Emerging Contaminants 12 (2026) 100725

Contents lists available at ScienceDirect

Emerging Contaminants

journal homepage: www.keaipublishing.com/en/journals/emerging-contaminants

Determination of per- and polyfluoroalkyl substances (PFAS) in plasma of Italian firefighters

Arianna Fornasari [a], Michele Pozzebon [b], Simone Santelli [a], Giovanni Dal Lago [a], Carla Arniani [c], Sofia Raniolo [d], Schiavonea Modesto [d], Jacopo Lenzi [e], Paolo Fais [a], Jennifer P. Pascali [a,*]

[a] Department of Medical and Surgical Sciences, Unit of Legal Medicine, University of Bologna, Bologna, Italy
[b] DTO, Viale Viareggio 34/E 30038, Spinea, VE, Italy
[c] Via pietro Mascagni 15, Bagnara di Romagna, RA, 48031, Italy
[d] Italian Firefighting Corp, Piazza del Viminale, 1, Rome, 00184, Italy
[e] Department of Biomedical and Neuromotor Sciences, University of Bologna, Bologna, Italy

ARTICLE INFO

Article history:
Received 9 June 2026
Received in revised form
19 August 2026
Accepted 20 August 2026
Available online 23 August 2026

Keywords:
PFAS
Perfluoroalkyl substances
Polyfluoroalkyl substances
Occupational exposure
Firefighters
Plasma
Italy

ABSTRACT

Per- and polyfluoroalkyl substances (PFAS) are highly persistent chemicals widely used in industrial processes and associated with extensive environmental contamination. Due to their bioaccumulative properties, PFAS exposure has been linked to adverse health effects, and some compounds have been classified as carcinogenic or possibly carcinogenic to humans. Some groups of workers are considered at risk, and particularly firefighters are considered a potentially high-risk occupational group because of repeated exposure to hazardous substances, such as PFAS. Therefore, this study aimed to evaluate PFAS exposure among firefighters operating in central and northern Italy by measuring plasma concentrations of PFAS and investigating anamnestic determinants. A total of 378 firefighters were voluntarily recruited and completed an anamnestic questionnaire. Plasma samples were analyzed using validated liquid chromatography–tandem mass spectrometry (LC–MS/MS) for 24 PFAS congeners from the EPA 533 list. Of the 24 PFAS investigated, 12 were detected in the analyzed samples: PFBA, PFHxS, PFOA, PFOS, PFDA, PFUnA, PFNA, PFHpS, PFHpA, 6:2 FTS, PFPeA, and 9Cl-PF3ONS. The mean summed concentration of the detected PFAS was 7.26 ng/mL (SD = 4.58 ng/mL). PFOS and PFOA were the predominant compounds, with mean concentrations of 4.34 ng/mL and 1.27 ng/mL, respectively. In primary analyses, significant differences emerged according to different headquarters, age, sex, fish and seafood consumption, use of waterproof clothing outside the workplace, qualification, years of service, and type of service. In age- and sex-adjusted sensitivity analyses, the association with different headquarters remained significant, and fish and seafood consumption, type of service, and qualification also remained associated with total PFAS levels, whereas the associations with years of service and use of waterproof clothing outside the workplace were attenuated. No significant associations emerged in the primary analysis for BMI, smoking habits, living environment, or organic food consumption.
© 2026 The Authors. Publishing services by Elsevier B.V. on behalf of KeAi Communications Co. Ltd. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

1. Introduction

Per- and polyfluoroalkyl substances (PFAS) are a large family of up to four thousands of synthetic molecules composed of a fully or partially fluorinated carbon chain of various lengths [1–4]. There is no all-inclusive list of products describing the uses of specific PFAS, but it is commonly established that industry strongly relies on these compounds for the production of many daily products. These

* Corresponding author. Legal Medicine Via Irnerio 49, 40126, Department of Medical and Surgical Sciences, University of Bologna Bologna, Italy.
E-mail address: jennifer.pascali@unibo.it (J.P. Pascali).
Peer review under the responsibility of Editorial Office of Emerging Contaminants

https://doi.org/10.1016/j.emcon.2026.100725
2405-6650/© 2026 The Authors. Publishing services by Elsevier B.V. on behalf of KeAi Communications Co. Ltd. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

substances are characterized by a marked chemical stability, which makes them resistant to extreme temperatures, chemical reactions and microbial degradation and therefore perfectly suitable for industrial activities and daily life objects production [5]. Therefore, PFAS are released into the environment through multiple sources of contamination, including emissions from manufacturing facilities, landfills, wastewater treatment plants, firefighting and military activities, indoor consumer products, and vehicles' emissions [6–11], which pose serious problems for environment and public health. Humans are exposed to PFAS via ingestion, absorption and inhalation of contaminated food, cosmetics and clothes and air. Many studies have attempted to estimate the burden of contamination in human specimens; however, sensitive analytical methods for PFAS detection have only become available in recent years [12,13] and still do not allow the identification of all possible PFAS congeners, as most methods target only the most common and legacy PFAS. Notwithstanding this limitation, many significant results are available nowadays, which testify increasing prevalence of PFAS in the environment, especially in water supplies [14] and consequently in human specimens [15–17]. PFAS are now considered pollutants of major concern ([18]; Jane et al., 2022; [19]), with suggested pathological effects on the endocrine system, the immune system (ASTR, 2018; [20–22]; Jane et al., 2022; [23–30]), on kidney and liver [31–48], on male and female reproductive systems, and on cardiovascular system [49–56]. Importantly, perfluorooctanoic acid (PFOA) and perfluorooctane sulfonic acid (PFOS) have been classified by the International Agency for Research on Cancer (IARC), respectively belonging to Group 1 (i.e., carcinogenic to humans) and as Group 2B (i.e., possibly carcinogenic to humans) [57].

In addition to lifestyle-related risks affecting the general population, certain occupational groups are recognized as being at higher risk of PFAS exposure. In scientific literature, firefighters have been consistently associated with specific health risks; beyond the intrinsic thermal and thermophysiological hazards of firefighting, factors contributing to acute injuries and physiological stress [58–60], chronic exposure to hazardous chemicals remains a major concern. Among these, PFAS are of relevance due to their allegedly use in personal protective equipment and firefighting foams. Firefighters are primarily exposed to PFAS via ingestion and inhalation, including accidental ingestion of aqueous film-forming foams and inhalation of aerosols generated during firefighting activities [61–64]. Additional exposure may occur through contact with protective garments treated with fluoropolymers and side-chain fluorinated polymers, which impart water- and oil-repellent properties [65,66]. Moisture barriers often contain polytetrafluoroethylene (PTFE), a PFAS polymer designed to prevent penetration of water and biological fluids [67,68]. Studies have further demonstrated that firefighters trained with aqueous film-forming foams show measurable increases in serum PFHxS and PFNA concentrations immediately following exposure [69]. Therefore, occupational exposure may be added to background environmental exposure [61,66,68,70] increasing consistently the PFAS total burden. In literature, besides firefighters, occupational exposure has also been reported in ski wax technicians and workers in manufacturing plants [71,72]. Studies were conducted in different time periods and locations, and a comprehensive evaluation among workers is challenging due to changes in PFAS production and use, with some compounds being phased out and replaced by others. In this context, updated studies are needed to characterize PFAS exposure in contemporary occupational settings.

To date, no data are available in literature regarding Italian firefighters' exposure to PFAS. The aim of the present study was to conduct a cross-sectional observational study to assess PFAS exposure among firefighters operating in nine provinces of central and northern Italy, with the objective of evaluating plasma concentrations and comparing the results with demographic, clinical, lifestyle, and occupational data. Data from this study were compared with findings from other published investigations, including general population in the same local area, community residents exposed to PFOA-contaminated drinking water [73] and occupationally exposed workers [64,74]. Finally, a revision of the data according to NASEM thresholds has been performed.

2. Materials and methods

This study was approved by the Bioethical Committee of the University of Bologna (protocols number: 7486, 08/01/2025).

2.1. Materials

Analytes and isotopically labelled standards (>98 purity) were purchased from Wellington Laboratories (Guelph, Canada). The PFAS chosen for the analysis in this study were 24 molecules among the 25 listed in the U.S. Environmental Protection Agency (EPA) regulatory method n. 533 (EPA 2025, ref [75]). Native analytes included perfluoro-*n*-butanoic acid (PFBA); perfluoro-*n*-pentanoic acid (PFPeA); perfluoro-*n*-hexanoic acid (PFHxA); perfluoro-*n*-heptanoic (PFHpA); perfluoro-*n*-octanoic acid (PFOA); perfluoro-*n*-nonanoic acid (PFNA); perfluoro-*n*-decanoic acid (PFDA); perfluoro-*n*-undecanoic acid (PFUnDA; also referred to as PFUnA); perfluoro-*n*-dodecanoic acid (PFDoA); tetrafluoro-2-heptafluoropropoxy propanoic acid (HFPO-Da); perfluoro-3-methoxypropanoic acid (PFMPA); perfluoro-4-methoxybutanoic acid (PFMBA); potassium-perfluoro-1-butanesulfonate (PFBS); sodium perfluoro-1-pentanesulfonate (PFPeS); potassium perfluorohexanesulfonate (PFHxS); sodium perfluoro-1-heptanesulfonate (PFHpS); potassium perfluoro-1-octanesulfonate (PFOS); sodium dodecafluoro-3H-4,8-Dioxanonanoate (DONA); 1H,1H, 2H,2H-perfluoro-1-hexane sufonate (4:2 FTS); 1H,1H, 2H,2H-perfluoro-1-octanesufonate (6:2 FTS); 1H,1H, 2H,2H-perfluoro-1-decanesufonate (8:2 FTS); potassium 9-chlorohexadecafluoro-3-oxanonane-1-sulfonate (9Cl-PF3ONS); potassium 11-chloroeicosafluoro-3-oxaundecane-1-sulfonate (11Cl-PF3OUdS); potassium perfluoro (2-ethoxyethane) sulfonate (PFEESA). Nonafluoro-3,6-dioxaheptanoic acid (NFDHA) was not included in the list of analytes, because it did not satisfy the sensibility and reproducibility criteria for validation. Isotopically labelled internal standards (surrogates) were added to the sample before extraction in a known amount to compensate for analyte losses and matrix effects during the extraction process. Since not all target PFAS have an isotopically labelled analogue, an alternate labelled compound is used as recommended in EPA-533 method (see Table S1 in supplementary material). Isotopically labelled standards were PFBA $^{13}C_4$ (MPFBA) used to quantify PFBA and PFMPA; PFPeA $^{13}C_5$ (M5PFPeA) used to quantify PFPeA and PFMBA; PFHxA $^{13}C_5$ (M5PFHxA) used to quantify PFHxA; PFHpA $^{13}C_4$ (M4PFHpA) used to quantify PFHpA and ADONA; PFOA $^{13}C_8$(M8PFOA) used to quantify PFOA; PFNA $^{13}C_9$ (M9PFNA) used to quantify PFNA; $^{13}C_6$PFDA (M6PFDA) used to quantify PFDA, PFUnA $^{13}C_7$(M7PFUnA) used to quantify PFUnA; PFDoA $^{13}C_2$ (MPFDoA) used to quantify PFDoA; HFPO-DA $^{13}C_3$ (M3HFPO-DA) used to quantify HFPO-DA, PFBS $^{13}C_3$ (M3PFBS) used to quantify PFBS and PFEESA; PFHxS $^{13}C_3$(M3PFHxS) used to quantify PFHxS and PFPeS; PFOS $^{13}C_8$ (M8PFOS) used to quantify PFOS, 9Cl-PF3ONS, 11Cl-PF3OUdS and PFHpS; 4:2FTS $13C_2$(M2-4:2FTS) used to quantify 4:2 FTS; 6:2 FTS $13C_2$ (M2-6:2FTS) used to quantify 6:2 FTS and 8:2 FTS $13C_2$ (M2-8:2FTS) used to quantify 8:2 FTS.

