Supplementary Material

A Scoping Literature Review of Toxicological Studies on Per- and Polyfluoroalkyl Substances (PFAS)

Marianthi Papathanasiou,[a] Haralampos Tzoupis,[b] Konstantinos D. Papavasileiou,[b,c,d] Francesco Dondero,[d,e] Dario Greco,[f,g] Iseult Lynch,[d,h] ,Antreas Afantitis[b,c,d,i], Georgia Melagraki[a]

[a] Division of Physical Sciences & Applications, Hellenic Military Academy, Vari 166 73, Greece

[b] Department of ChemoInformatics, Novamechanics Ltd., Nicosia 1070, Cyprus

[c] Department of Chemoinformatics, Novamechanics MIKE, Piraeus 18545, Greece

[d] Division of Data Driven Innovation, Entelos Institute, Larnaca 6059, Cyprus

[e] Department of Science and Technological Innovation, Università del Piemonte Orientale, 15121 Alessandria, Italy

[f] Finnish Hub for Development and Validation of Integrated Approaches (FHAIVE), Faculty of Medicine and Health Technology, Tampere University, 33520 Tampere, Finland

[g] Division of Pharmaceutical Biosciences, Faculty of Pharmacy, University of Helsinki, 00014 Helsinki, Finland

[h] School of Geography, Earth and Environmental Sciences, University of Birmingham Edgbaston, Birmingham B15 2TT, UK

[i] Department of Pharmacy, Frederick University, Nicosia 1036, Cyprus

Number of pages: 83

Number of Tables: 5

Table S1: PRISMA-ScR checklist for scoping reviews

Table S2: Overview of the PFAS chemicals commonly found in the studies reviewed, indicating their prevalence in the studies included in the scoping review.

Methodology

Inclusion and exclusion criteria of the scoping review (ScR) in detail

Results

Table S3: Species found in the *in vivo* studies from selected papers, the predominant effects observed in each species, the substances likely responsible for these effects, and the conditions (concentrations and time duration) that species have been exposed to.

Table S4: Summary of in vitro PFAS toxicological studies, including the substances used in each study, exposure concentrations, cell lines, (human) tissue types, and main observed effects.

Tables S5: Comparative toxicological evidence for alternative PFAS relative to legacy PFAS (PFOS, PFOA). The table summarizes key toxicological endpoints, highlights similarities and differences in potency and bioaccumulation, and provides supporting references.

Table S6: Full inventory of included human epidemiological studies (n = 69) identified in the ScR. Health outcome/endpoint was extracted verbatim from study titles using a rule-based parsing approach. Table 7 in the main manuscript presents an illustrative subset of these studies. Two studies appear in 2024 journal issues but were published online in 2023 and retained per the eligibility rule.

References

Table S1. PRISMA-ScR checklist for scoping reviews

SECTION	ITEM	PRISMA-ScR CHECKLIST ITEM	REPORTED ON PAGE #
TITLE			
Title	1	Identify the report as a scoping review.	Page 1
ABSTRACT			
Structured summary	2	Provide a structured summary that includes (as applicable): background, objectives, eligibility criteria, sources of evidence, charting methods, results, and conclusions that relate to the review questions and objectives.	Page 2
INTRODUCTION			
Rationale	3	Describe the rationale for the review in the context of what is already known. Explain why the review questions/objectives lend themselves to a scoping review approach.	Pages 4-8
Objectives	4	Provide an explicit statement of the questions and objectives being addressed with reference to their key elements (e.g., population or participants, concepts, and context) or other relevant key elements used to conceptualize the review questions and/or objectives.	Pages 7-8
METHODS			
Protocol and registration	5	Indicate whether a review protocol exists; state if and where it can be accessed (e.g., a Web address); and if available, provide registration information, including the registration number.	Page 8
Eligibility criteria	6	Specify characteristics of the sources of evidence used as eligibility criteria (e.g., years considered, language, and publication status), and provide a rationale.	Pages 8-11
Information sources*	7	Describe all information sources in the search (e.g., databases with dates of coverage and contact with authors to identify additional sources), as well as the date the most recent search was executed.	Pages 11,12
Search	8	Present the full electronic search strategy for at least 1 database, including any limits used, such that it could be repeated.	Page 12 and Supporting Information page 7
Selection of sources of evidence†	9	State the process for selecting sources of evidence (i.e., screening and eligibility) included in the scoping review.	Section 2.4 main text, page12

