REVIEW ARTICLE

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Polyfluorinated compounds in the workplace: assessing occupational exposure and cancer risk mechanisms

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ABSTRACT

Introduction: Occupational exposure to per- and polyfluorinated substances (PFAS) poses health risks, particularly for firefighters. Mitigation includes stricter personal protective equipment (PPE) use, PF-free foam alternatives, and ongoing exposure monitoring. **Objective:** To summarize the evidence on the health impacts of occupational exposure to PFAS. **Methods:** A systematic review was conducted in the PubMed and Web of Science databases, considering all studies available at the time of the search, following the PRISMA 2020 guidelines. The MeSH terms utilized included "occupational exposure and PFAS", “occupational exposures and polyfluorinated compounds,” and "occupational exposures and Per- and polyfluoroalkyl substances (PFAS)". These articles were assessed by three independent reviewers. The selection followed the PECO model. **Results:** A total of 12 studies published between 2010 and 2023 were included. Sample sizes ranged from 11 to 225 participants, with studies conducted mainly in the USA, Europe, China, and Australia. Overall, the evidence indicates that firefighters consistently present higher body burdens of PFAS compared to the general population. Additionally, significant PF contamination was reported in occupational environments beyond emergency services, such as offices and classrooms, with elevated levels of PFNA, PFTeDA, FTOHs, PFOA, and PFHxS. These findings suggest that exposure is not limited to high-risk professions but may also occur in seemingly low-exposure settings. **Conclusion:** The reviewed studies point to firefighters having greater occupational exposure to PFAS, and an alternative to minimize this exposure would be more rigorous application of PPE protocols, replacement of firefighting foams containing PFAS with safer alternatives, and constant monitoring.

Keywords: Hazardous Substances; Human Health; Occupational Health.

INTRODUCTION

Perfluoroalkyl and polyfluoroalkyl substances (PFAS) are synthetic chemical compounds widely used in various industrial sectors due to their high resistance to degradation, heat, and their hydrophobic and lipophobic properties[1]. While these characteristics provide industrial benefits, they also lead to environmental persistence and bioaccumulation in humans. Research shows that the general population is exposed mainly through the consumption of contaminated water and food[2].

While the general population is exposed to PFAS, workers directly involved in their production, handling, or use face heightened exposure risks. Occupational exposure occurs via compound volatilization during use, ingestion of contaminated dust, or dermal contact[3,4]. Despite scientific advances, occupational exposure to PFAS is still poorly understood, especially due to the diversity of work environments affected.

Occupational exposure to PFAS is a significant public health concern, linked to elevated body burdens associated with increased risks of testicular and kidney cancers, immune suppression, and metabolic disorders involving adverse lipid profiles[5]. As awareness of PFAS risks grows, assessing occupational exposures is crucial to inform more effective safety protocols and regulatory policies.

The purpose of this systematic literature review is to compile and critically analyze the available evidence on the association between occupational exposure to PFAS and their health impacts, aiming to contribute to a better understanding of the risks involved and to inform prevention strategies in the workplace.

Therefore, the objective of the study was to review the mechanisms of carcinogenesis associated with PFAS.

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METHODS

The review followed the PRISMA 2020 guidelines (Figure 1). Data were collected from two key databases, PubMed and Web of Science, and the search encompassed all studies published up to the present. The literature search was restricted to these databases, as preliminary testing demonstrated a substantial overlap of retrieved records across different sources.

The eligibility criteria included original research articles that investigated occupational exposure to PFAS and examined potential carcinogenic mechanisms associated with these substances. Only studies explicitly addressing the link between PF exposure and occupational settings were considered for inclusion. Studies unrelated to occupational exposure or not published in peer-reviewed journals were excluded.

The MeSH terms utilized in the search included "occupational exposure and PFAS", “occupational exposures and polyfluorinated compounds” and "occupational exposures and Per- and polyfluoroalkyl substances (PFAS)". Human studies of all types were considered. The risk of bias in each study was evaluated using the Cochrane Risk of Bias tool and the Newcastle-Ottawa Scale (NOS), and Most studies were classified as having a low risk of bias.

The study employed the PECO (Population, Exposure, Comparator, Outcome) framework, defined as follows: a) P (Population): Adults with occupational exposure to PFAS. b) E (Exposure): Exposure could involve various types of PFAS, measured through environmental monitoring, self-reporting, or occupational data. c) C (Comparator): Different exposure levels to PFAS, with some studies including a control group of non-exposed individuals from the general population. d) O (Outcome): The impact of occupational exposure to PFAS.