Mass-labelled perfluoro-n- [2,3,4 $^{13}C_3$] butanoic acid (M3PFBA); perfluoro-n-[1,2-$^{13}C_2$] octanoic acid (M2PFOA), and

mass-labelled sodium perfluoro-1-[1,2,3,4-$^{13}C_4$] octanesulfonate (MPFOS) at chemical purities > 98% and isotopic purities of ≥ 99% were used as injection standards. They were added to the purified extracts before injection to monitor instrument stability and provide a reference for the precision of the injection.

Target perfluoroalkyl analytes, surrogate mass-labelled compounds and injection standards were at the following concentration: 500 ng/ml, 500-2000 ng/ml, 1000-3000 ng/ml. Working solution of surrogate mass-labelled compounds and injection standards solutions were prepared at the concentration of 25-100 ng/ml and 100-300 ng/ml, respectively, in methanol and stored at −20°C.

Methanol, acetonitrile and formic acid 98–100% (all LC–MS grade) were supplied by Merck (Darmstadt, Germany). Ultrapure water (18.2 MΩ cm resistivity) for mobile phase and sample preparation was produced by Sartorius Arium mini apparatus (Sartorius, Goettingen, Germany). Ammonium acetate was provided by Sigma-Aldrich (S.Louis, MO, USA). Human blank serum to experimentally check LOQ and MDL was acquired by Merck (Darmstadt, Germany).

2.2. Data collection

The volunteers were recruited throughout the provinces of Emilia-Romagna region (Bologna, Ferrara, Forlì-Cesena, Modena, Parma, Piacenza, Ravenna, Reggio Emilia, Rimini) and from the province of Arezzo, Tuscany. Sampling followed a convenience approach based on voluntary participation, with study invitations distributed through official regional and provincial commands. The enrolled personnel included different professional qualifications and operational roles. Although volunteer firefighters were eligible to join the study, they represented a minor fraction of the final cohort (n = 5, 1.3%), reflecting the predominantly permanent composition of the workforce in the involved commands.

An anamnestic questionnaire was administered to each participant to collect general information (e.g., age, sex, weight, food consumption), medical history (e.g., known chronic pathologies), and occupational-related data (e.g., qualification, years of service, current duty station). The complete questionnaire is reported in Table S4 (see supplementary material). Plasma samples were then obtained from non-fasting volunteers by collecting whole blood in EDTA containing tubes after venipuncture from the antecubital vein. Procedural blank samples were obtained by using water as a test matrix collected into vacutainer tubes and processed as samples. Plasma samples were stored in freezer at −20°C until analysis.

2.3. Sample preparation and analysis

Sample preparation and analysis followed the method previously published [76]. Briefly, aliquots of 200 μL of plasma samples were spiked with 2,5 μL of an isotopically labelled standard working solution (surrogate mass-labelled compounds, final concentration 6.25-25 ng/ml). Protein precipitation was performed by adding 400 μL of 1% formic acid in acetonitrile to each sample, followed by immediate and vigorous vortex-mixing for 30 s. Samples were centrifuged for 10 min at 14000 rpm. The supernatant was diluted 1:2 (v/v) with ultrapure water (18.2 MΩ cm, Sartorius Arium mini). Injection standards (2 μL, final concentration 3.3-10 ng/ml)) were added automatically by the autosampler's injection program during the sample's injection into the LC-MS/MS system. QC samples and calibration standards were prepared following the same procedure described for the plasma samples. QC samples were prepared for each analyte in plasma. Quantification was performed by isotope dilution analysis; calibration curves were prepared at concentrations of 0.1, 0.2, 0.5, 1, 2.5, 3, 5, and 10 ng/ml for each analyte. Analytical calibration curves were evaluated applying a 1/x weighting factor without forcing through the origin, exhibiting high linearity ($R^2 = 0.990$–0.999) across linear (PFUnDA, PFOS, PFHxS, PFHpS, PFHpA, PFDoA, 9-Cl-PF3ONS) and quadratic models for the remaining target congeners. Method Detection Limits (MDL) and Limits of Quantification (LOQ) ranged between 0.01 and 0.39 ng/mL and 0.04 – 1.20 ng/mL, respectively. Procedural blanks showed no detectable target PFAS levels above the LOQ. Method validation demonstrated high accuracy (bias < 16%), recoveries of 86–102%, and matrix effects below 30% for all analytes. Dilution integrity test (up to 1:10) maintained accuracy within 20% of theoretical values. Chromatographic conditions successfully separated linear and branched isomers of PFOS, PFOA, and PFHxS; these were co-integrated and quantified as total concentration. Ongoing precision and accuracy during routine sample analysis were strictly ensured by including QC samples (spiked matrix), procedural blanks, and injection standards alongside calibrators in each analytical run, maintaining both intra- and inter-day precision (RSD%) strictly below 10%.

Analyses were carried out in a LC-MS/MS system composed of 1290 Infinity II coupled to a 6495-triple quadrupole (Agilent Technologies, Santa Clara, USA) via ESI source. Chromatographic separations were achieved in a Poroshell EC-C18 column (2.1 × 100 mm, 1.9 μm), (Agilent Technologies) at 50°C. The mobile phase consisted of 10 mM ammonium acetate in water (mobile phase A) and 10 mM ammonium acetate in 75% acetonitrile 25% methanol (mobile phase B). The flow rate was set at 0.3 mL/min. The optimized gradient was as follow: 10% B at time 0 min; 30% B at 1 min, 95% B at 16 min, 10% B at 18 min, 10% B at 21, post run up to 25 min. The injection volume was optimized to 5 μL. Mass spectrometer operated in negative acquisition mode and acquisition was performed in multiple reaction monitoring (MRM) (see Tables S1–3 in supplementary material for details). Data analysis was carried out by MassHunter software (version 12.0, Agilent Technologies).

2.4. Statistical analysis

Categorical variables were summarized as counts and percentages, while PFAS concentrations were reported as mean ± SD, median, interquartile range (IQR), and full range (min–max). Values reported as non-detected or below the relevant analytical quantification threshold (LOQ) were coded as 0 ng/mL in the statistical dataset and retained as such for descriptive summaries, calculation of total PFAS concentrations, detection-frequency estimates, and inferential analyses. Detection frequency was defined as the proportion of samples with concentrations >0 ng/mL. Continuous questionnaire variables were categorized for descriptive and inferential analyses: age and years since last transfer were dichotomized at the median, and body mass index (BMI) was classified according to WHO guidelines.

Group differences in total plasma PFAS levels were assessed using the Wilcoxon test for two groups or the Kruskal–Wallis test for more than two groups, due to the presence of extreme outliers. Analyses were repeated focusing on characteristics significantly associated with total PFAS levels ($p \leq 0.05$) and on individual PFAS compounds detected in ≥ 5% of the sample.

As a sensitivity analysis, all non-demographic variables that were significantly associated with total plasma PFAS levels in the primary analysis were re-evaluated using age- and sex-adjusted rank-based linear regression models [77]. Total plasma PFAS concentrations were replaced by their sample ranks, and separate models were fitted for each predictor of interest, including continuous age and sex as adjustment variables. Global p-values

for categorical predictors were obtained using Wald tests based on heteroskedasticity-robust standard errors.

To account for multiple testing, p-values from the primary analyses and from the age- and sex-adjusted sensitivity analyses were adjusted using the Simes–Benjamini–Hochberg procedure to control the false discovery rate [78,79].

All analyses were performed using Stata 18 (StataCorp. 2023. Stata Statistical Software: Release 18. College Station, TX: StataCorp LLC), with a two-sided significance level of 0.05. Missing values for categorical variables were handled by including a dedicated "missing" category.

3. Results

3.1. Population data

A total of 378 firefighters were recruited for this study from nine headquarters across Italy. Participants were evenly distributed among the sites, and a graphical representation is provided in Fig. S1 (see supplementary material). Participants' characteristics were categorized by age group, sex, body mass index, living environment, dietary habits (organic, processed, and fish consumption), smoking status, and use of waterproof clothing. Occupational variables included permanent or volunteer service, job qualification, years of service, and mobility across headquarters. Chronic diseases were also recorded, although only 16% of participants reported any condition. In brief, half of the participants were younger than 50 years (n = 191, 50.5%; mean = 48.5 years; SD = 7.9; median = 49; range = 26–66), and 96.8% were male, most of whom lived in an urban environment (n = 285, 75.4%). Occasional consumption of organic, processed, and fish products was reported by 70%, 68%, and 75% of participants, respectively. Regular use of waterproof clothing was reported by 43.4% of volunteers.

Tobacco cigarette use was reported by 85% of participants, while chronic pathologies were present in only 16% of cases; however, no further details were collected.

The study population was mainly composed of individuals in permanent service positions (n = 373, 98.7%), with nearly half having more than 20 years of service (n = 182, 48.1%). Only 24.9% reported a recent transfer from another headquarters, most of which had occurred less than three years before the present study.

A thorough report of the result is provided in Table 1.

3.2. PFAS results

Among the 24 PFAS included in this study, 12 were detected with a detection frequency in the range 0.3%-100% (see Table 2). Only 5 congeners showed occurrence above 40%, namely PFBA (43.4%), PFHxS (76.5%), PFOA (99.7%), PFNA (57.9%) and PFOS (100%). Mean and median values were higher for PFOA (1.27 ng/ml; 1.10 ng/ml) and PFOS (4.34 ng/ml; 3.71 ng/ml). Maximum concentrations were observed for PFOS (24.7 ng/mL), PFOA (5.80 ng/ml), and PFHxS (3.72 ng/mL). According to the National Academies of Sciences, Engineering, and Medicine (NASEM), the summed concentration of seven PFAS (MeFOSAA, PFHxS, PFOA, PFOS, PFDA, PFUnDA, and PFNA) can be interpreted using concentration-based thresholds for clinical care and health-risk assessment. In particular, plasma or serum concentrations lower than 2 ng/mL are considered not harming for human health ("*Adverse health effects not expected*"); concentrations ranging between 2 and 20 ng/mL are considered potentially related to adverse effects ("*Potential for adverse health effects, especially for sensitive populations*"); concentrations higher than 20 ng/mL are considered harming for human health ("*Increased risk of adverse health effects*") ([13]. Chapter: 5). In our analytical panel, MeFOSAA was not included because it is not part of EPA Method 533. Therefore, for the purpose of comparison with the NASEM thresholds, we calculated the NASEM-comparable total PFAS concentration as the sum of the six NASEM-listed compounds quantified in this study: PFHxS, PFOA, PFOS, PFDA, PFUnDA, and PFNA. This NASEM-comparable sum, rather than the sum of all detected PFAS, was used to classify participants according to the <2 ng/ml, 2–20 ng/ml, and >20 ng/mL NASEM categories. Most of the participants of our study (n = 341, 90.2%) had total plasma PFAS concentrations between 2 and 20 ng/ml, while 8.2% (n = 31) exhibited values below 2 ng/ml. A small proportion (n = 6, 1.6%) showed concentrations exceeding 20 ng/ml. In the overall sample, the mean total plasma concentration of all detected PFAS was 7.26 ng/mL (SD = 4.58), with a median of 6.18 ng/mL and a range of 0.42–34.85 ng/mL. PFAS data were not uniformly distributed, as significant differences were observed across the analyzed variables.