SECTION	ITEM	PRISMA-ScR CHECKLIST ITEM	REPORTED ON PAGE #
Data charting process‡	10	Describe the methods of charting data from the included sources of evidence (e.g., calibrated forms or forms that have been tested by the team before their use, and whether data charting was done independently or in duplicate) and any processes for obtaining and confirming data from investigators.	Section 2.4, pages 12-13
Data items	11	List and define all variables for which data were sought and any assumptions and simplifications made.	Section 2.5, page 13
Critical appraisal of individual sources of evidence§	12	If done, provide a rationale for conducting a critical appraisal of included sources of evidence; describe the methods used and how this information was used in any data synthesis (if appropriate).	N/A
Synthesis of results	13	Describe the methods of handling and summarizing the data that were charted.	Section 2.5, page 13
RESULTS			
Selection of sources of evidence	14	Give numbers of sources of evidence screened, assessed for eligibility, and included in the review, with reasons for exclusions at each stage, ideally using a flow diagram.	Pages 14-16
Characteristics of sources of evidence	15	For each source of evidence, present characteristics for which data were charted and provide the citations.	Pages 21-24, 28-30 and 33-35
Critical appraisal within sources of evidence	16	If done, present data on critical appraisal of included sources of evidence (see item 12).	N/A
Results of individual sources of evidence	17	For each included source of evidence, present the relevant data that were charted that relate to the review questions and objectives.	Supporting Information pages 7-68
Synthesis of results	18	Summarize and/or present the charting results as they relate to the review questions and objectives.	Pages 16, 17-38
DISCUSSION			
Summary of evidence	19	Summarize the main results (including an overview of concepts, themes, and types of evidence available), link to the review questions and objectives, and consider the relevance to key groups.	Pages 35-38
Limitations	20	Discuss the limitations of the scoping review process.	Pages 38-40
Conclusions	21	Provide a general interpretation of the results with respect to the review questions and objectives, as well as potential implications and/or next steps.	Page 41

SECTION	ITEM	PRISMA-ScR CHECKLIST ITEM	REPORTED ON PAGE #
FUNDING			
Funding	22	Describe sources of funding for the included sources of evidence, as well as sources of funding for the scoping review. Describe the role of the funders of the scoping review.	Page 43

From: Tricco AC, Lillie E, Zarin W, O'Brien KK, Colquhoun H, Levac D, et al. PRISMA Extension for Scoping Reviews (PRISMAScR): Checklist and Explanation. Ann Intern Med. 2018;169:467–473. doi: 10.7326/M18-0850.

Table S2. Overview of the PFAS chemicals commonly found in the studies reviewed, indicating their prevalence in the studies included in the scoping review.

Acronym	Chemical Name	CAS Registry Number	Carbon Chain length	Molecular weight (g/mol)	No. of studies[a]
6:2 Cl-PFESA	Perfluoro(2-((6-chlorohexyl) oxy) ethanesulfonic acid)	756426-58-1	C8	532.58	28
8:2 Cl-PFESA	11-Chloroperfluoro-3-oxaundecanesulfonic acid	763051-92-9	C10	632.60	10
6:2 FTOH	2-(Perfluorohexyl)ethanol	647-42-7	C8	364.10	8
6:2 FTSA	2-(Perfluorohexyl)ethane-1-sulfonic acid	27619-97-2	C8	428.17	8
HFPO- DA (or GenX)	Hexafluoropropylene oxide dimer acid	62037-80-3	C6	330.05	42
HFPO-TA	Hexafluoropropylene oxide trimer acid	13252-14-7	C9	496.07	17
OBS	Sodium p-perfluorous nonenoxybenzene sulfonate	70829-87-7	C15	626.22	7
PFBA	Perfluorobutanoic acid	375-22-4	C4	214.04	25
PFBS	Perfluorobutane sulfonic acid	375-73-5	C4	300.10	50
PFDA	Perfluorodecanoic acid	335-76-2	C10	514.08	56
PFDoA	Perfluorododecanoic acid	307-55-1	C12	614.10	28
PFHpA	Perfluoroheptanoic acid	375-85-9	C7	364.06	36