During the initial screening, the authors independently reviewed the titles and abstracts of articles to identify eligible studies, with full-text reviews conducted as needed. All included

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studies were published in English, and only human studies were considered. A total of 16 studies were identified under the MeSH term "occupational exposure and PFAS," 9 studies under "occupational exposures and polyfluorinated compounds, "and 681 studies under" occupational exposures and Per- and polyfluoroalkyl substances", totaling 706 studies. Independent reviewers assessed all abstracts, selecting 29 studies that met the eligibility criteria for full review. After a comprehensive full-text assessment, 12 articles met the eligibility criteria and were included in the final systematic review (Figure 1).

RESULTS

Occupational exposure to PFAS

This study reviewed 12 articles published between 2010 and 2023, which investigated occupational exposures to PFAS and their implications for worker contamination. Among the studies, four were from the USA, two from China, four from Europe, one from Australia, and one multinational. Exposed groups included various occupational categories with sample sizes from 11 to 225. Most outcomes measured PFAS levels in serum and urine. Key data are summarized in Table 1.

Lu et al.[6] examined occupational exposure to PFAS in Chinese factory workers. Six PFAS congeners (∑6PFAS) predominated, with plasma levels averaging 1,770 ng/mL in exposed workers versus 22.2 ng/mL in controls. The exposure was linked to oxidative stress, disrupted fatty acid metabolism, and kidney damage, highlighting serious health risks.

The study of Nilsson et al.[7] studied professional ski waxers exposed to high levels of dust and PFAS during World Cup events from 2007 to 2010.

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Personal exposure exceeded occupational limits in 37% of cases, with dust concentrations up to 15 mg/m³, highlighting the need for preventive measures, as exposures exceed safety standards.

Tefera et al.[8] investigated changes in serum PFAS levels among 130 South Australian firefighters following the cessation of PFAS-containing foam use. Blood samples collected in 2018–2019 and again in 2021–2022 showed a decline in perfluorooctane sulfonate (PFOS), perfluorohexane sulfonic acid (PFHxS), and perfluorooctanoic acid (PFOA) levels (the predominant PFAS detected). Median total PFAS concentrations dropped from 21.5 ng/mL to 15 ng/mL. Annual declines averaged 13% for PFOS, 7% for PFHxS, and 4.4% for PFOA.

Tanner et al.[9] compared serum PFOS and PFOA levels in individuals with occupational PFAS exposure. Participants had serum PFOS and PFOA levels 25% and 80% higher, respectively, than NHANES study averages. Among 68 individuals in PFAS-associated jobs, those with high cumulative exposure had 34% higher PFOS levels, while longer exposure duration was linked to a 26% increase, adjusted for age, sex, and income.

In the CELSPAC-FIREexpo study by Palesová et al.[10], studied PFAS and polycyclic aromatic hydrocarbons (PAH) exposure in 52 professional firefighters, 58 recruits, and 54 controls, using blood and urine samples alongside exposure questionnaires. Bayesian weighted quantile sum (BWQS) regression revealed a significant association between combined PFAS/PAH exposure and increased total bilirubin (β=28.6%, 95% CrI: 14.6–45.7%). Stratified analyses also showed positive associations with total cholesterol (β=29.5%) and LDL cholesterol (β=26.7%) in both firefighters and controls.

In another work of the same group[11], internal levels of PFAS and PAH were measured in 166 participants, including newly recruited and professional firefighters and a control group. Firefighters showed significantly higher total PFAS levels, linked to career length, age, blood donation, and population size. Exceedances of Human Biomonitoring Value I (BM-I) (no risk

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concentration) and human biomonitoring value II (HBM-II) (increased risk for adverse health effects) values occurred in 10.9% (PFOS) and 7.6% (PFOA) of measurements. Urinary PAH levels rose after training with burning wooden pallets but remained below genotoxic effect thresholds.