A heterogeneous statistically significative distribution was observed across the different headquarters, with higher mean values detected among firefighters serving in coastal stations (mean = 6.17–10.51 ng/mL) compared to those from inland areas (mean = 5.31–6.88 ng/mL (see Fig. S2 in supplementary material).

Significantly higher mean concentrations were observed among participants aged over 50 years (mean = 8.33 ng/mL, SD = 5.39) and in males, although this group was overrepresented (mean = 7.37 ng/mL, SD = 4.60). Consistent with the effect of age, a service duration of more than 20 years was also statistically associated with increased PFAS concentrations (mean = 8.46 ng/mL, SD = 5.27).

Lifestyle habits were also considered, particularly those related to diet, environmental exposure, and health conditions. Participants who reported a higher frequency of fish and seafood consumption, which accounted for almost 98% of interviewed, and frequent use of waterproof clothing exhibited significantly higher PFAS concentrations, respectively mean = 8.66 ng/mL, SD = 6.10 and mean = 7.96 ng/mL, SD = 5.26.

Only a small proportion of participants reported never consuming processed foods (n = 65, 17.2%), and this group showed higher PFAS levels (mean = 8.85 ng/mL, SD = 6.17) than those who reported frequent consumption of processed foods (n = 55, 14.6%; mean = 6.51 ng/mL, SD = 4.20). This finding is noteworthy, as it appears to contrast with most of the existing literature and with our a priori expectations regarding processed food as a source of PFAS exposure. However, this result should be interpreted with caution, as it may be affected by a bias in the representativeness of the data.

No statistically significant differences emerged associated with BMI, urban or rural environment, smoking habits, presence of chronic pathologies nor exclusive selection of organic food consumption.

In the age- and sex-adjusted sensitivity analysis based on rank-transformed total plasma PFAS concentrations, the association with fire station remained highly significant after FDR correction (Table 4). Fish and seafood consumption, type of service, and qualification also remained associated with total PFAS levels, whereas the associations with processed food consumption, use of waterproof clothing outside the workplace, and years of service were attenuated after adjustment. These findings indicate that the geographic pattern was not explained by the age and sex distribution of the cohort, while some occupational indicators partly overlapped with age-related cumulative exposure.

Evaluation of individual PFAS in relation to the investigated variables was feasible only for those compounds with a detection frequency greater than 9%. The overall pattern of associations is

Table 1
Sample characteristics and distribution of total plasma PFAS levels by sociodemographic, behavioral, and occupational variables.

	n (%)	PFAS (ng/mL)[b]			*p*-value
		Mean [SD]	Median [IQR]	Min-Max	
All	378 (100.0%)	7.26 [4.58]	6.18 [4.43-9.22]	0.42-34.85	———
Fire station					<0.001[d]
Parma and Piacenza	46 (12.2%)	5.38 [2.14]	5.28 [4.20-6.18]	1.48-11.92	
Reggio Emilia	36 (9.5%)	5.81 [2.51]	5.63 [4.08-6.93]	1.57-15.49	
Modena	39 (10.3%)	5.31 [3.38]	5.05 [2.49-6.61]	0.42-15.69	
Bologna	60 (15.9%)	5.45 [3.50]	5.01 [2.77-6.87]	0.51-15.25	
Ferrara	22 (5.8%)	9.06 [3.68]	9.19 [6.42-11.40]	3.21-17.52	
Ravenna	36 (9.5%)	10.51 [6.17]	8.73 [6.32-13.01]	1.33-30.54	
Forlì-Cesena	50 (13.2%)	9.43 [6.00]	8.59 [5.96-11.04]	1.42-34.85	
Rimini	37 (9.8%)	9.37 [4.86]	8.62 [6.26-10.88]	2.64-25.86	
Arezzo	52 (13.8%)	6.88 [3.62]	6.16 [4.44-8.27]	1.94-21.94	
Age					<0.001[d]
<50 years	191 (50.5%)	6.22 [3.33]	5.52 [3.94-7.63]	1.15-21.94	
≥50 years	187 (49.5%)	8.33 [5.39]	6.84 [4.77-10.88]	0.42-34.85	
Sex					0.002[d]
Male	366 (96.8%)	7.37 [4.60]	6.19 [4.47-9.36]	0.42-34.85	
Female	12 (3.2%)	3.93 [2.41]	3.44 [1.71-6.13]	1.10-7.44	
***Body mass index* (kg/m^2)**					0.194
<25.0	168 (44.4%)	7.72 [4.80]	6.41 [4.56-10.36]	0.51-28.46	
≥25.0 and < 30.0	183 (48.4%)	6.98 [4.38]	6.16 [4.36-8.75]	0.42-34.85	
≥30.0	27 (7.1%)	6.33 [4.45]	6.11 [3.43-7.51]	1.86-25.09	
Living environment					0.287
Rural	93 (24.6%)	7.59 [4.60]	6.65 [4.77-9.85]	1.15-28.46	
Urban	285 (75.4%)	7.15 [4.58]	6.11 [4.35-9.07]	0.42-34.85	
Organic food consumption					0.404
Never	33 (8.7%)	7.47 [6.69]	5.42 [3.62-7.33]	1.57-30.54	
Occasionally	266 (70.4%)	7.25 [4.56]	6.18 [4.21-9.37]	0.42-34.85	
Often	79 (20.9%)	7.22 [3.56]	6.44 [5.18-9.26]	0.51-18.49	
Processed food consumption					0.036[d]
Never	65 (17.2%)	8.85 [6.17]	6.73 [5.31-11.41]	1.15-34.85	
Occasionally	258 (68.3%)	7.02 [4.10]	6.18 [4.44-8.62]	0.51-30.54	
Often	55 (14.6%)	6.51 [4.20]	5.26 [3.08-9.07]	0.42-17.57	
Fish and seafood consumption					0.019[d]
Never	7 (1.9%)	4.45 [1.92]	5.14 [3.77-5.49]	0.51-6.38	
Occasionally	285 (75.4%)	6.91 [3.97]	6.11 [4.27-9.05]	0.42-28.46	
Often	86 (22.8%)	8.66 [6.10]	6.70 [5.11-11.02]	1.78-34.85	
Cigarette smoking					0.537
No	321 (84.9%)	7.24 [4.37]	6.19 [4.44-9.36]	0.42-34.85	
Yes	57 (15.1%)	7.38 [5.70]	5.84 [4.12-8.75]	1.57-30.54	
Use of waterproof clothing outside the workplace					0.036[d]
No	214 (56.6%)	6.73 [3.92]	6.00 [4.20-8.38]	0.42-28.46	
Yes	164 (43.4%)	7.96 [5.26]	6.69 [4.54-10.37]	0.51-34.85	
Known chronic diseases					
No	318 (84.1%)	7.21 [4.41]	6.18 [4.44-9.21]	0.51-30.54	
Yes	60 (15.9%)	7.54 [5.43]	6.24 [4.25-9.41]	0.42-34.85	
Type of service					<0.001[d]
Permanent	373 (98.7%)	7.33 [4.57]	6.19 [4.44-9.26]	0.51-34.85	
Volunteer	5 (1.3%)	2.18 [1.78]	1.78 [1.15-2.49]	0.42-5.05	
Qualification[a]					<0.001[d]
Senior fire officer	10 (2.6%)	10.08 [6.91]	7.27 [6.31-10.80]	6.16-28.46	
Fire officer	67 (17.7%)	8.78 [5.36]	7.04 [5.74-11.34]	1.39-30.54	
Non-commissioned officer (NCO)	162 (42.9%)	7.33 [4.48]	6.57 [4.54-9.22]	0.51-34.85	
Senior firefighter	88 (23.3%)	6.29 [3.50]	5.52 [3.87-7.37]	1.57-18.49	
Firefighter	21 (5.6%)	5.31 [3.40]	4.50 [3.21-5.90]	1.10-13.54	
Technical specialist	23 (6.1%)	7.97 [4.69]	6.58 [4.02-11.43]	1.94-17.57	
Administrative staff	2 (0.5%)	4.43 [4.26]	4.43 [1.42-7.44]	1.42-7.44	
Volunteer	5 (1.3%)	2.18 [1.78]	1.78 [1.15-2.49]	0.42-5.05	
Years of service					<0.001[d]
1-5 years	22 (5.8%)	5.84 [3.57]	4.96 [4.04-6.16]	1.15-14.06	
6-20 years	174 (46.0%)	6.19 [3.50]	5.53 [3.80-7.63]	0.42-21.94	
>20 years	182 (48.1%)	8.46 [5.27]	6.88 [5.21-11.01]	0.51-34.85	
Current station of service					0.404
Headquarters	250 (66.1%)	7.39 [4.59]	6.21 [4.43-9.44]	0.51-30.54	
Branch station	128 (33.9%)	7.01 [4.59]	6.12 [4.44-8.62]	0.42-34.85	

(*continued on next page*)

Table 1 (*continued*)

	n (%)	PFAS (ng/mL)[b]			*p*-value
		Mean [SD]	Median [IQR]	Min-Max	
Recent transfer from another station					0.200
No	284 (75.1%)	7.12 [4.33]	6.00 [4.36-8.97]	0.42-28.46	
Yes	94 (24.9%)	7.69 [5.27]	6.78 [4.70-10.09]	0.51-34.85	
Years since transfer from another station[c]					0.742
≤3	57 (60.6%)	7.79 [5.31]	6.92 [4.73-9.52]	0.51-34.85	
>3	37 (39.4%)	7.54 [5.27]	6.58 [4.70-10.09]	1.19-30.54	

Abbreviations: SD, standard deviation; IQR, interquartile range.
Differences between groups were assessed using the Mann–Whitney *U* test or the Kruskal–Wallis test, depending on the groups sample size (two vs. more than two, respectively).
[a] The "Senior fire officer" category included the highest-ranking positions within the organizational structure, such as directors and senior managers. The "Fire officer" category comprised station officers, coordinators, and other personnel with operational supervisory roles. "Non-commissioned officers" included crew leaders, deputy crew leaders, and firefighters with coordination duties. The "Senior firefighter" category included firefighters with substantial experience or advanced qualifications. "Firefighters" represented the basic operational level of the permanent corps. Individuals with specific professional roles (e.g., instructors, technicians, logistics personnel, or administrative staff with technical duties) were classified as "Technical specialists". A separate category was used for "Administrative staff", while "Volunteer firefighters" included members of the corps who were not part of the permanent workforce.
[b] In this table, the term PFAS refers to all the analytes which concentration was higher than the LOQ.
[c] Total n = 94.
[d] $p \leq 0.05$.