PFHpS	Perfluoroheptanesulfonic acid	375-92-8	C7	450.12	8
PFHxA	Perfluorohexanoic acid	307-24-4	C6	314.05	40
PFHxS	Perfluorohexane sulfonic acid	355-46-4	C6	400.12	86
PFNA	Perfluorononanoic acid	375-95-1	C9	464.08	72
PFOA	Perfluorooctanoic acid	335-67-1	C8	414.07	202
PFOS	Perfluorooctane sulfonic acid	1763-23-1	C8	500.13	198
PFOSA	Perfluorooctanesulfonamide	754-91-6	C8	499.15	17
PFPeA	Perfluoropentanoic acid	2706-90-3	C5	264.05	15
PFTrDA	Perfluorotridecanoic acid	72629-94-8	C13	664.10	14
PFUnDA	Perfluoroundecanoic acid	2058-94-8	C11	564.09	44

[a] The number of studies in this review that include the respective compound

Note: All information for PFAS was derived from PubChem (Kim et al., 2025). Molecular weight and carbon chain length are included due to their utility in *in silico* toxicological modeling and structure-activity relationship analysis.

Methodology

Search strategy

“Full text” and “English” filters were applied. Only in PubMed “Associated Data” filter applied for double checking records with providing data during the full text screening.

Queries in PubMed

1. ("toxicity" OR "toxicology" OR "chemoinformatics" OR "cheminformatics" OR "QSAR" OR "QSPR" OR "machine learning" OR "modeling") AND ("PFAS" OR "PFOA" OR "PFOS") → 1,869 results
2. ("toxicity" OR "toxicology") AND ("PFAS" OR "PFOA" OR "PFOS") AND ("chemoinformatics" OR "cheminformatics" OR "QSAR" OR "QSPR" OR "machine learning" OR "modeling") → 116 results

Queries in Scopus

1. ("toxicity" OR "toxicology" OR "chemoinformatics" OR "cheminformatics" OR "QSAR" OR "QSPR" OR "machine learning" OR "modeling") AND ("PFAS" OR "PFOA" OR "PFOS") → 1,714 results
2. ("toxicity" OR "toxicology") AND ("PFAS" OR "PFOA" OR "PFOS") AND ("chemoinformatics" OR "cheminformatics" OR "QSAR" OR "QSPR" OR "machine learning" OR "modeling") → 95 results

Query in ACS Publications

1. ("toxicity" OR "toxicology") AND ("PFAS" OR "PFOA" OR "PFOS") → 185 results

Table S3. Species models found in *in vivo* studies from selected papers, the predominant effects observed in each species, the substances likely responsible for these effects, and the conditions (concentrations and time duration) that species have been exposed to.

Model organism	PFAS used	Concentration and time of exposure	Route of exposure	Effects	Study
Long- Evans male rats	PFOA, PFOS, PFBA, PFBS	1 to 10^3 ng/L for 14 days	Oral (water)	Alterations in testosterone (T) and luteinizing hormone (LH) levels; LH-stimulated Leydig cell T production.	[1]
Pregnant SD rats	NBP2 or PFOS	0.1, 0.3, 1, 3, 10, or 30 mg/kg	Oral gavage	Changes in the highest dose associated with lipid and carbohydrate metabolism; NBP2: developmental toxicant in the rat → neonatal mortality, ↓ pup bodyweight, liver glycogen and thyroid hormones.	[2]
	HFPO-DA (or GenX)	10, 30, 62.5, 125, or 250 mg/kg from GD8 – PND2		Dose-responsive ↓ birthweight and ↑ body weight of dams; genes related to glucose metabolism affected; Comparison of neonatal mortality between HFPO-DA and previously published PFOS data indicate similar potency of both.	[3]
	Mixture of HFPO-DA, Nafion BP2 and PFOS	110 mg/kg, 10 mg/kg, and 3 mg/kg (top doses) daily; top dose solution diluted at 33.3, 10, 3.3, and 1 % dose	Oral	↓ hepatic glycogen accumulation; ↓ maternal thyroid hormone levels; Upregulated genes related to liver function after exposure to HFPO-DA and mixture.	[4]
	PFOA, PFOS, and their mixture	10–250 mg/kg, 0.1–5 mg/kg, and 2 mg/kg PFOS mixed with 3–80 mg/kg PFOA daily for 17 days	Oral	↓ maternal bodyweight and birthweight; total thyroid hormones decreased; hepatocyte hypertrophy (mixture). Mixture effects similar to individual substance effects.	[2]