Christensen et al.[12] conducted a biomonitoring study of 166 male anglers aged 50 and older in Wisconsin, examining PFAS exposure through fish consumption, demographics, and health outcomes. Seven PFAS were detected in at least 30% of participants, with PFOS showing the highest median level (19.0 ng/mL), followed by PFOA, PFHxS, and perfluorononanoic acid (PFNA). Older age correlated with higher PFAS levels, while higher body mass index was linked to lower levels. Alcohol use was associated with elevated perfluoroheptanesulfonic acid (PFHpS), PFHxS, and PFOA. Fish consumption showed limited association with PFAS, except for PFDA and PFHpS from local and restaurant fish. Perfluoroundecanoic Acid (PFuDA), PFNA, and PFDA were linked to increased pre-diabetes/diabetes risk, and PFHpS to high cholesterol, with no associations found for cardiovascular disease.

In another study, Fraser et al.[13] characterized PFAS levels in indoor dust from offices, homes, and vehicles to assess exposure among office workers in Boston, MA. Dust from homes, offices, and vehicles, and serum samples from 31 office workers (collected in 2009) were analyzed for multiple PFAS, including PFOA, PFOS, fluorotelomer Alcohols (FTOHs), and sulfonamidoethanols (FOSEs). PFNA, PFOA, PFOS, and 8:2 FTOH were detected in over 50% of all environments. Office dust showed the highest PFAS levels, notably 8:2 FTOH (309 ng/g), while PFOS was most concentrated in home and vehicle dust. Despite higher PFAS levels in office dust, serum PFOA was not associated with dust concentrations after adjusting for PFAS in office air, suggesting that indoor dust may not be a major PFAS exposure source for office workers.

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A follow-up study by the same group[14] examined indoor office air as a source of PFAS exposure among office workers in Boston, Massachusetts. Air sampling over one week in 31 offices measured FTOHs, fluorinated sulfonamides (FOSAs), and FOSEs, while serum samples from the same participants were analyzed for 12 PFAS. The highest airborne concentration was 8:2-FTOH (GM=9,920 pg/m³), with significant variation across buildings. Serum PFOA levels showed significant positive correlations with air concentrations of 6:2-FTOH (r=0.43), 8:2-FTOH (r=0.60), and 10:2-FTOH (r=0.62). FTOH levels in air significantly predicted serum PFOA ($p<0.001$), explaining 36% of its variation.

Goosey and Harrad[15] measured PFAS in dust from homes, cars, classrooms, and offices across several countries. PFAS levels were lowest in Kazakhstan and Thailand. N-Methyl Perfluorooctane Sulfonamido Ethanol (MeFOSE) and N-Ethyl Perfluorooctane Sulfonamido Ethanol (EtFOSE) were lower in Canada than in the UK and the US, while PFHxS was lower in Canada than in the UK, and EtFOSA was higher in Australia than in the UK. Classrooms had higher PFOS, PFOA, PFHxS, and MeFOSE but lower MeFOSA and FOSAs compared to other settings. In the UK, diet is the primary exposure route for PFOS, PFOA, and PFHxS, but dust ingestion may also be important. Even with high exposure, PFOS and PFOA levels stayed within tolerable daily intake limits.

The study by D'Hollander et al.[16] measured levels of PFAS in indoor dust from Flemish homes and offices. Total PFAS in homes ranged from 0.2–336 ng/g (median 3.0), while in offices they ranged from 2.2–647 ng/g (median 10), with PFOA, PFOS, and perfluorohexanoic acid (PFHxA) being the dominant compounds. Human exposure to PFOS and PFOA remained below US-EPA reference doses and EFSA provisional tolerable daily intakes. Notably, PFHxA was found at relatively high levels in dust samples.

Wu et al.[17] analyzed PFAS levels in indoor dust from clothing shops and urine samples from saleswomen in Shanghai, China. Dust PFAS concentrations ranged from 0.42 ng/g

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(PFDA) to 5.04 ng/g (PFDoA), with PFDoA and PFHxS most abundant (medians 2.95 ng/g and 1.49 ng/g). Urine PFAS levels ranged from 10.15 ng/L (PFDS) to 666.1 ng/L (PFOA), with PFOA highest (207-907 ng/L). A significant positive correlation was observed between long-chain PFAS in dust and urine ($p<0.01$). Estimated daily intake via dust ingestion for PFOA and PFOS (36.5 pg/day and 56.7 pg/day) remained well below tolerable intake limits.

Carcinogenic mechanisms of PFAS

While reviewed studies suggest a potential link between PFAS exposure and carcinogenesis, the underlying molecular mechanisms remain unclear. The observational studies did not directly measure biological markers to elucidate these pathways, which future research will clarify based on existing literature[18-31].