Table 2
Overall distribution of each PFAS molecule (unstratified).

	Mean [SD]	Median [IQ range]	Min.-Max.	% >0 ng/mL
PFBA	0.49 [1.20]	0.00 [0.00-0.60]	0.00-14.00	43.4%
PFMPA	0.00 [0.00]	0.00 [0.00-0.00]	0.00-0.00	0.0%
PFPeA	0.00 [0.01]	0.00 [0.00-0.00]	0.00-0.20	0.3%
PFMBA	0.00 [0.00]	0.00 [0.00-0.00]	0.00-0.00	0.0%
4:2 FTS	0.00 [0.00]	0.00 [0.00-0.00]	0.00-0.00	0.0%
PFBS	0.00 [0.00]	0.00 [0.00-0.00]	0.00-0.00	0.0%
PFHxA	0.00 [0.00]	0.00 [0.00-0.00]	0.00-0.00	0.0%
PFEESA	0.00 [0.00]	0.00 [0.00-0.00]	0.00-0.00	0.0%
PFPeS	0.00 [0.00]	0.00 [0.00-0.00]	0.00-0.00	0.0%
PFHpA	0.00 [0.02]	0.00 [0.00-0.00]	0.00-0.27	1.3%
ADONA	0.00 [0.00]	0.00 [0.00-0.00]	0.00-0.00	0.0%
6:2 FTS	0.00 [0.03]	0.00 [0.00-0.00]	0.00-0.60	0.3%
PFHxS	0.71 [0.59]	0.65 [0.50-0.97]	0.00-3.72	76.5%
PFOA	1.27 [0.79]	1.10 [0.75-1.56]	0.00-5.80	99.7%
PFHpS	0.01 [0.07]	0.00 [0.00-0.00]	0.00-1.00	0.8%
PFNA	0.31 [0.35]	0.31 [0.00-0.52]	0.00-2.81	57.9%
8:2 FTS	0.00 [0.00]	0.00 [0.00-0.00]	0.00-0.00	0.0%
PFOS	4.34 [3.03]	3.71 [2.50-5.20]	0.20-24.70	100.0%
PFDA	0.08 [0.17]	0.00 [0.00-0.00]	0.00-1.30	22.5%
PFUnDA	0.04 [0.13]	0.00 [0.00-0.00]	0.00-1.00	9.5%
PFDoA	0.00 [0.00]	0.00 [0.00-0.00]	0.00-0.00	0.0%
9CL-PF3ONS	0.00 [0.02]	0.00 [0.00-0.00]	0.00-0.19	2.1%

Abbreviations: PFBA, perfluorobutanoic acid; PFMPA, perfluoromethoxypropanoic acid; PFPeA, perfluoropentanoic acid; PFMBA, perfluoromethoxybutanoic acid; 4:2 FTS, 4:2 fluorotelomer sulfonate; PFBS, perfluorobutanesulfonate; PFHxA, perfluorohexanoic acid; PFEESA, perfluoroethoxyethanesulfonate; PFPeS, perfluoropentanesulfonate; PFHpA, perfluoroheptanoic acid; ADONA, perfluorodimethoxyacetic acid (alternative PFOA replacement); 6:2 FTS, 6:2 fluorotelomer sulfonate; PFHxS, perfluorohexanesulfonate; PFOA, perfluorooctanoic acid; PFHpS, perfluoroheptanesulfonate; PFNA, perfluorononanoic acid; 8:2 FTS, 8:2 fluorotelomer sulfonate; PFOS, perfluorooctanesulfonate; PFDA, perfluorodecanoic acid; PFUnDA, perfluoroundecanoic acid; PFDoA, perfluorododecanoic acid; 9CL-PF3ONS 9-Chlorohexadecafluoro-3-Oxanone-1-Sulfonic Acid.

reported in Table 3. The most notable findings included: higher PFNA and PFUnDA levels among participants with higher fish consumption, higher PFNA and PFDA levels among those frequently wearing waterproof clothing, and associations of PFHxS, PFOS, PFNA, and PFOA with specific types of employment.

4. Discussion

This snapshot cross-sectional study provided the first characterization of PFAS plasma levels in a large cohort of Italian firefighters and offered novel insights into the contribution of occupational and lifestyle-related factors to the overall exposure burden. Data from this study were compared to national and international studies in general population, community residents in contaminated areas and workers usually considered occupationally exposed to PFAS. When compared to national general population, no appreciable differences were observed in detection frequency of PFOS, PFOA, and PFHxS in this population respect to the general population from the same geographical area [80]. Differences were mainly related to the type of congeners, where PFHxA and PFBS were not detected in our cohort. In both studies, PFOS was detected in 100% of cases, followed by PFOA and PFHxS, whereas PFNA, PFBA, and PFDA appeared to be more characteristic of the present investigation. One of the most comprehensive studies conducted on the U.S. general population on PFAS was carried out by NHANES [81] in a continuous survey conducted in two-year cycles by CDC's National Center for Health Statistics (NCHS) since 2000 up to 2018, which analyzed serum measurements from 5000 persons. The most recent data of 2017-2020 reported geometric mean for the most frequently detected PFAS, namely PFOS, PFOA, PFHxS and PFNA of respectively 3.93 ng/mL, 1.38 ng/mL, 0.46 ng/mL, showing a decreasing trend of 16-87% respect to 2000s. The most detected compounds in NHANES were PFOA (99.8%), PFOS (99.8%), PFHxS (99.2%) and 9CL-PF3ONS (7.98%). According to NHANES data, PFAS concentrations observed in the present study were broadly comparable to those reported in the general U.S. population, with PFOS emerging as the most abundant congener. In contrast, Borg et al. [74] reported markedly different concentration levels in the Swedish general population, with mean values of 27.5 ng/mL for PFOS, 5.2 ng/mL for PFOA, and 8 ng/mL for PFHxS. Due to these substantial differences, a direct comparison with our findings is not feasible. However, these discrepancies further support the notion that PFAS exposure is strongly influenced by geographic distribution and region-specific industrial activities.

In fact, considering national biomonitoring studies carried out in industry-contaminated areas, such as the nearby Veneto region, differences were observed for type and PFAS concentration, in particular for PFOA, PFOS and PFHxS. In fact, the study by Pitter et al. [73] distributions, markedly right skewed, reached values at 95th percentile of 190 ng/mL, 10.7 ng/mL and 18.1 ng/mL respectively, although median values could be comparable. Another study by Ingelido et al. [82] indicated that, in the Italian population with background environmental exposure, the median serum

Table 3
Associations between selected categories from the anamnestic questionnaire (Table 1) and the most relevant PFAS plasma concentrations.

	Station	Age	Sex	Processed food	Fish and seafood	Waterproof clothing	Type of service	Qualification	Years of service
PFBA	<0.001[a]	0.956	0.611	0.435	0.361	0.506	0.391	0.915	0.589
PFHxS	<0.001[a]	<0.001[a]	0.003[a]	0.336	0.164	0.356	0.035[a]	<0.001[a]	<0.001[a]
PFOA	<0.001[a]	<0.001[a]	0.004[a]	0.336	0.306	0.103	0.003[a]	<0.001[a]	<0.001[a]
PFNA	<0.001[a]	0.018[a]	0.413	0.284	0.003[a]	0.030[a]	0.044[a]	0.035[a]	0.018[a]
PFOS	<0.001[a]	0.001[a]	0.004[a]	0.106	0.095	0.054	<0.001[a]	<0.001[a]	<0.001[a]
PFDA	<0.001[a]	0.516	0.156	0.194	0.236	0.001[a]	0.648	0.800	0.635
PFUnDA	<0.001[a]	0.406	0.413	0.487	0.040[a]	0.054	1.000	0.800	0.834

Reported p-values were obtained using the Mann–Whitney *U* test or the Kruskal–Wallis test, depending on the number of groups. P-values were adjusted for multiple comparisons using the Simes–Benjamini–Hochberg procedure to control the false discovery rate (FDR).
[a] **$p \leq 0.05$**.

Table 4
Age- and sex-adjusted sensitivity analysis of the association between selected variables and total plasma PFAS concentrations.

Variable	*n*	Age- and sex-adjusted	FDR-adjusted
		p-value	*p*-value
Fire station	378	<0.001[a]	<0.001[a]
Processed food consumption	378	0.040[a]	0.055
Fish and seafood consumption	378	0.002[a]	0.003[a]
Waterproof clothing outside the workplace	378	0.196	0.229
Type of service	378	<0.001[a]	0.001[a]
Qualification	378	0.001[a]	0.002[a]
Years of service	378	0.396	0.396

Total plasma PFAS concentrations were rank-transformed. For each predictor, a separate linear regression model was fitted including the predictor of interest, continuous age, and sex. *P*-values correspond to global Wald tests based on heteroskedasticity-robust standard errors and were adjusted using the Simes–Benjamini–Hochberg procedure to control the false discovery rate.
$^{*}p \leq 0.05$.

concentration of PFOA was approximately 2.4 ng/mL, while the median concentration of PFOS was approximately 4.6 ng/mL. Comparable values have also been reported in specific subgroups, such as women of reproductive age, with median levels of PFOA of 1.55 ng/g and PFOS of 2.43 ng/g [83]. Notably, the latter study included participants from multiple Italian regions, reporting data related to different people, geographically distributed along the peninsula. Earlier investigations reported slightly higher median concentrations for legacy PFOA and PFOS, respectively at 3.59 ng/g and 6.31 ng/g, suggesting a general downward trend over time [84]. More specifically, median concentrations of the main legacy PFAS (PFOS, PFOA, PFHxS and PFNA) in our cohort fell within the means observed in these populations, although PFOA in the Veneto residents remained markedly elevated, reflecting the historical impact of local contamination.

Among occupations typically regarded as at risk for occupational PFAS exposure, our study considered data from large-scale surveys conducted in firefighters and ski wax workers. In this context, ski wax workers exhibited serum concentrations for all measured congeners (PFOA, PFOS, PFNA, PFDA, PFBA, PFHxS, and PFUnDA) that were consistently at least one order of magnitude higher than those observed in our cohort, with PFOS reaching mean levels of approximately 1070 ng/mL. The study by Rotander et al. [64] quantified serum concentrations of multiple PFAS in 149 Australian firefighters with previous occupational exposure to PFOS-based and telomer-based AFFF. PFOS, PFHxS, and PFOA were detected in all samples, with PFOS and PFHxS levels approximately 6–15 times higher than those reported for the Australian and Canadian general populations, whereas PFOA concentrations were comparable to background values. Serum PFOS ranged from 3 to 391 ng/mL (mean 74 ng/ml) and PFHxS from 1 to 277 ng/mL (mean 33 ng/ml), and concentrations of both compounds increased significantly with years in jobs involving foam contact and with age. Blood donation and female sex were associated with lower PFAS levels, while exposure indices derived from self-reported skin contact with foam did not predict serum concentrations. A representative comparison of PFAS concentration and occurrence across different communities and worker groups is shown in Figs. S3–5 (see supplementary material).