Pregnant SD rats	PFOS	0, 1.7, 5 and 15 mg/L from gestation to adulthood	Drinking water	Inhibition of the long-term potentiation → effects in synaptic transmission and plasticity both in pre- and post- synaptic cells; ↑ levels of AMPA receptors → PFOS-induced cognitive function impairment.	5
		0, 1 or 5 mg/kg for 6 days 0.03 mg/kg/bw for 20 days	Oral gavage	Upregulated inflammatory cytokines released by the inflammasome; higher expression of the VEGFA (lung development proteins) Lower birth weight; liver changes in pathways, such as steroid biosynthesis, antigen processing etc.; liver metabolites changes.	6,7
		0.3 mg/kg.bw/day from GD1 to birth and PND1 to PND21		Decreased bodyweight; ↑ IL-1β, IL-6 and TNFα; Altered composition of gut microbiota and blood metabolites.	8
		50 μg/mL 10 μg/mL and 50 μg/mL	Oral (water)	Fetal growth restriction with postnatal catch-up in females, but not males and development of hypertension in both sexes. Placental steroidogenesis-associated genes and hormone levels.	9,10
Male SD rats		0 (control), 1 and 10 mg/kg	Intraperitoneal injection	PFOS-induced myocardial injury, cardiac fibrosis and hypertrophy; p53 upregulated → association with myocardial apoptosis; IL-1β accumulation and significant upregulation of TNF-α expression.	11
	6:2 Cl-PFESA	0 (control) and 0.5 mL for 28 days	Oral	↓ α diversity of rat gut microbiome; alterations in serum hormones and upregulated genes associated with steroid hormone biosynthesis.	12

SD rats	PFOA, PFOS	0.1, 1 and 10 mg/kg daily for 5 days	subcutaneous injection	Pubertal developmental changes in female rats; estradiol hormone levels affected; reduced kisspeptin-immunoreactive fiber.	[13]
	PFOA, PFHpA	0, 250, and 1000 mg/kg bw/day for 2 weeks	Dermal	At 1000 mg/kg, most exposed rats died, due to severe ulcerative dermatitis; Systemic changes in the kidney, liver and testes, and renal tubular necrosis; Hepatocellular necrosis.	[14]
	PFOS	1 or 10 mg/kg bw every other day for 15 days	Intraperitoneal injection	↑ serum urea nitrogen → renal damage induced nephrotoxicity in rats; upregulated apoptosis of tubular epithelial cells and expression of Cx43 (protein playing role in renal toxicity).	[15]
				Promotion of inflammatory bowel disease (IBD)-like intestinal dysfunction through histological lesions in the proximal duodenum; inflammatory cytokines ↑ infiltration.	[16]
	PFOA	1.25, 5 and 20 mg/kg bw/day for 28 days	Oral gavage	↑ serum markers of liver injury e.g., alanine aminotransferase (ALT); ↑ expression of glucose and lipid related indexes; altered expression of AMPK/mTOR.	[17]
	6:2 Cl-PFESA (or F-53B)	5, 20, and 100 mg/kg/day for 28 days	Oral	↓ total thyroid hormone serum concentrations; development of thyroid follicular hyperplasia.	[18]
Male and female SD rats	HFPO-TeA	0, 0.3, 0.9, 2.3, 6.3, 17, 45.9, 124, and 335.2 mg/kg/day	Oral gavage	Body weight loss, liver weight changes, and clinical signs of toxicity at ≥6.3 mg/kg/day; premature deaths at ≥45.9 mg/kg/day. ↓ T3 and ↓ T4 at ≥6.3 mg/kg/day. Higher plasma and liver HFPO-TeA accumulation in	[19]