Evidence has demonstrated the PFAS' potential to cause damage to humans and the environment through various mechanisms[32-35]. Among the PFAS, PFOA is the most reported[7,8,9,11-13,15], followed by PFOS[8,9,11-14], and PFHxS[12,15,17]. A summary of carcinogenic mechanisms of PFAS is described in Table 2.

PFAS can activate the transcription factor PPARα and, to a lesser extent, PPARγ, promoting cell proliferation linked to tumorigenesis[32]. Temkin et al.[21] and Singh & Hsieh[36] support that PFAS exposure increases oxidative stress, causing DNA, RNA, and lipid damage, partly mediated by PPAR nuclear receptors and inflammatory pathways, highlighting the multifactorial toxicity of PFAS.

The available data converge on a multifactorial hypothesis of carcinogenesis associated with PFA exposure. This hypothesis involves complex interactions among hormonal dysregulation, oxidative stress, and epigenetic alterations, with phenotypic cell effects varying by compound structure and exposure pathways.

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DISCUSSION

The exposure to PFAS can occur in various ways for the general population, as previously mentioned. However, some groups are more susceptible to this exposure, such as firefighters. In this case, occupational exposure mainly occurs due to frequent physical contact with aqueous film-forming foams (AFFF) used in firefighting operations. PFAS are not limited to these materials; they can also be released in combustion reactions and smoke inhaled by firefighters during incidents, through contact with contaminated equipment, and via food grown near areas like fire stations where PFAS are handled[8,37-39]. Several studies have highlighted firefighters' occupational exposure to PFAS via dermal absorption, inhalation, and dust during emergency responses and training.

An Australian study by Tefera et al.[40] assessed PFAS levels in 916 firefighters, with follow-up in 185 participants. Exposure data and serum concentrations of PFOS, PFHxS, and PFOA were analyzed. PFOS was detected in 98.5% of samples, PFHxS in 78%, and PFOA in 73%, with median levels of 11, 5, and 2 ng/mL, respectively. Most exposures occurred during training. Annual declines were observed 13% for PFOS, 7% for PFHxS, and 4.4% for PFOA, slower in older or highly exposed individuals. The study emphasizes the need for long-term monitoring to accurately assess occupational PFAS exposure.

The CELSPAC – FIREexpo study by Řiháčková et al.[11] investigated long-term PFAS and PAH exposure among Czech firefighters aged 18-35, focusing on associated biomarkers and health risks. The study included 164 participants, divided into newly trained firefighters (n=59), experienced firefighters (n=51), and controls (n=54), with biomonitoring conducted over four phases across 11 weeks. Serum PFAS concentrations were consistently 1.1 to 1.4 times higher in both firefighter groups compared to controls, with significant increases in PFOA, PFNA, PFDA, and PFOS. No differences were found between rookies and experienced firefighters. Urine analysis revealed significant spikes in OH-PAH metabolites shortly after

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combustion training in recruits (3.4-fold at 1 hour and 5.7-fold at 4 hours), which returned to baseline within weeks. These findings highlight the acute and chronic exposure risks faced by firefighters and the need for ongoing monitoring and preventive strategies.

The study found that experienced firefighters who donated blood had lower serum levels of PFOA, PFNA, PFOS, and PFHpS. Although the link between career length and PFAS levels was weak, it was present. Firefighters recently involved in large-scale fires had significantly higher urinary PAH metabolites. No PFAS differences were seen between recent foam users and non-users, likely due to infrequent foam use. Despite the small sample, the study offers valuable longitudinal exposure insights.

The study by Pálešová et al.[10] evaluated the link between PFAS, PAHs, and cardiovascular risk in Czech male firefighters. PFNA and PFOA were the most frequent PFAS detected, while all samples contained polycyclic aromatic hydrocarbons (OH-PAHs) (1-OH-NAP, 2-OH-NAP, 2-OH-FLU, 2/3-OH-PHEN). Liver enzymes and lipid profiles were also analyzed to assess health impacts.

Moreover, the findings of Batzella et al.[41,42] study demonstrate a significant link between serum PFAS levels and cardiovascular risk biomarkers, highlighting the need for clinical monitoring in highly exposed occupational groups. And so, Gaines and Nylander-French[43] demonstrated that PFAS exposure is associated with multiple adverse health outcomes; however, occupational PFAS exposure has been characterized in only a limited subset of professions.