Compared with the Australian firefighter cohort studied by Rotander et al. [64], both detection frequencies and plasma concentrations in our study were substantially lower for all PFAS congeners. When considering firefighters activity, PFHxS represented a distinctive marker, being the second most frequently detected compound (100%) and occurring at higher concentrations (average 33 ng/mL). By contrast, in all general-population datasets considered here, alongside results from our study, PFOA consistently emerged as the second most abundant congener, both in terms of detection frequency and concentration. Studies conducted in relatively small firefighter cohorts (n = 8) [69] and (n = 116) [85] reported similar serum levels of PFHxS and PFOA, with PFOS consistently emerging as the most abundant congener; however, all PFAS concentrations in these populations were markedly lower than those described by Rotander et al. [64] in Australian AFFF-exposed firefighters.

Overall, the PFAS concentrations measured in our firefighter cohort were comparable to background levels observed in the general population, rather than showing the marked occupational elevation reported in earlier studies or in studies conducted in non-European countries, where firefighting activities may be organized differently. This discrepancy may also be largely driven by the regulatory landscape in Italy and the European Union, where strict regulations (such as EU Regulation 2019/1021) have progressively restricted and phased out legacy PFAS, particularly PFOS and PFOA, in firefighting foams (AFFF) and protective gear over the last two decades, significantly mitigating the primary occupational exposure pathways for this contemporary cohort.

A key finding of our study was the association between elevated PFAS concentrations and considered occupational variables, including assignment to coastal commands, longer service

duration, higher rank, and permanent employment status. Firefighters stationed in coastal areas exhibited markedly higher mean plasma levels compared to those from inland provinces, suggesting a combined contribution of occupational exposure and local environmental contamination, potentially mediated by water sources and seafood consumption.

The positive association with years of service and rank supports the concept of cumulative exposure, whereby prolonged employment in operational roles and sustained contact with contaminated equipment, fire scenes, and station environments contribute to PFAS bioaccumulation. As a matter of fact, firefighters with longer occupational careers are likely to have experienced greater cumulative exposure, as they were active during periods when aqueous film-forming foams (AFFF) containing PFAS were routinely used. More recently, the Italian National Fire Service implemented higher operational standards by restricting the use of synthetic AFFF agents to fires involving flammable liquids and introducing fluorine-free foam agents for suppression of fires involving solid combustible materials, thereby reducing the use of fluorinated firefighting foams where effective alternatives were available. Accordingly, firefighters with longer service histories are expected to have accumulated higher body burdens of PFAS over time, reflecting the persistence and bioaccumulative properties of these compounds following repeated occupational exposure. These findings were consistent with previous studies reporting increased levels of PFHxS, PFOS, and PFOA among firefighters, particularly among those with longer careers or more frequent involvement in foam-based training and firefighting activities [85]. However, the attenuation of the association with years of service after adjustment for age and sex suggests that years of service should be interpreted primarily as a correlated proxy of age-related cumulative occupational history rather than as an independent determinant. By contrast, the persistence of the association with qualification after adjustment supports the relevance of career-related occupational patterns in explaining PFAS burden.

Lifestyle and environmental factors also emerged as important contributors to PFAS exposure in this cohort. In line with the recognized role of diet, frequent fish and seafood consumption was associated with higher levels of several PFAS, including PFNA and PFUnDA, which are known to bioaccumulate in marine food. This finding was particularly relevant for firefighters working in coastal areas, where local dietary habits may amplify the impact of occupational exposure. The association between frequent use of hydro repellent garments and higher PFNA and PFDA concentrations supported concerns about fluorinated polymers and side-chain fluorinated coatings as a source of PFAS release from water- and oil-repellent textiles. These results were consistent with recent studies reporting measurable fluorine and PFAS content in technical clothing, including items not exclusively used in professional firefighting contexts.

An unexpected finding was the higher PFAS burden among individuals who reported never consuming processed foods compared with those declaring frequent intake of such products. This result contrasted with the hypothesis, and with several reports, that processed and packaged foods may act as relevant PFAS sources due to migration from contact materials. Several explanations could be hypothesized, including residual confounding by unmeasured dietary or environmental factors, misclassification of food categories, or differential reporting related to health-conscious behaviors. In particular, participants avoiding processed foods may rely more heavily on fresh fish or local products, which in some Italian regions have been documented as important PFAS exposure pathways. Given the small size and limited representativeness of this subgroup, these observations should be interpreted with caution and warrant further investigation in specifically designed studies.

Notably, no statistically significant associations were found between PFAS levels and BMI, smoking habits, living environment (urban versus rural), presence of chronic diseases, or exclusive consumption of organic foods. These results were partially consistent with previous published results, for evidence on the association between individual variables and PFAS levels was inconsistent. Indeed, a 2025 systematic review identified mixed associations between BMI and PFAS concentrations: among non-pregnant adult populations, associations for PFOA were 40% positive, 15% negative, and 45% null; for PFOS, 26.3% positive, 10.5% negative, and 63.2% null [86]. Some studies have reported positive associations between PFAS exposure and obesity, whereas others have found negative or null associations ([87]; Ding et al., 2005; [88–90]).

For what concerned smoking habits, several biomonitoring studies have found that non-smokers tend to exhibit higher PFAS levels than smokers. A Danish study conducted on 652 participants reported that non-smokers had significantly higher serum concentrations of PFOA and PFOS compared to current smokers [91]. Analogous findings have been observed in studies carried out in Korea and Italy [82,92]. However, in certain highly exposed populations, tobacco smoking has been identified as a significant determinant of serum PFAS levels [93].

Despite some inconsistencies in the available results, studies more often reported higher PFAS levels in populations living in urbanized settings, supporting the notion that urbanization could be a recurrent determinant of PFAS exposure in the scientific literature [82,94]. Any association regarding sex or consumption of organic food could not be considered for the limited number of cases in our population.

However, despite the substantial sample size and the huge of collected information, it remained challenging to disentangle the relative contributions of occupational exposures, residential environment, and individual lifestyle, as these determinants were often closely intertwined in firefighters' everyday lives. Most participants had PFAS concentrations below commonly cited potentially harmful ranges (i.e., <20 ng/mL), which can be regarded as reassuring from a health-risk perspective. At the same time, the inability to separate occupational exposures from lifestyle-related determinants underscores the importance of implementing regular biomonitoring programs, particularly for long-serving and operationally active personnel. Such programs would facilitate early identification of highly exposed individuals, support decisions on work organization, and guide interventions aimed at minimizing contact with known sources, including legacy foams and contaminated gear. On this basis, integrating PFAS assessment into existing health surveillance systems for firefighters appears justified, as it may enhance our understanding of long-term health outcomes potentially associated with chronic exposure and improve the targeting of preventive measures. Furthermore, it should be acknowledged that our study evaluated a targeted panel of 24 PFAS, out of the thousands currently in existence. Future biomonitoring efforts should therefore include broader analytical panels to assess emerging congeners and substitute substances, thereby ensuring a comprehensive evaluation of potential exposures to as-yet uncharacterized PFAS among Italian firefighters.

On one side, this study contributed to new data generation to the still limited body of evidence regarding PFAS exposure in Italian occupational settings and on the other side complemented previous investigations conducted in the general population and in other high-exposure groups, such as residents of contaminated areas. By situating firefighters within this broader exposure landscape, our results emphasized the interplay between environmental, occupational, and lifestyle determinants of PFAS

burden. The participation of firefighters from each provincial command represented the strength of the present study, as it allowed analysis on groups distributed across the entire territory of the Emilia-Romagna region, or at least across its main provinces. Expanding surveillance to additional regions, including appropriate control groups and longitudinal follow-up, will be essential to clarify temporal trends, refine risk estimates, and evaluate the effectiveness of emerging policies and mitigation strategies.

Despite the large study population, which placed this work among the largest cohorts investigated in firefighters, and the encouraging results that were broadly consistent with existing scientific literature, some limitations should nevertheless be acknowledged. A major challenge concerned the comparison of PFAS concentrations across published datasets, which variously reported values in whole blood, plasma, or serum; in the present work, no conversion factors were applied to harmonize other authors' data, potentially affecting the power of cross-study comparisons. In addition, differences in sampling periods between our cohort and previously published studies introduced heterogeneous temporal windows of observation, which may further complicate data interpretation, particularly in the context of evolving PFAS use and regulatory restrictions. Moreover, a possible sampling bias was present due to the recruitment procedures for participants' selection. Enrollment was conducted on a voluntary basis; therefore, the study population may not be fully representative of the target occupational group.

5. Conclusion

Per- and polyfluoroalkyl substances (PFAS) are widespread environmental contaminants associated with potential risks to human health. Exposure may arise from multiple sources; however, certain occupational groups appear to be particularly vulnerable, presenting an increased risk of accumulation and of developing related diseases. Among these groups, firefighters were widely recognized by the scientific community as a population at risk due to their concurrent exposure to a variety of hazardous chemicals, including PFAS.

The findings of this study confirmed the associations between PFAS exposure and specific risk factors, such as dietary habits and geographic location. Median PFAS concentrations observed in the study group were comparable to those reported in studies conducted on the general population, without marked differences. Nevertheless, 98% of participants exhibited plasma PFAS levels below ranges generally considered harmful to health.

Targeted biomonitoring protocols for fire service personnel would be auspicial with the aim of strengthening preventive measures and protecting occupational health. Biomonitoring, which has already proven effective in other high-risk occupational groups, may also expand current knowledge regarding exposure pathways and support the design of prospective studies capable of clarifying the causal relationship between PFAS exposure and the onset of specific diseases.

CRediT authorship contribution statement

Arianna Fornasari: Formal analysis, Data curation. **Michele Pozzebon:** Supervision, Software, Resources. **Simone Santelli:** Formal analysis, Data curation. **Giovanni Dal Lago:** Formal analysis, Data curation. **Carla Arniani:** Resources, Data curation. **Sofia Raniolo:** Software, Data curation. **Schiavonea Modesto:** Resources, Data curation. **Jacopo Lenzi:** Software, Methodology. **Paolo Fais:** Writing – review & editing, Writing – original draft, Validation, Supervision. **Jennifer P. Pascali:** Writing – review & editing, Software, Conceptualization.

Ethical Approval

This study was approved by the Bioethical Committee of the University of Bologna (protocols number: 7486, 08/01/2025).

Data availability statement

The data supporting the findings of this study are available from the corresponding author upon reasonable request, subject to institutional and ethical approval. Due to privacy and confidentiality restrictions, the data are not publicly available.

This study was approved by the Bioethical Committee of the University of Bologna (protocols number: 7486, 08/01/2025).

Declaration of generative AI and AI-assisted technologies in the writing process

During the preparation of this work, the authors used Perplexity to improve the readability, grammar, and language of the manuscript. After using this tool, the authors carefully reviewed and edited the content and take full responsibility for the final version of the manuscript.

Funding

No funding

Declaration of competing interests

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Acknowledgements

The authors gratefully acknowledge the support of Carla Arniani, Annamaria De Luca, and Michele Pozzebon for their valuable assistance in this work.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.emcon.2026.100725.