				females.	
CD-1 mice	PFOS	1 mg/kg daily	Oral (water)	Open field test analysis→ strong trend for increased rearing and significantly increased distance travelled.	20
		1 or 5 μg/g daily	Oral gavage	Alterations in fat and glycogen metabolism and downregulation of the insulin-signaling pathways.	21
		1 or 5 μg/g bw daily for 21 days	Oral (diet)	Altered hypothalamic metabolome.; Perturbation in Luteinizing hormone (LH)/ LH receptor (LHr) circuit, altered testicular transcriptome, ↓ sperm motility	22
	PFHxS, PFBS, and OBS	800 μg/L, 800 μg/L and 3 μg/L for 6 weeks	Oral (diet, water)	Alterations in the composition of the gut microbiota relating to pancreatic toxicity.	23
	PFOS or GenX	5, 10, and 20 mg/kg or 10, 20, and 100 mg/kg for 14 days	Oral gavage	Alterations in gut microbiota and potentially disruption of liver metabolome; PFOS has strongest impact on colon microbiota; both PFAS affected several host-microbiome pathways.	24
	PFOA	1, 5, 10, or 20 mg/kg daily for 10 days		↓ expression levels of DNA methyltransferases primarily in the small intestine; dysregulated translocation and tight junctions' genes in small intestine; → alterations in genes essential for maintaining the physical barrier of the gut.	25
	PFOA	1, 5, 10, or 20 mg/kg daily for 10 days	Oral gavage	↑ liver weight; antral follicles have altered steroidogenic gene expression and sex hormone production.	26

Pregnant CD-1 mice	PFOA or GenX	0, 1, or 5 mg/kg or 0, 2, or 10 mg/kg, daily from embryonic day (E) 1.5 to 17.5 0.1, 1.0 mg/kg or 0.2, 1.0, 2.0 mg/kg from GD 1.5 to 17.5	Oral gavage	GenX induced alterations in liver histopathology, ↑ placental weights and embryo-placenta weight ratios. Upregulated genes associated with bile and fatty acid metabolism, peroxisome, adipogenesis, and oxidative phosphorylation. Males displayed more characteristics of metabolic disease and females exhibited liver damage → metabolic outcomes are diet- and sex-dependent.	27–29
	PFDMO2HpA or PFDMO2OA	0, 0.04, 0.16, 0.63, 2.5, and 10 mg/kg daily or 0, 0.01, 0.04, 0.16, 0.63, and 2.5 mg/kg daily		Alterations in the expression of genes involved in inflammation and immunity in the placenta upon PFAS exposure at GD18 (late-pregnancy).	30
	PFOS, PFOA, PFHxS, and their mixture	1 mg/kg for each individual and 3 mg/kg for the mixture (1:1:1)		↑ serum ALT after PFOS exposure; ↑ liver triglycerides in the PFAS mixture group; High-fat diet mice and all PFAS groups lipid accumulation; PFAS mixture → very distinct effects compared to single PFAS treatment.	31
C57BL/6 mice	PFOS	Diet with or without 0.003 %, 0.006 % or 0.012 % of PFOS 0, 0.2, and 2.0 mg/kg daily for 6 months	Oral (diet)	Dose-dependent alterations in hepatic metabolism pathways; effects in gut microbiota metabolism. PFOS-induced heart injury; cognitive-related behavior after sub-chronic exposure; neurotoxicity.	17,32
	PFOS	0.0003% for 10 weeks	Oral- standard diet (SD) or high fat diet (HFD)	Lipid loss association when switched to SD; ↑ hepatic lipid accumulation in mice established on HFD.	33