This longitudinal study of 799 participants found significantly elevated serum PFOS, PFHxS, and PFHpS levels among AFFF-exposed workers compared to the general Australian population. These patterns align with known use of PFOS/PFHxS-based foams. Workers hired after the AFFF phase-out had background-level PFAS, demonstrating the effectiveness of substitution. Estimated half-lives (PFOA: 5.0; PFHxS: 7.8; PFHpS: 7.4; PFOS: 6.5 years)

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exceeded those in the general population, suggesting ongoing low-level exposure or concentration-dependent clearance. The findings support AFFF replacement and long-term biological monitoring[44,45].

Mazumder et al.[46] confirmed elevated PFAS serum levels in firefighters (PFOS: 27 ng/mL) and prolonged elimination half-lives (5.0–7.8 years). Serum levels correlated with PFOS/PFHxS-based AFFF use, while individuals recruited after foam replacement showed background-level concentrations, validating the effectiveness of the intervention. PFOA is classified as potentially carcinogenic, with firefighting studies linking exposure to increased risks of several cancers. These findings support transitioning to PFAS-free foams, enhancing gear decontamination, and implementing long-term biomonitoring.

Modern risk assessments incorporate chemical-specific data, though PFAS metabolic pathways remain unclear. As reviewed by Tansel et al.[47] PFAS, which bind to proteins rather than accumulate in lipids, are prevalent at e-waste sites, confirming these as significant exposure hotspots. Although PFAS and PAH concentrations vary by region and firefighting practices, studies consistently show that firefighters have significantly higher levels than the general population, underscoring their elevated occupational risk. Epidemiological and biomonitoring research has been key in quantifying this gap. These findings highlight the urgent need for stronger exposure controls, including strict PPE use, effective decontamination, regular health monitoring, and the transition to PFAS-free foams to reduce long-term health risks.

The main limitations of this review include the small number of eligible studies and the heterogeneity of study designs and populations. Furthermore, most available evidence focused on firefighters, limiting the generalizability of the findings to other occupational groups. Potential language and publication biases should also be acknowledged. Nevertheless, the findings reinforce the urgent need for regulatory attention and public awareness, while also

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highlighting persisting gaps in exposure assessment, toxicological data, and protective measures for workers.

Conclusion

This review underscores that occupational exposure to per- and polyfluoroalkyl substances (PFAS) represents a significant public health concern, particularly for chronically exposed workers such as firefighters, chemical manufacturers, and enclosed environments where PFAS-containing materials are present. Continued research, stronger enforcement of occupational safety standards, and the development of safer alternatives are essential to reducing occupational risks and safeguarding long-term health in PFAS-exposed environments.

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Figure 1: PRISMA flow diagram.

