barton CA, Botelho MA. Understanding potential exposure sources of perfluorinated carboxylic acids in the workplace. Ann Occup Hyg. 2010;54:915–22.
40. Liesivuori J, Kiviranta H, Laitinen J, Hesso A, Haemeilae M, Tornaeus J, et al. Airborne aerosols in application of polyfluoro polymer-based ski waxes. Ann Occup Hyg. 1994;38:931–7.
41. Freberg BI, Olsen R, Thorud S, Ellingsen DG, Daae HL, Hersson M, et al. Chemical exposure among professional ski waxers-characterization of individual work operations. Ann Occup Hyg. 2013;57:286–95.
42. Nilsson H, Karrman A, Rotander A, van Bavel B, Lindstrom G, Westberg H. Biotransformation of fluorotelomer compound to perfluorocarboxylates in humans. Environ Int. 2013;51:8–12.
43. Freberg BI, Olsen R, Daae HL, Hersson M, Thorud S, Ellingsen DG, et al. Occupational exposure assessment of airborne chemical contaminants among professional ski waxers. Ann Occup Hyg. 2014;58:601–11.
44. Freberg BI, Haug LS, Olsen R, Daae HL, Hersson M, Thomsen C, et al. Occupational exposure to airborne perfluorinated compounds during professional ski waxing. Environ Sci Technol. 2010;44:7723–8.
45. Ubel FA, Sorenson SD, Roach DE. Health status of plant workers exposed to fluorochemicals—a preliminary report. Am Ind Hyg Assoc J. 1980;41:584–9.
46. Schlummer M, Gruber L, Fiedler D, Kizlauskas M, Muller J. Detection of fluorotelomer alcohols in indoor environments and their relevance for human exposure. Environ Int. 2013;57-58:42–49.
47. Sha B, Dahlberg AK, Wiberg K, Ahrens L. Fluorotelomer alcohols (FTOHs), brominated flame retardants (BFRs), organophosphorus flame retardants (OPFRs) and cyclic volatile methylsiloxanes (cVMSs) in indoor air from occupational and home environments. Environ Pollut. 2018;241:319–30.
48. Fromme H, Dreyer A, Dietrich S, Fembacher L, Lahrz T, Volkel W. Neutral polyfluorinated compounds in indoor air in Germany—the LUPE 4 study. Chemosphere. 2015;139:572–8.
49. Yao Y, Zhao Y, Sun H, Chang S, Zhu L, Alder AC, et al. Per- and polyfluoroalkyl substances (pFASs) in indoor air and dust from homes and various microenvironments in China: implications for human exposure. Environ Sci Technol. 2018;52:3156–66.
50. Nilsson H, Karrman A, Rotander A, van Bavel B, Lindstrom G, Westberg H. Inhalation exposure to fluorotelomer alcohols yield perfluorocarboxylates in human blood? Environ Sci Technol. 2010;44:7717–22.
51. Simonetti G, Di Filippo P, Riccardi C, Pomata D, Sonego E, Buiarelli F. Occurrence of halogenated pollutants in domestic and occupational indoor dust. Int J Environ Res Public Health. 2020;17:3813–28.
52. Young AS, Sparer-Fine EH, Pickard HM, Sunderland EM, Peaslee GF, Allen JG. Per- and polyfluoroalkyl substances (PFAS) and total fluorine in fire station dust. J Expo Sci Environ Epidemiol. 2021;31:930–42.
53. Gilliland FD, Mandel JS. Mortality among employees of a perfluorooctanoic acid production plant. J Occup Med. 1993;35:950–4.
54. Gilliland FD, Mandel JS. Serum perfluorooctanoic acid and hepatic enzymes, lipoproteins, and cholesterol: a study of occupationally exposed men. Am J Ind Med. 1996;29:560–8.