C57BL/6 mice		5 mg/kg	Intraperitoneally injection	Liver inflammation and fibrosis with increased expression of inflammatory factors (TNF-α, IL-1β, IL-6).	[34]
		0-10 mg/kg/bw for 28 days	Oral (diet) and cell lines combined	Organic anion transporting polypeptide 3a1 (Oatp3a1) played an important role in PFOS-induced male reproductive toxicity.	[35]
		0.1, 0.25, and 1 mg/kg bw for 14 days	Oral gavage	Lactational PFOS exposure → long-lasting effect on learning and memory; Male adults exposed to PFOS during lactational period have ↑ GABA and ↑ Glu levels in the dorsal hippocampus; Potential indirect alterations in the function of the hippocampus.	[36]
		1 mg/kg bw for 14 days		Lactational PFOS exposure → motor coordination; Neurotransmitter release from parallel fibers was affected in PFOS-exposed mice; ↓ long-term depression; ↑ expression of Munc18-1 and mGluR1 in PFOS-exposed cerebellum.	[37]
	6:2 Cl- PFESA	0, 1, 3 and 10 μg/L for 10 weeks	Oral (diet)	Liver bioaccumulation → hepatomegaly; Increased risk of metabolic diseases; High binding affinity towards PPARγ; Liver damage.	[38]
	PFOS+ PFOA	0, 1.88, 3.75, 7.5, or 10 mg/kg bw	Oral gavage	↑ relative liver weights; ↓ antigen-specific antibody production.	[39]
	PFOS or PFOA	7 mg/kg or 70 mg/kg total administered dose of each PFAS for 28 days		Both PFAS ↑ lung tissue inflammation upregulates Th2 cytokine production and aggravated asthma in asthmatic mice; Regulation of the balance of Th1/Th2.	[40]

C57BL/6 mice	PFOS or PFBS	500 μg/L PFOS or 10 μg/L, 500 μg/L PFBS for 28 days	Oral (water)	Cell apoptosis and ↓ catalase (CAT) in PFBS groups, ↑CAT activity and triacylglycerols in PFOS groups; Dose-dependent lipid changes; ↑TNFα and IL-1β levels in the ileum in low-dose PFBS exposure; GSH related glucose metabolism was main contributor to PFBS toxicity, with sulfur amino acid metabolism as the main pathway of PFOS toxicity.	[41,42]
	Mixture of 5 PFAS (PFOA, PFOS, PFNA, PFHxS, and GenX)	each at a concentration of 2 mg/L for 12 weeks		↑circulating cholesterol, sterol metabolites, and bile acids with dimorphic sexual effects; hepatic injury (hepatic inflammation, and plasma ALT levels); ↑ female inflammation compared to the males. Altered expression of multiple Covid-19 related genes; ↓ circulating testosterone levels; ↓ circulating IFN-γ.	[43,44]
	PFOS or PFHxS	0.0003% for 29 weeks	Oral - high-fat diet (HFD) or low-fat diet (LFD)	↑ risk of metabolic and inflammatory disease induced by diet; dysregulated lipid metabolism and oxidative stress.	[45]
	PFOA	1mg/kg/d for 16 weeks		HFD induced hepatic steatosis, lobular inflammation, and progressive fibrosis; Activation of PPARα, CAR, and PXR, regardless of the diet; PFOA induced hepatocytes growth, while ↓ severity of hepatic steatosis; in HFD-fed mice ↓ hepatic triglyceride levels	[46]
	PFOA	1, 5, 10, and 20 mg/kg bw daily for 28 days	Oral gavage	PFOA triggered inflammatory markers (TNF-α, HGF, IL-6, and IFN-γ) and hepatic enzymes (AST and ALT).	[47]

C57BL/6 mice		0 or 7.5 mg/kg for 15 days		B-cell development and survival may be hindered by PFOA exposure; suppression of primary IgM antibody response, leading to changes in specific B cell subsets.	48
		1 mg/kg/day for 28 days		PFOA-induced lipid accumulation and NLRP3 inflammasome activation.	49
		1 or 3 mg/kg/d for 7 days		PFOA modulated the expression and function of hepatic Ces (hepatic carboxylesterases) enzymes, in part through PPARα.	50
	PFOA or PFBA	0.1, 1 and 5 mg/kg bw or 5 mg/kg bw	Oral (water)	PFOA accumulated in serum at ↑ levels than PFBA; PFOA induced hepatotoxicity; no oxidative damage in liver, and no evidence of genotoxicity in somatic and germ cells.	51
	PFMOAA or PFMOPrA, PFMOBA (PFOA as positive control)	0.00025, 0.025, 2.5 mg/kg or 0.5, 5, 50 mg/kg for 30 days		Changes in splenic cellularity in males exposed to PFMOPrA; ↓ numbers of B cells and NK cells in males and females exposed to PFMOBA.	52
	HFPO-TA	2,10,200 μg/L for 6 weeks		Altered diversity of intestinal gut microbiota; Changes in relative abundance of several phyla and genera; Effects in various metabolic pathways. Anaemia and immunological imbalance.	53
C57BL/6 WT or GPR40-KO mice	PFOS	0.5, 1, 5, and 10 mg/kg (1 h after 12-h fasting)	Oral gavage	↑ fasting serum insulin levels (significant in males, not females); effect absent in GPR40-KO mice → implicates GPR40-mediated mechanism.	54
	PFOA	0, 0.4, 2, and 10 mg/kg daily for 28 days		Overactivation of spleen macrophages → anemia and immunological imbalance; Main cause of splenic atrophy.	55