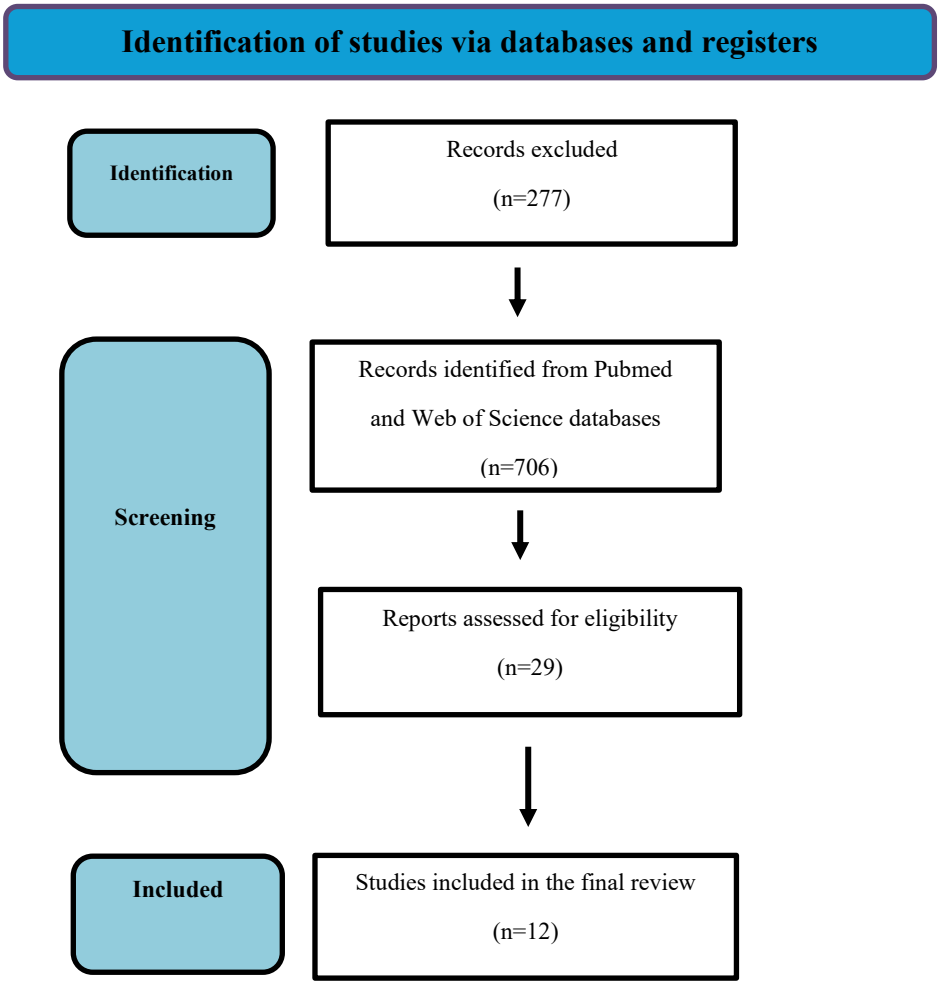

Table 1: Characteristics of the selected studies for the systematic review

Author/year	Country	Type of PF	Type of exposure	Number of individuals	Outcome
Lu et al. 2019[6]	China	PFAS	Fabric workers	40 exposed and 52 controls	Occupationally exposed workers have plasmatic levels of PFAS linked to biomarkers of oxidative stress, fatty acid β-oxidation disorder, and kidney injury.
Nilsson et al. 2013[7]	Sweeden	PFOA fumes	Ski waxers	11 exposed	Aerosol levels in wax cabins during work exceed the acceptable occupational exposure limits.
Tefera et al. 2023[8]	Australia	PFOS, PFHs, PFOAs	Firefighters	130 exposed	Decreasing trend in serum PFAS levels over time among metropolitan firefighters after workplace measures were implemented, which included the elimination of PFAS-containing foams.
Tanner et al. 2018[9]	USA	PFOS and PFOAs	Industries	154 exposed	Participants with high cumulative workplace exposure had serum PFOS levels that were 34% higher than those of participants with no occupational exposure.
Pálešová et al. 2023[10]	Czech Republic	PFAS and PAH	Firefighters	Professional firefighters (n=52), newly recruited firefighters in training (n=58), and controls (n=54)	Increased exposure to a mixture of these compounds is linked to higher total bilirubin levels and changes in serum lipids, which can lead to an unfavorable cardiometabolic profile.
Řiháčková et al. 2023[11]	Czech Republic	PFOS, PAHs, PFOAs	Firefighters	166 exposed and unexposed	The ΣPFAS levels in firefighters were significantly higher than those in the control group and were primarily linked to the duration of their firefighting careers.
Christensen et al. 2016[12]	USA	PFDA, PFHpS, PFHxS, PFNA, PFOA, PFOS, PFuDA	Anglers	50 exposed	PFuDA, PFNA, and PFDA were all linked to an elevated risk of pre-diabetes and/or diabetes, while PFHpS was associated with a significantly higher risk of high cholesterol.
Fraser et al. 2013[13]	USA	PFOAs, PFOS, PFNA, PFTeDA, FTOHs, FOSEs	Office workers	31 exposed	Offices had the highest concentrations of PFNA, PFTeDA, and FTOHs.
Fraser et al. 2012[14]	USA	PFOAs and PFOS, and FTOHs	Office workers	31 exposed	FTOH concentrations in office air significantly predict serum PFOA levels in office workers.
Goosey and Harrad, 2011[15]	Australian, Canadian, French, German, Kazahkstani, Thai, UK, and US homes	PFOS, PFOA, PFHxS, and MeFOSE	Classrooms	225 exposed	PFOA, PFHxS, and MeFOSE concentrations were significantly higher in classrooms compared to cars, homes, and offices.
D'Hollander et al. 2010[16]	Belgium	PFOA, PFOS, and PFHxA	Office workers	Homes (n=43) and offices (n=10)	PFOA, PFOS, and PFHxA were detected in both office and house dust samples.
Wu et al. 2019[17]	China	PFDA, PFDoA and PFHxS	Salewomen from clothing shops	73 exposed	Urine concentrations of PFAS were influenced by indoor dust.