55. Olsen GW, Burris JM, Mandel JH, Zobel LR. Serum perfluorooctane sulfonate and hepatic and lipid clinical chemistry tests in fluorochemical production employees. J Occup Environ Med. 1999;41:799–806.
56. Olsen GW, Burris JM, Burlew MM, Mandel JH. Plasma cholecystokinin and hepatic enzymes, cholesterol and lipoproteins in ammonium perfluorooctanoate production workers. Drug Chem Toxicol. 2000;23:603–20.
57. Girardi P, Merler E. A mortality study on male subjects exposed to polyfluoroalkyl acids with high internal dose of perfluorooctanoic acid. Environ Res. 2019;179:108743.
58. Olsen GW, Burris JM, Burlew MM, Mandel JH. Epidemiologic assessment of worker serum perfluorooctanesulfonate (PFOS) and perfluorooctanoate (PFOA) concentrations and medical surveillance examinations. J Occup Environ Med. 2003;45:260–70.
59. Olsen GW, Zobel LR. Assessment of lipid, hepatic, and thyroid parameters with serum perfluorooctanoate (PFOA) concentrations in fluorochemical production workers. Int Arch Occup Environ Health. 2007;81:231–46.
60. Sakr CJ, Leonard RC, Kreckmann KH, Slade MD, Cullen MR. Longitudinal study of serum lipids and liver enzymes in workers with occupational exposure to ammonium perfluorooctanoate. J Occup Environ Med. 2007;49:872–9.
61. Olsen GW, Logan PW, Hansen KJ, Simpson CA, Burris JM, Burlew MM, et al. An occupational exposure assessment of a perfluorooctanesulfonyl fluoride production site: biomonitoring. AIHA J. 2003;64:651–9.
62. Costa G, Sartori S, Consonni D. Thirty years of medical surveillance in perfluooctanoic acid production workers. J Occup Environ Med. 2009;51:364–72.
63. Olsen GW, Burris JM, Ehresman DJ, Froehlich JW, Seacat AM, Butenhoff JL, et al. Half-life of serum elimination of perfluorooctanesulfonate,perfluorohexanesulfonate, and perfluorooctanoate in retired fluorochemical production workers. Environ Health Perspect. 2007;115:1298–305.
64. Winquist A, Steenland K. Modeled PFOA exposure and coronary artery disease, hypertension, and high cholesterol in community and worker cohorts. Environ Health Perspect. 2014;122:1299–305.
65. Olsen GW, Ehresman DJ, Buehrer BD, Gibson BA, Butenhoff JL, Zobel LR. Longitudinal assessment of lipid and hepatic clinical parameters in workers involved with the demolition of perfluoroalkyl manufacturing facilities. J Occup Environ Med. 2012;54:974–83.
66. Wang J, Zhang Y, Zhang W, Jin Y, Dai J. Association of perfluorooctanoic acid with HDL cholesterol and circulating miR-26b and miR-199-3p in workers of a fluorochemical plant and nearby residents. Environ Sci Technol. 2012;46:9274–81.
67. Alexander BH, Olsen GW, Burris JM, Mandel JH, Mandel JS. Mortality of employees of a perfluorooctanesulphonyl fluoride manufacturing facility. Occup Environ Med. 2003;60:722–9.
68. Lu Y, Gao K, Li X, Tang Z, Xiang L, Zhao H, et al. Mass spectrometry-based metabolomics reveals occupational exposure to per- and polyfluoroalkyl substances relates to oxidative stress, fatty acid beta-oxidation disorder, and kidney injury in a manufactory in China. Environ Sci Technol. 2019;53:9800–9.