Male BALB/c mice	PFOA or HFPO-DA and HFPO-TA or HFPO-TeA	0, 1, 5, 10, and 20 mg/kg bw or 0, 0.05, 0.1, 0.5 or 1 mg/kg bw	Oral	HFPO induced disruption of the blood-testis barrier on mice; HFPO-DA and HFPO-TA show stronger disruption than PFOA.	56
	PFO2HxA, PFO3OA, PFO4DA (PFECAs) or PFOA PFMO2HpA, PFMO3NA, GenX (PFECAs) or PFOA	0.4, 2, or 10 mg/kg/d of each chemical for 28 days	Oral gavage	PFECA liver/serum ratio lower than that of PFOA in both studies → higher accumulation of PFOA; PFO4DA ↓ urea cycle-related enzyme protein levels and serum ammonia; PFO4DA induced hepatomegaly and karyolysis. PFOA, GenX, and PFMO3NA ↑relative liver weight, and bile acid metabolism; PFMO2HpA led to lower toxicity → potential alternative.	57,58
	HFPO-DA, HFPO-TA, PFO4DA, PFO5DoDA (PFECAs) or PFOA	2 or 10 μg/kg daily for 140 days		Long- terms PFECAs exposure suppressed cellular stress in male mice → hepatomegaly; PFO5DoDA inhibitory effect on the glucocorticoid signal.	59
		0.4, 2, or 10 μg/kg/d for 140 days	Oral (water)	PFECA compounds show ↑ serum half-life with increased molecular weight; PFECAs considerably inhibit glucocorticoid receptor signaling. Calculated RfDs → potential hazard of PFECAs to human health.	60
	PFOA	15 and 30 mg/kg bw for 10 days	Oral	↓ heart, lung and testis weight; ↓ CAT activity in lung; ↑ total glutathione (GSH) levels, activities of superoxide dismutase (Cu-Zn SOD) and CAT in heart; ↓ GSH levels and ↑Cu-Zn SOD and CAT activities in testes; oxidative stress involved in the toxicity mechanism of PFOA.	61

Pregnant BALB/c	PFOA and GenX	1 and 2 mg/kg daily	Oral (diet)	Increased levels of indicators of the TLR4 pathway showing liver inflammation. Hepatic alterations through the gut-liver axis.	62
ICR female mice (pregnant)	PFOA	1 mg/kg and 5 mg/kg daily for 28 days	Oral (water)	PFOA exposure impairs oocyte maturation, by ↓rate of embryonic foam rupture; Mitochondrial dysfunction → ↓ ATP levels, ↑ ROS; ↑ proportion of γ-H2AX (DNA damage marker) → abnormal arrangements of the spindle and chromosomes during oocyte maturation.	63
		0, 2.5 and 5 mg/kg daily for 28 days	Oral	PFOA exposure induces mitochondrial dysfunction in 2-cell stage; Production of ROS → occurrence of autophagy and apoptosis; Potential toxic effects on ovarian function → higher incidence of meiotic defects in F1 female offspring.	64
	PFOS	10 mg/kg from E7.5 until E16.5	Oral gavage	Preeclampsia-like phenotypes in pregnant mice; Mitochondrial damages accompanied by the activation of p38 and JNK MAPK signaling pathway.	65
ICR male mice	PFOS	0.25, 2.5, 25, and 50 mg/kg daily for 28 days	Oral gavage	Downregulation of tight-junctions proteins and disruption of BBB; Phosphorylated p38 activation was involved in PFOS-induced astrocytic damages; Crosstalk between endothelial cells and astrocytes aggravated BBB disruption.	66
A/J mice	Mixture of 8 PFAS	6-27 ng/g (based on	Oral (diet)	Alterations in the dopaminergic system related to cognitive behavior, reproduction and metabolism. Changes in the lipid metabolism and	67,68