References

[1] J.A. Faust, PFAS on atmospheric aerosol particles: a review, Environ. Sci. Process. Impacts 25 (2) (2023) 133–150, https://doi.org/10.1039/d2em00002d.
[2] E. Kissa, Fluorinated surfactants and repellents, second edition revised and expanded, in: Surfactant Science Series 97, Marcel Dekker, New York (NY), 2001, p. 640.
[3] OECD, Toward a new comprehensive global database of per- and polyfluoroalkyl substances (PFASs): summary report on updating the OECD 2007 list of per- and polyfluoroalkyl substances (PFASs) (OECD Environment, Health and Safety Publications. Series on Risk Management No. 39), OECD Publishing, 2021. https://www.oecd.org/chemicalsafety/portal-perfluorinated-chemicals/. (Accessed 18 August 2025).
[4] U.S. EPA (Environmental Protection Agency), CompTox Chemicals Dashboard, 2025. Available at: https://www.epa.gov/comptox-tools/comptox-chemicals-dashboard. (Accessed 18 August 2025).
[5] M.G. Evich, M.J.B. Davis, J.P. McCord, B. Acrey, J.A. Awkerman, D.R.U. Knappe, A.B. Lindstrom, T.F. Speth, C. Tebes-Stevens, M.J. Strynar, Z. Wang, E.J. Weber, W.M. Henderson, J.W. Washington, Per- and polyfluoroalkyl substances in the environment, Science (New York, N.Y.) 375 (6580) (2022) eabg9065, https://doi.org/10.1126/science.abg9065.
[6] L. Ahrens, M. Shoeib, T. Harner, S.C. Lee, R. Guo, E.J. Reiner, Wastewater treatment plant and landfills as sources of polyfluoroalkyl compounds to the atmosphere, Environ. Sci. Technol. 45 (19) (2011) 8098–8105, https://doi.org/10.1021/es1036173.

[7] C.A. Barton, L.E. Butler, C.J. Zarzecki, J. Flaherty, M. Kaiser, Characterizing perfluorooctanoate in ambient air near the fence line of a manufacturing facility: comparing modeled and monitored values, J. Air Waste Manag. Assoc. 56 (1) (2006) 48–55, https://doi.org/10.1080/10473289.2006.10464429.
[8] J.E. Galloway, A.V.P. Moreno, A.B. Lindstrom, M.J. Strynar, S. Newton, A.A. May, L.K. Weavers, Evidence of air dispersion: HFPO-DA and PFOA in Ohio and West Virginia surface water and soil near a fluoropolymer production facility, Environ. Sci. Technol. 54 (12) (2020) 7175–7184, https://doi.org/10.1021/acs.est.9b07384.
[9] K. Prevedouros, I.T. Cousins, R.C. Buck, S.H. Korzeniowski, Sources, fate and transport of perfluorocarboxylates, Environ. Sci. Technol. 40 (1) (2006) 32–44, https://doi.org/10.1021/es0512475.
[10] Y. Tian, Y. Yao, S. Chang, Z. Zhao, Y. Zhao, X. Yuan, F. Wu, H. Sun, Occurrence and phase distribution of neutral and ionizable Per- and polyfluoroalkyl substances (PFASs) in the atmosphere and plant leaves around landfills: a case study in Tianjin, China, Environ. Sci. Technol. 52 (3) (2018) 1301–1310, https://doi.org/10.1021/acs.est.7b05385.
[11] I. Weinberg, A. Dreyer, R. Ebinghaus, Waste water treatment plants as sources of polyfluorinated compounds, polybrominated diphenyl ethers and musk fragrances to ambient air, Environ. Pollut. 159 (1) (2011) 125–132, https://doi.org/10.1016/j.envpol.2010.09.023.
[12] W. Dui, M.P. Smith, S.H. Bartock, Development, validation, and clinical assessment of a liquid chromatography-tandem mass spectrometry serum assay for per- and polyfluoroalkyl substances (PFAS) recommended by the National Academies of Science, Engineering, and Medicine (NASEM), Anal. Bioanal. Chem. 416 (28) (2024) 6333–6344, https://doi.org/10.1007/s00216-024-05519-y.
[13] NASEM, Guidance on PFAS testing and health outcomes, Available at: https://www.nationalacademies.org/projects/DELS-BEST-20-01, 2022.
[14] J.L. Domingo, M. Nadal, Human exposure to per- and polyfluoroalkyl substances (PFAS) through drinking water: a review of the recent scientific literature, Environ. Res. 177 (2019) 108648, https://doi.org/10.1016/j.envres.2019.108648.
[15] Y. Cao, C. Ng, Absorption, distribution, and toxicity of per- and polyfluoroalkyl substances (PFAS) in the brain: a review, Environ. Sci. Process. Impacts 23 (11) (2021) 1623–1640, https://doi.org/10.1039/d1em00228g.
[16] S.E. Fenton, A. Ducatman, A. Boobis, J.C. DeWitt, C. Lau, C. Ng, J.S. Smith, S.M. Roberts, Per- and polyfluoroalkyl substance toxicity and human health review: current state of knowledge and strategies for informing future research, Environ. Toxicol. Chem. 40 (3) (2021) 606–630, https://doi.org/10.1002/etc.4890.
[17] E. Piva, A. Giorgetti, P. Ioime, L. Morini, F. Freni, F.L. Faro, F. Pirani, M. Montisci, P. Fais, J.P. Pascali, Hair determination of per- and polyfluoroalkyl substances (PFAS) in the Italian population, Toxicology 458 (2021) 152849, https://doi.org/10.1016/j.tox.2021.152849.
[18] A. Cordner, G. Goldenman, L.S. Birnbaum, P. Brown, M.F. Miller, R. Mueller, S. Patton, D.H. Salvatore, L. Trasande, The true cost of PFAS and the benefits of acting now, Environ. Sci. Technol. 55 (14) (2021) 9630–9633, https://doi.org/10.1021/acs.est.1c03565.
[19] G.M. Sinclair, S.M. Long, O.A.H. Jones, What are the effects of PFAS exposure at environmentally relevant concentrations? Chemosphere 258 (2020) 127340 https://doi.org/10.1016/j.chemosphere.2020.127340.
[20] J.A. Bjork, C. Lau, S.C. Chang, J.L. Butenhoff, K.B. Wallace, Perfluorooctane sulfonate-induced changes in fetal rat liver gene expression, Toxicology 251 (1–3) (2008) 8–20, https://doi.org/10.1016/j.tox.2008.06.007.
[21] S. Chang, J.L. Butenhoff, G.A. Parker, P.S. Coder, J.D. Zitzow, R.M. Krisko, J.A. Bjork, K.B. Wallace, J.G. Seed, Reproductive and developmental toxicity of potassium perfluorohexanesulfonate in CD-1 mice, Reprod. Toxicol. 78 (2018) 150–168, https://doi.org/10.1016/j.reprotox.2018.04.007.
[22] J.C. DeWitt, W.C. Williams, N.J. Creech, R.W. Luebke, Suppression of antigen-specific antibody responses in mice exposed to perfluorooctanoic acid: role of PPARα and T- and B-cell targeting, J. Immunot. 13 (1) (2016) 38–45, https://doi.org/10.3109/1547691X.2014.996682.
[23] C. Lau, J.L. Butenhoff, J.M. Rogers, The developmental toxicity of perfluoroalkyl acids and their derivatives, Toxicol. Appl. Pharmacol. 198 (2) (2004) 231–241, https://doi.org/10.1016/j.taap.2003.11.031.
[24] C. Lau, K. Anitole, C. Hodes, D. Lai, A. Pfahles-Hutchens, J. Seed, Perfluoroalkyl acids: a review of monitoring and toxicological findings, Toxicol. Sci.: Off. J. Soc. Toxicol. 99 (2) (2007) 366–394, https://doi.org/10.1093/toxsci/kfm128.
[25] H. Li, S. Hammarstrand, B. Midberg, Y. Xu, Y. Li, D.S. Olsson, T. Fletcher, K. Jakobsson, E.M. Andersson, Cancer incidence in a Swedish cohort with high exposure to perfluoroalkyl substances in drinking water, Environ. Res. 204 (Pt C) (2022) 112217, https://doi.org/10.1016/j.envres.2021.112217.
[26] E. Mariussen, Neurotoxic effects of perfluoroalkylated compounds: mechanisms of action and environmental relevance, Arch. Toxicol. 86 (9) (2012) 1349–1367, https://doi.org/10.1007/s00204-012-0822-6.
[27] E. Panieri, K. Baralic, D. Djukic-Cosic, A. Buha Djordjevic, L. Saso, PFAS molecules: a major concern for the human health and the environment, Toxics 10 (2) (2022) 44, https://doi.org/10.3390/toxics10020044.
[28] A.M. Seacat, P.J. Thomford, K.J. Hansen, L.A. Clemen, S.R. Eldridge, C.R. Elcombe, J.L. Butenhoff, Sub-chronic dietary toxicity of potassium perfluorooctanesulfonate in rats, Toxicology 183 (1–3) (2003) 117–131, https://doi.org/10.1016/s0300-483x(02)00511-5.
[29] M. Stylianou, M.K. Björnsdotter, P.E. Olsson, I. Ericson Jogsten, J. Jass, Distinct transcriptional response of Caenorhabditis elegans to different exposure routes of perfluorooctane sulfonic acid, Environ. Res. 168 (2019) 406–413, https://doi.org/10.1016/j.envres.2018.10.019.
[30] J.R. Thibodeaux, R.G. Hanson, J.M. Rogers, B.E. Grey, B.D. Barbee, J.H. Richards, J.L. Butenhoff, L.A. Stevenson, C. Lau, Exposure to perfluorooctane sulfonate during pregnancy in rat and mouse. I: maternal and prenatal evaluations, Toxicol. Sci. : Off. J. Soc. Toxicol. 74 (2) (2003) 369–381, https://doi.org/10.1093/toxsci/kfg121.
[31] R. Attanasio, Sex differences in the association between perfluoroalkyl acids and liver function in US adolescents: analyses of NHANES 2013-2016, Environ. Pollut. 254 (Pt B) (2019) 113061, https://doi.org/10.1016/j.envpol.2019.113061.
[32] J. Bassler, A. Ducatman, M. Elliott, S. Wen, B. Wahlang, J. Barnett, M.C. Cave, Environmental perfluoroalkyl acid exposures are associated with liver disease characterized by apoptosis and altered serum adipocytokines, Environ. Pollut. 247 (2019) 1055–1063, https://doi.org/10.1016/j.envpol.2019.01.064.
[33] K.P. Das, C.R. Wood, M.T. Lin, A.A. Starkov, C. Lau, K.B. Wallace, J.C. Corton, B.D. Abbott, Perfluoroalkyl acids-induced liver steatosis: effects on genes controlling lipid homeostasis, Toxicology 378 (2017) 37–52, https://doi.org/10.1016/j.tox.2016.12.007.
[34] V. Gallo, G. Leonardi, B. Genser, M.J. Lopez-Espinosa, S.J. Frisbee, L. Karlsson, A.M. Ducatman, T. Fletcher, Serum perfluorooctanoate (PFOA) and perfluorooctane sulfonate (PFOS) concentrations and liver function biomarkers in a population with elevated PFOA exposure, Environ. Health Perspect. 120 (5) (2012) 655–660, https://doi.org/10.1289/ehp.1104436.
[35] J.A. Gleason, G.B. Post, J.A. Fagliano, Associations of perfluorinated chemical serum concentrations and biomarkers of liver function and uric acid in the US population (NHANES), 2007-2010, Environ. Res. 136 (2015) 8–14, https://doi.org/10.1016/j.envres.2014.10.004.
[36] X. Jiao, N. Liu, Y. Xu, H. Qiao, Perfluorononanoic acid impedes mouse oocyte maturation by inducing mitochondrial dysfunction and oxidative stress, Reprod. Toxicol. 104 (2021) 58–67, https://doi.org/10.1016/j.reprotox.2021.07.002.
[37] Y. Liao, J. Wang, Q.S. Huang, C. Fang, R. Kiyama, H. Shen, S. Dong, Evaluation of cellular response to perfluorooctane sulfonate in human umbilical vein endothelial cells, Toxicol. Vitro: Int. J. Publ. Assoc. BIBRA 26 (3) (2012) 421–428, https://doi.org/10.1016/j.tiv.2012.01.011.
[38] M.T. Martin, R.J. Brennan, W. Hu, E. Ayanoglu, C. Lau, H. Ren, C.R. Wood, J.C. Corton, R.J. Kavlock, D.J. Dix, Toxicogenomic study of triazole fungicides and perfluoroalkyl acids in rat livers predicts toxicity and categorizes chemicals based on mechanisms of toxicity, Toxicol. Sci.: Off. J. Soc. Toxicol. 97 (2) (2007) 595–613, https://doi.org/10.1093/toxsci/kfm065.
[39] M. Nian, Q.Q. Li, M. Bloom, Z.M. Qian, K.M. Syberg, M.G. Vaughn, S.Q. Wang, Q. Wei, M. Zeeshan, N. Gurram, C. Chu, J. Wang, Y.P. Tian, L.W. Hu, K.K. Liu, B.Y. Yang, R.Q. Liu, D. Feng, X.W. Zeng, G.H. Dong, Liver function biomarkers disorder is associated with exposure to perfluoroalkyl acids in adults: isomers of C8 Health Project in China, Environ. Res. 172 (2019) 81–88, https://doi.org/10.1016/j.envres.2019.02.013.
[40] Y. Qian, A. Ducatman, R. Ward, S. Leonard, V. Bukowski, N. Lan Guo, X. Shi, V. Vallyathan, V. Castranova, Perfluorooctane sulfonate (PFOS) induces reactive oxygen species (ROS) production in human microvascular endothelial cells: role in endothelial permeability, J. Toxicol. Environ. Health, Part A 73 (12) (2010) 819–836, https://doi.org/10.1080/15287391003689317.
[41] C.J. Sakr, K.H. Kreckmann, J.W. Green, P.J. Gillies, J.L. Reynolds, R.C. Leonard, 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. 49 (10) (2007) 1086–1096, https://doi.org/10.1097/JOM.0b013e318156eca3.
[42] X. Shi, B. Zhou, The role of Nrf2 and MAPK pathways in PFOS-induced oxidative stress in zebrafish embryos, Toxicol. Sci.: Off. J. Soc. Toxicol. 115 (2) (2010) 391–400, https://doi.org/10.1093/toxsci/kfq066.
[43] C.L. Souders 2nd, C.L. Sanchez, W. Malphurs, J.J. Aristizabal-Henao, J.A. Bowden, C.J. Martyniuk, Metabolic profiling in human SH-SY5Y neuronal cells exposed to perfluorooctanoic acid (PFOA), Neurotoxicology 85 (2021) 160–172, https://doi.org/10.1016/j.neuro.2021.05.009.
[44] K.R. Taibl, S. Schantz, M.T. Aung, A. Padula, S. Geiger, S. Smith, J.S. Park, G.L. Milne, J.F. Robinson, T.J. Woodruff, R. Morello-Frosch, S.M. Eick, Associations of per- and polyfluoroalkyl substances (PFAS) and their mixture with oxidative stress biomarkers during pregnancy, Environ. Int. 169 (2022) 107541, https://doi.org/10.1016/j.envint.2022.107541.
[45] M. Yamaguchi, K. Arisawa, H. Uemura, S. Katsuura-Kamano, H. Takami, F. Sawachika, M. Nakamoto, T. Juta, E. Toda, K. Mori, M. Hasegawa, M. Tanto, M. Shima, Y. Sumiyoshi, K. Morinaga, K. Kodama, T. Suzuki, M. Nagai, H. Satoh, Consumption of seafood, serum liver enzymes, and blood levels of PFOS and PFOA in the Japanese population, J. Occup. Health 55 (3) (2013) 184–194, https://doi.org/10.1539/joh.12-0264-oa.
[46] H.T. Wan, Y.G. Zhao, X. Wei, K.Y. Hui, J.P. Giesy, C.K. Wong, PFOS-induced hepatic steatosis, the mechanistic actions on β-oxidation and lipid transport, Biochim. Biophys. Acta 1820 (7) (2012) 1092–1101, https://doi.org/10.1016/j.bbagen.2012.03.010.
[47] L. Wang, Y. Wang, Y. Liang, J. Li, Y. Liu, J. Zhang, A. Zhang, J. Fu, G. Jiang, Specific accumulation of lipid droplets in hepatocyte nuclei of PFOA-exposed BALB/c mice, Sci. Rep. 3 (2013) 2174, https://doi.org/10.1038/srep02174.
[48] M. Wielsøe, M. Long, M. Ghisari, E.C. Bonefeld-Jørgensen, Perfluoroalkylated substances (PFAS) affect oxidative stress biomarkers in vitro, Chemosphere