69. Sakr CJ, Kreckmann KH, Green JW, Gillies PJ, Reynolds JL, Leonard RC. Cross-sectional study of lipids and liver enzymes related to a serum biomarker of exposure (ammonium perfluorooctanoate or APFO) as part of a general health survey in a cohort of occupationally exposed workers. J Occup Environ Med. 2007;49:1086–96.
70. Leary DB, Takazawa M, Kannan K, Khalil N. Perfluoroalkyl substances and metabolic syndrome in firefighters: a pilot study. J Occup Environ Med. 2020;62:52–7.
71. Shaw SD, Berger ML, Harris JH, Yun SH, Wu Q, Liao C, et al. Persistent organic pollutants including polychlorinated and polybrominated dibenzo-p-dioxins and dibenzofurans in firefighters from Northern California. Chemosphere. 2013;91:1386–94.
72. Dobraca D, Israel L, McNeel S, Voss R, Wang M, Gajek R, et al. Biomonitoring in California firefighters: metals and perfluorinated chemicals. J Occup Environ Med. 2015;57:88–97.
73. Trowbridge J, Gerona RR, Lin T, Rudel RA, Bessonneau V, Buren H, et al. Exposure to perfluoroalkyl substances in a cohort of women firefighters and office workers in San Francisco. Environ Sci Technol. 2020;54:3363–74.
74. Clarity C, Trowbridge J, Gerona R, Ona K, McMaster M, Bessonneau V, et al. Associations between polyfluoroalkyl substance and organophosphate flame retardant exposures and telomere length in a cohort of women firefighters and office workers in San Francisco. Environ Health. 2021;20:97.
75. Khalil N, Ducatman AM, Sinari S, Billheimer D, Hu C, Littau S, et al. Per- and polyfluoroalkyl substance and cardio metabolic markers in firefighters. J Occup Environ Med. 2020;62:1076–81.
76. Graber JM, Black TM, Shah NN, Caban-Martinez AJ, Lu SE, Brancard T, et al. Prevalence and predictors of per- and polyfluoroalkyl substances (PFAS) serum levels among members of a suburban US Volunteer Fire Department. Int J Environ Res Public Health. 2021;18:3730.
77. Rotander A, Toms LM, Aylward L, Kay M, Mueller JF. Elevated levels of PFOS and PFHxS in firefighters exposed to aqueous film forming foam (AFFF). Environ Int. 2015;82:28–34.
78. Jin C, Sun Y, Islam A, Qian Y, Ducatman A. Perfluoroalkyl acids including perfluorooctane sulfonate and perfluorohexane sulfonate in firefighters. J Occup Environ Med. 2011;53:324–8.
79. Laitinen JA, Koponen J, Koikkalainen J, Kiviranta H. Firefighters' exposure to perfluoroalkyl acids and 2-butoxyethanol present in firefighting foams. Toxicol Lett. 2014;231:227–32.
80. Rotander A, Karrman A, Toms LM, Kay M, Mueller JF, Gomez Ramos MJ. Novel fluorinated surfactants tentatively identified in firefighters using liquid chromatography quadrupole time-of-flight tandem mass spectrometry and a case-control approach. Environ Sci Technol. 2015;49:2434–42.
81. Nilsson H, Karrman A, Westberg H, Rotander A, van Bavel B, Lindstrom G. A time trend study of significantly elevated perfluorocarboxylate levels in humans after using fluorinated ski wax. Environ Sci Technol. 2010;44:2150–5.
82. Gomis MI, Vestergren R, Nilsson H, Cousins IT. Contribution of direct and indirect exposure to human serum concentrations of perfluorooctanoic acid in an occupationally exposed group of ski waxers. Environ Sci Technol. 2016;50:7037–46.
83. Zhou Z, Shi Y, Vestergren R, Wang T, Liang Y, Cai Y. Highly elevated serum concentrations of perfluoroalkyl substances in fishery employees from Tangxun Lake, China. Environ Sci Technol. 2014;48:3864–74.
84. Bloom MS, Kannan K, Spliethoff HM, Tao L, Aldous KM, Vena JE. A preliminary study of temporal differences in serum concentrations of perfluoroalkyl acids, among New York anglers, in the absence of known changes in manufacturing practices. Toxicol Environ Chem. 2009;91:1387–97.
85. Lu C, Shi YL, Zhou Z, Liu NN, Meng ZF, Cai YQ. Perfluorinated compounds in blood of textile workers and barbers. Chin Chem Lett. 2014;25:1145–8.
86. Olsen GW, Chang SC, Noker PE, Gorman GS, Ehresman DJ, Lieder PH, et al. A comparison of the pharmacokinetics of perfluorobutanesulfonate (PFBS) in rats, monkeys, and humans. Toxicology. 2009;256:65–74.
87. Hall SM, Patton S, Petreas M, Zhang S, Phillips AL, Hoffman K, et al. Per- and polyfluoroalkyl substances in dust collected from residential homes and fire stations in North America. Environ Sci Technol. 2020;54:14558–67.
88. Goosey E, Harrad S. Perfluoroalkyl compounds in dust from Asian, Australian, European, and North American homes and UK cars, classrooms, and offices. Environ Int. 2011;37:86–92.
89. D'Hollander W, Roosens L, Covaci A, Cornelis C, Reynders H, Campenhout KV, et al. Brominated flame retardants and perfluorinated compounds in indoor dust from homes and offices in Flanders, Belgium. Chemosphere. 2010;81:478–87.
90. Leonard RC, Kreckmann KH, Sakr CJ, Symons JM. Retrospective cohort mortality study of workers in a polymer production plant including a reference population of regional workers. Ann Epidemiol. 2008;18:15–22.