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Table 2: Summary of carcinogenic mechanisms associated with the PFAS.

Mechanism	Perfluorinated substances	Main findings	Authors/Years
Epigenetic	PFOS and PFOA	Global histone modifications, hypo- and hypermethylation of specific gene regions and global genome	Pierozan et al. 2020[18]
	PFHxS	Reduction in the H3K9ac and H3K4me3 levels; Elevation of H3K27ac levels	Pierozan et al.2022[19]
	PFOS and PFOA mixture	Decrease in the H3K27ac and H3K9me2 levels	Pierozan et al. 2023[20]
	PFOS and PFOA	Global histone modifications, hypo- and hypermethylation of specific gene regions and global genome	Temkin et al. 2020[21]
Hormonal receptors	PFOA	Activation of estrogen receptor alpha (ERα) and G protein-coupled estrogen receptor (GPER), triggering PI3K/Akt and MAPK/Erk pathways linked to cell migration and invasiveness	Liu et al. 2023[22] Benninghoff et al. 2012[23]
	PFOSA	Human estrogen receptor (hER) transcription activation.	Evans et al. 2022[24]
	8:2 and 6:2 FTOH	hER activation and agonism	
	PFHxS	hER activation and agonism; constitutive androstane receptor (CAR) stimulation	Pierozan et al.2022[19], Evans et al. 2022[24]
	PFOS and PFOA mixture	Activation of the pregnane X receptor (PXR) culminating in cell proliferation	Pierozan et al. 2023[20]
Oxidative stress	PFOS and PFHxS	Increase in intracellular reactive oxygen species (ROS) and mitochondrial membrane potential decrease	Feng et al. 2023[25]
	PFOS and PFOA mixture	Involvement of ROS increase in cell death	Pierozan et al. 2023[20]
	PFOA	Lipid peroxidation, increase of H_2O_2 levels, nitrosative stress, Nrf2 expression decrease, and antioxidant enzymes inhibition associated with inflammation and apoptosis	Temkin et al. 2020[21]
	PFOS, PFDA, PFNA, PFUnA, and PFHxS	Oxidative/nitrosative stress, including changes in the α-tocopherol, glutathione, and ascorbate pathways	
Cellular effects	PFOS and PFHxS	Oocyte viability impairment by disrupting cell division structures and reducing the cleavage rates	Feng et al. 2023[25]
	PFDA	Macrophage M2 polarization via Wnt/β-catenin pathway activation, which is crucial to increase ovarian cancer metastases in animal models	Cui et al. 2024[26]
	PFOA and PFOS	Cell-cycle proteins change (cyclin D1, CDK6, p21, p53, p27, ERK 1/2 and p38) and decreased expression of adhesion proteins (E-cadherin, occludin, β-integrins), suggesting compromised cell adhesion and induction of epithelial-mesenchymal transition. Hepatocyte nuclear factor 4 alpha (HNF4α) degradation impaired lipid metabolism and liver function.	Pierozan et al. 2020[18] Beggs et al. 2016[29]
	PFOS and PFOA mixture	Cell proliferation resulting from an increase in cyclin D1 and CDK6/4 levels, a decrease in p21 and p53 levels, regulation of p-Akt and β-catenin, and PXR activation	Pierozan et al. 2023[20]
	PFHxS	Affects regulatory cell-cycle proteins (cyclin D1, cyclin E, CDH2, CDK4, CDK6, p27, p53, and ERK) and induces cell proliferation, at least in part through CAR and the peroxisome proliferator-activated receptor alpha (PPARα) activation. High concentrations decreased the cell viability	Pierozan et al.2022[19] Sim et al. 2023[30]
	PFDoA	Gene dysregulation related to inflammatory responses, cell signaling, and disruptions in estrogen- and androgen-receptor-mediated hormone pathways, contributing to malignant cell transformation	Johnson et al. 2013[33], Mao et al. 2024[34]
	PFOA, PFUA, and PFDA	Increase the risk of non-alcoholic fatty liver disease, which was associated with PPARα activation, upregulation of hepatic acyl-CoA oxidase 1 (ACOX1) transcription, oxidative stress, and subsequent fat accumulation.	Yang et al. 2023[28]

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