129 (2015) 239–245, https://doi.org/10.1016/j.chemosphere.2014.10.014.
[49] M. Averina, J. Brox, S. Huber, A.S. Furberg, Exposure to perfluoroalkyl substances (PFAS) and dyslipidemia, hypertension and obesity in adolescents. The Fit Futures study, Environ. Res. 195 (2021) 110740, https://doi.org/10.1016/j.envres.2021.110740.
[50] A.J. Blomberg, Y.H. Shih, C. Messerlian, L.H. Jørgensen, P. Weihe, P. Grandjean, Early-life associations between per- and polyfluoroalkyl substances and serum lipids in a longitudinal birth cohort, Environ. Res. 200 (2021) 111400, https://doi.org/10.1016/j.envres.2021.111400.
[51] K.T. Eriksen, O. Raaschou-Nielsen, J.K. McLaughlin, L. Lipworth, A. Tjønneland, K. Overvad, M. Sørensen, Association between plasma PFOA and PFOS levels and total cholesterol in a middle-aged Danish population, PLoS One 8 (2) (2013) e56969, https://doi.org/10.1371/journal.pone.0056969.
[52] S.D. Geiger, J. Xiao, A. Ducatman, S. Frisbee, K. Innes, A. Shankar, The association between PFOA, PFOS and serum lipid levels in adolescents, Chemosphere 98 (2014) 78–83, https://doi.org/10.1016/j.chemosphere.2013.10.005.
[53] R.C. Jensen, M.S. Andersen, P.V. Larsen, D. Glintborg, C. Dalgård, C.A.G. Timmermann, F. Nielsen, M.B. Sandberg, H.R. Andersen, H.T. Christesen, P. Grandjean, T.K. Jensen, Prenatal exposures to perfluoroalkyl acids and associations with markers of adiposity and plasma lipids in infancy: an odense child cohort study, Environ. Health Perspect. 128 (7) (2020) 77001, https://doi.org/10.1289/EHP5184.
[54] P.M. Lind, S. Salihovic, B. van Bavel, L. Lind, Circulating levels of perfluoroalkyl substances (PFASs) and carotid artery atherosclerosis, Environ. Res. 152 (2017) 157–164, https://doi.org/10.1016/j.envres.2016.10.002.
[55] P.M. Lind, L. Lind, Are persistent organic pollutants linked to lipid abnormalities, atherosclerosis and cardiovascular disease? A review, J. Lipid Atherosclerosis 9 (3) (2020) 334–348, https://doi.org/10.12997/jla.2020.9.3.334.
[56] J. Yang, K. Zhang, C. Shen, P. Tang, S. Tu, J. Li, L. Chen, W. Yang, The association of hypertension with perfluoroalkyl and polyfluoroalkyl substances, Int. Heart J. 64 (6) (2023) 1079–1087, https://doi.org/10.1536/ihj.23-036.
[57] IARC, Int. Agency Res. Cancer Monograph 135 (2025). Available at: https://www.iarc.who.int/news-events/iarc-monographs-volume-135-perfluorooctanoic-acid-pfoa-and-perfluorooctanesulfonic-acid-pfos/.
[58] S. Mandal, N.U. Mazumder, R.J. Agnew, G. Song, R. Li, Characterization and modeling of thermal protective and thermo-physiological comfort performance of polymeric textile Materials-A review, Materials (Basel, Switzerland) 14 (9) (2021) 2397, https://doi.org/10.3390/ma14092397.
[59] N.-U.-S. Mazumder, Characterizing the Tensile Strength of Outer Layer Fabrics Used in Firefighters' Protective Clothing Under Radiant Heat Exposure, Oklahoma State University, Stillwater, OK, United States, 2021.
[60] N.U. Mazumder, S. Mandal, R.J. Agnew, A. Petrova, L.M. Boorady, G. Song, Characterizing the tensile strength of the fabrics used in firefighters' bunker gear under radiant heat exposure, Polymers 14 (2) (2022) 296, https://doi.org/10.3390/polym14020296.
[61] A.O. De Silva, J.M. Armitage, T.A. Bruton, C. Dassuncao, W. Heiger-Bernays, X.C. Hu, A. Kärrman, B. Kelly, C. Ng, A. Robuck, M. Sun, T.F. Webster, E.M. Sunderland, PFAS exposure pathways for humans and wildlife: a synthesis of current knowledge and key gaps in understanding, Environ. Toxicol. Chem. 40 (3) (2021) 631–657, https://doi.org/10.1002/etc.4935.
[62] N.U. Mazumder, M.T. Hossain, F.T. Jahura, A. Girase, A.S. Hall, J. Lu, R.B. Ormond, Firefighters' exposure to per-and polyfluoroalkyl substances (PFAS) as an occupational hazard: a review, Front. Mater. 10 (2023), https://doi.org/10.3389/fmats.2023.1143411.
[63] P.E. Rosenfeld, K.R. Spaeth, L.L. Remy, V. Byers, S.A. Muerth, R.C. Hallman, J. Summers-Evans, S. Barker, Perfluoroalkyl substances exposure in firefighters: sources and implications, Environ. Res. 220 (2023) 115164, https://doi.org/10.1016/j.envres.2022.115164.
[64] A. Rotander, L.M. Toms, L. Aylward, M. Kay, J.F. Mueller, Elevated levels of PFOS and PFHxS in firefighters exposed to aqueous film forming foam (AFFF), Environ. Int. 82 (2015) 28–34, https://doi.org/10.1016/j.envint.2015.05.005.
[65] B.J. Henry, J.P. Carlin, J.A. Hammerschmidt, R.C. Buck, L.W. Buxton, H. Fiedler, J. Seed, O. Hernandez, A critical review of the application of polymer of low concern and regulatory criteria to fluoropolymers, Integrated Environ. Assess. Manag. 14 (3) (2018) 316–334, https://doi.org/10.1002/ieam.4035.
[66] A.S. Young, E.H. Sparer-Fine, H.M. Pickard, E.M. Sunderland, G.F. Peaslee, J.G. Allen, Per- and polyfluoroalkyl substances (PFAS) and total fluorine in fire station dust, J. Expo. Sci. Environ. Epidemiol. 31 (5) (2021) 930–942, https://doi.org/10.1038/s41370-021-00288-7.
[67] M.T. Islam, M.M. Rahman, N.U.S. Mazumder, Polymers for textile production, in: Frontiers of Textile Materials: Polymers, Nanomaterials, Enzymes, and Advanced Modification Techniques, John Wiley & Sons, Ltd, New York, USA, 2020, pp. 13–59.
[68] D.J. Muensterman, L. Cahuas, I.A. Titaley, C. Schmokel, F.B. De la Cruz, M.A. Barlaz, C.C. Carignan, G.F. Peaslee, J.A. Field, Per- and polyfluoroalkyl substances (PFAS) in facemasks: potential source of human exposure to PFAS with implications for disposal to landfills, Environ. Sci. Technol. Lett. 9 (4) (2022) 320–326, https://doi.org/10.1021/acs.estlett.2c00019.
[69] J.A. Laitinen, J. Koponen, J. Koikkalainen, H. Kiviranta, Firefighters' exposure to perfluoroalkyl acids and 2-butoxyethanol present in firefighting foams, Toxicol. Lett. 231 (2) (2014) 227–232, https://doi.org/10.1016/j.toxlet.2014.09.007.
[70] L. Tao, K. Kannan, K.M. Aldous, M.P. Mauer, G.A. Eadon, Biomonitoring of perfluorochemicals in plasma of New York state personnel responding to the world trade center disaster, Environ. Sci. Technol. 42 (9) (2008) 3472–3478, https://doi.org/10.1021/es8000079.
[71] K.A. Crawford, B.T. Doherty, D. Gilbert-Diamond, M.E. Romano, B. Claus Henn, Waxing activity as a potential source of exposure to per- and polyfluoroalkyl substances (PFAS) and other environmental contaminants among the US ski and snowboard community, Environ. Res. 215 (Pt 3) (2022) 114335, https://doi.org/10.1016/j.envres.2022.114335.
[72] X. Jia, X. Li, L. Zhou, Y. Hui, W. Li, Y. Cai, Y. Shi, Variations of the level, profile, and distribution of PFAS around POSF manufacturing facilities in China: an overlooked source of PFCA, Environ. Sci. Technol. 57 (13) (2023) 5264–5274, https://doi.org/10.1021/acs.est.2c08995.
[73] G. Pitter, F. Da Re, C. Canova, G. Barbieri, M. Zare Jeddi, F. Daprà, F. Manea, R. Zolin, A.M. Bettega, G. Stopazzolo, S. Vittorii, L. Zambelli, M. Martuzzi, D. Mantoan, F. Russo, Serum levels of perfluoroalkyl substances (PFAS) in adolescents and young adults exposed to contaminated drinking water in the Veneto region, Italy: a cross-sectional study based on a health surveillance program, Environ. Health Perspect. 128 (2) (2020) 27007, https://doi.org/10.1289/EHP5337.
[74] D. Borg, B.O. Lund, N.G. Lindquist, H. Håkansson, Cumulative health risk assessment of 17 perfluoroalkylated and polyfluoroalkylated substances (PFASs) in the Swedish population, Environ. Int. 59 (2013) 112–123, https://doi.org/10.1016/j.envint.2013.05.009.
[75] N. Ding, C.A. Karvonen-Gutierrez, W.H. Herman, A.M. Calafat, B. Mukherjee, S.K. Park, Perfluoroalkyl and polyfluoroalkyl substances and body size and composition trajectories in midlife women: the study of women's health across the nation 1999-2018, Int. J. Obes. 45 (9) (2021) 1937–1948, https://doi.org/10.1038/s41366-021-00848-9.
[76] A. Fornasari, P. Fais, A.M. De Luca, G. Dal Lago, S. Santelli, G. Pelletti, E. Piva, J.P. Pascali, Direct analysis of Per- and polyfluoroalkyl substances (PFAS) in plasma by liquid chromatography coupled to mass spectrometry (LC–MS) for biomonitoring purposes, Expo. Health 18 (2026) 28, https://doi.org/10.1007/s12403-026-00763-2.
[77] W.J. Conover, R.L. Iman, Rank transformations as a bridge between parametric and nonparametric statistics, Am. Statistician 35 (3) (1981) 124–129, https://doi.org/10.2307/2683975.
[78] Y. Benjamini, Y. Hochberg, Controlling the false discovery rate: a practical and powerful approach to multiple testing, J. Roy. Stat. Soc. B 57 (1) (1995) 289–300.
[79] R.J. Simes, An improved bonferroni procedure for multiple tests of significance, Biometrika 73 (3) (1986) 751–754.
[80] M. Sech, M. Irmici, E. Piva, M.P. Bonasoni, A. Seidenari, J. Pascali, P. Fais, Assessment of per and polyfluoroalkyl substances (PFAS) in maternal blood. Biochimica Clinica, 2024, https://doi.org/10.19186/BC_2024.037.
[81] J.C. Botelho, K. Kato, L.Y. Wong, A.M. Calafat, Per- and polyfluoroalkyl substances (PFAS) exposure in the U.S. population: NHANES 1999-March 2020, Environ. Res. 270 (2025) 120916, https://doi.org/10.1016/j.envres.2025.120916.
[82] A.M. Ingelido, A. Abballe, E. Dellatte, F. Ferri, N. Iacovella, V. Marra, S. Valentini, E. De Felip, Ten years of PFOS and PFOA human biomonitoring in Italy: exposure levels and determinants of exposure, Chemosphere 376 (2025) 144297, https://doi.org/10.1016/j.chemosphere.2025.144297.
[83] E. De Felip, A. Abballe, F.L. Albano, T. Battista, V. Carraro, M. Conversano, S. Franchini, L. Giambanco, N. Iacovella, A.M. Ingelido, A. Maiorana, F. Maneschi, V. Marra, A. Mercurio, R. Nale, B. Nucci, V. Panella, F. Pirola, M.G. Porpora, E. Procopio, V. Vecchiè, Current exposure of Italian women of reproductive age to PFOS and PFOA: a human biomonitoring study, Chemosphere 137 (2015) 1–8, https://doi.org/10.1016/j.chemosphere.2015.03.046.
[84] A.M. Ingelido, V. Marra, A. Abballe, S. Valentini, N. Iacovella, P. Barbieri, M.G. Porpora, A.d Domenico, E. De Felip, Perfluorooctanesulfonate and perfluorooctanoic acid exposures of the Italian general population, Chemosphere 80 (10) (2010) 1125–1130, https://doi.org/10.1016/j.chemosphere.2010.06.025.
[85] J.M. Graber, T.M. Black, N.N. Shah, A.J. Caban-Martinez, S.E. Lu, T. Brancard, C.H. Yu, M.E. Turyk, K. Black, M.B. Steinberg, Z. Fan, J.L. Burgess, Prevalence and predictors of Per- and polyfluoroalkyl substances (PFAS) serum levels among members of a suburban US volunteer fire department, Int. J. Environ. Res. Publ. Health 18 (7) (2021) 3730, https://doi.org/10.3390/ijerph18073730.
[86] I. Obeegadoo, R. Luo, L. Pelland-St-Pierre, R. Dugandzic, S. Donaldson, S. Hayes, S. Cakmak, K. Larsen, Association between per- and polyfluoroalkyl chemicals and adult overweight, obesity and gestational weight gain: a scoping review, Sci. Total Environ. 1011 (2026) 181134, https://doi.org/10.1016/j.scitotenv.2025.181134.
[87] Z. Chen, W. Wei, Y. Hu, Q. Niu, Y. Yan, Associations between co-exposure to per- and polyfluoroalkyl substances and metabolic diseases: the mediating roles of inflammation and oxidative stress, Sci. Total Environ. 953 (2024) 176187, https://doi.org/10.1016/j.scitotenv.2024.176187.
[88] T. Schillemans, N. Iszatt, S. Remy, G. Schoeters, M.F. Fernández, S.C. D'Cruz, A. Desalegn, L.S. Haug, S. Lignell, A.K. Lindroos, L. Fábelová, L.P. Murinova, T. Kosjek, Ž. Tkalec, C. Gabriel, D. Sarigiannis, S. Pedraza-Díaz, M. Esteban-López, A. Castaño, L. Rambaud, A. Åkesson, Cross-sectional associations between exposure to per- and polyfluoroalkyl substances and body mass index among European teenagers in the HBM4EU aligned studies, Environ. Pollut. 316 (Pt 1) (2023) 120566, https://doi.org/10.1016/j.envpol.2022.120566.
[89] Y.P. Tian, X.W. Zeng, M.S. Bloom, S. Lin, S.Q. Wang, S.H.L. Yim, M. Yang, C. Chu,