91. Grice MM, Alexander BH, Hoffbeck R, Kampa DM. Self-reported medical conditions in perfluorooctanesulfonyl fluoride manufacturing workers. J Occup Environ Med. 2007;49:722–9.
92. Lundin JI, Alexander BH, Olsen GW, Church TR. Ammonium perfluorooctanoate production and occupational mortality. Epidemiology. 2009;20:921–8.
93. Steenland K, Zhao L, Winquist A. A cohort incidence study of workers exposed to perfluorooctanoic acid (PFOA). Occup Environ Med. 2015;72:373–80.
94. Karnes C, Winquist A, Steenland K. Incidence of type II diabetes in a cohort with substantial exposure to perfluorooctanoic acid. Environ Res. 2014;128:78–83.
95. Raleigh KK, Alexander BH, Olsen GW, Ramachandran G, Morey SZ, Church TR, et al. Mortality and cancer incidence in ammonium perfluorooctanoate production workers. Occup Environ Med. 2014;71:500–6.
96. Kreckmann KH, Sakr CJ, Leonard RC, Dawson BJ. Estimation and validation of biomarker-based exposures for historical ammonium perfluorooctanoate. J Occup Environ Hyg. 2009;6:511–6.
97. Steenland K, Woskie S. Cohort mortality study of workers exposed to perfluorooctanoic acid. Am J Epidemiol. 2012;176:909–17.
98. Zeng XW, Lodge CJ, Dharmage SC, Bloom MS, Yu Y, Yang M, et al. Isomers of per- and polyfluoroalkyl substances and uric acid in adults: isomers of C8 Health Project in China. Environ Int. 2019;133(Pt A):105160.
99. Glüge J, Scheringer M, Cousins IT, DeWitt JC, Goldenman G, Herzke D, et al. An overview of the uses of per- and polyfluoroalkyl substances (PFAS). Environ Sci Process Impacts. 2020;22:2345–73.
100. Cousins IT, Goldenman G, Herzke D, Lohmann R, Miller M, Ng CA, et al. The concept of essential use for determining when uses of PFASs can be phased out. Environ Sci Process Impacts. 2019;21:1803–15.
101. Steenland K, Tinker S, Frisbee S, Ducatman A, Vaccarino V. Association of perfluorooctanoic acid and perfluorooctane sulfonate with serum lipids among adults living near a chemical plant. Am J Epidemiol. 2009;170:1268–78.
102. Kato K, Kalathil AA, Patel AM, Ye X, Calafat AM. Per- and polyfluoroalkyl substances and fluorinated alternatives in urine and serum by on-line solid phase extraction-liquid chromatography-tandem mass spectrometry. Chemosphere. 2018;209:338–45.
103. Ericson I, Gómez M, Nadal M, van Bavel B, Lindström G, Domingo JL. Perfluorinated chemicals in blood of residents in Catalonia (Spain) in relation to age and gender: a pilot study. Environ Int. 2007;33:616–23.
104. Karrman A, Mueller JF, van Bavel B, Harden F, Toms LM, Lindstrom G. Levels of 12 perfluorinated chemicals in pooled australian serum, collected 2002–2003, in relation to age, gender, and region. Environ Sci Technol. 2006;40:3742–8.
105. Karrman A, van Bavel B, Jarnberg U, Hardell L, Lindstrom G. Perfluorinated chemicals in relation to other persistent organic pollutants in human blood. Chemosphere. 2006;64:1582–91.
106. Tanner EM, Bloom MS, Wu Q, Kannan K, Yucel RM, Shrestha S, et al. Occupational exposure to perfluoroalkyl substances and serum levels of perfluorooctanesulfonic acid (PFOS) and perfluorooctanoic acid (PFOA) in an aging population from upstate New York: a retrospective cohort study. Int Arch Occup Environ Health. 2018;91:145–54.

ACKNOWLEDGEMENTS

This work was supported in part by an interagency agreement between the National Institute for Occupational Safety and Health (NIOSH) and the National Institute of Environmental Health Sciences (AES22002-001-0000) as a collaborative National Toxicology Program research activity.

AUTHOR CONTRIBUTIONS

BTC was responsible for designing the search strategy, screening for eligible studies, extracting relevant data, interpreting study results, producing tables and figures, producing the initial draft of the manuscript, and revising the manuscript. MMC was responsible for designing the search strategy, screening for eligible studies, producing tables and figures, interpreting results, and revising the manuscript. The CDC Thacker Library provided technical assistance in designing the search strategy and ran the search strategy through multiple databases. Other Contributions: The CDC Thacker Library provided technical assistance in designing the search strategy and ran the search strategy through multiple databases. Additionally, Jessica Rinsky, Susan Moore, Susan Fenton, Kristen Ryan, Pei Li Yao, and Kristin Eccles reviewed the paper and provided helpful feedback.

COMPETING INTERESTS

The authors declare no competing interests.

ADDITIONAL INFORMATION

Supplementary information The online version contains supplementary material available at https://doi.org/10.1038/s41370-023-00536-y.

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