N. Gurram, L.W. Hu, K.K. Liu, B.Y. Yang, D. Feng, R.Q. Liu, M. Nian, G.H. Dong, Isomers of perfluoroalkyl substances and overweight status among Chinese by sex status: isomers of C8 health project in China, Environ. Int. 124 (2019) 130–138, https://doi.org/10.1016/j.envint.2019.01.006.

[90] M. Wu, Z. Zhu, R. Wan, J. Xu, Exposure to per- and polyfluoroalkyl substance and metabolic syndrome: a nationally representative cross-sectional study from NHANES, 2003-2018, Environ. Pollut. 346 (2024) 123615, https://doi.org/10.1016/j.envpol.2024.123615.

[91] K.T. Eriksen, M. Sørensen, J.K. McLaughlin, A. Tjønneland, K. Overvad, O. Raaschou-Nielsen, Determinants of plasma PFOA and PFOS levels among 652 Danish men, Environ. Sci. Technol. 45 (19) (2011) 8137–8143, https://doi.org/10.1021/es100626h.

[92] J. Eom, J. Choi, J. Kim, Y. Kim, A survey of exposure level and lifestyle factors for perfluorooctanoate and perfluorooctane sulfonate in human plasma from selected residents in Korea, Int. J. Environ. Res. Publ. Health 11 (7) (2014) 7231–7241, https://doi.org/10.3390/ijerph110707231.

[93] K.E. Barton, A.P. Starling, C.P. Higgins, C.A. McDonough, A.M. Calafat, J.L. Adgate, Sociodemographic and behavioral determinants of serum concentrations of per- and polyfluoroalkyl substances in a community highly exposed to aqueous film-forming foam contaminants in drinking water, Int. J. Hyg Environ. Health 223 (1) (2020) 256–266, https://doi.org/10.1016/j.ijheh.2019.07.012.

[94] Y. Liang, H. Zulin, M. Wucheng, S. Wenqing, Z. Qiang, G. Wenzhou, X. Xiaolei, Urban soils encounter more severe per- and polyfluoroalkyl substance contaminations and associated exposure risks, Environ. Pollut. 393 (2026) 127686, https://doi.org/10.1016/j.envpol.2026.127686.