		environmentally PFAS mixture detected in earthworms)		oxidative stress after PFAS exposure, through interaction between PFAS and nuclear receptors (e.g. PPAR-α and β, LXR-α and β).	
Female B6C3F1 mice	PFOA	0, 1.88 and 7.5 mg/kg/day for 28 days	Oral (water)	Long-term PFOA exposure ↓ IgM response at lower doses; Alterations in Th1/Th2 and pro-inflammatory cytokines following KLH stimulation; Potential role for T helper cells in the toxicity of PFOA.	[69]
Murine mouse model	PFOA	4-days 0.5-2% w/v, or 12.5-50 mg/kg/dose	Dermal exposure	PFOA-induced immunotoxicity: reduction of IgM antibody in spleen and decreased thymus, spleen and liver weights.	[70]
	PFBA PFHpA ,PFHxA, PFPeA	15-day 375 mg/kg/dose and 28-day 93.8-187.5 mg/kg/dose 28- day 31.25-125 mg/kg/dose	Dermal exposure	PFBA-induced liver damage and alterations of PPAR target genes. Alternative PFAS induced systemic toxicity and immunological disruption through PPARα, δ and γ.	[71,72]
Male mice	PFOS	0.1, 0.3, and 1.0 mg/kg	Oral gavage	PFOS promoted M1 macrophage polarization through the NF-κB pathway; Induced atherosclerosis, inflammation.	[73]
	PFOA, HFPO-DA	1 mg/kg BW daily and 5mg/kg BW daily for 90 days	Oral	Chronic exposure → hepatomegaly and hepatic inflammation (hepatotoxicity); Alterations in genes involved in stimulus hepatic stress-sensing genes; Disruption of bile acid metabolism.	[74]
Obese (KK.Cg-Ay/j) and lean (KK.Cg-a/a)	PFOA	2.5 mg/kg for 15 days (obese and lean group)	Oral	↓ ovary weight in lean but not obese mice; Altered ovarian abundance of proteins involved in DNA damage sensing; Repair and reproduction pathways altered	[75]

				differentially in lean and obese mice → reduced female fecundity.	
female mice		0.5 mg/kg/day for 100 days	Oral (water)	↑ liver weight in both groups; Serum metabolites affected by PFOA differed due to obesity; Obesity impacted PFOA-induced hepatic chemical biotransformation gene mRNA changes.	[76]
Obese male mice				Abnormal behavior; neuroinflammation; activated glial cells; decreased nerve growth factor; altered gut microbiota.	[77]
Kunming pregnant mice	PFOA	1, 2.5, 5, and 110 mg/kg bw for 17 days	Oral gavage	Adverse effects in growth and development of the pups; Liver damage, disrupting the secretion of enzymes involved in fatty acid oxidation induced by PPARα → liver oxidative stress and ↓degree of histone acetylation. ↑ HDAC → downstream fatty acid metabolism disorders through PPARα.	[78]
		0, 2.5, 5, 10 mg/kg daily for 13 days		↓ placental weight and interstitial edema of placenta; ↓ numbers of uNK cells; Upregulated levels of Bax and cleaved-caspase 3 proteins; Rupture of nuclear membrane and fragmentation.	[79]
Kunming pregnant mice	PFOA	1,5,10,20, and 40 mg/kg bw for 7 days	Oral gavage	↑ liver weights and ↓uterus index in a dose-dependent manner; ↑ doses of PFOA → ↓ levels of SOD and GSH-Px; 20 and 40 mg/kg	

				of PFOA → more substantial harm to the uterus; ↑ expression of FAS, FASL, Bax, and Caspase-3 in decidual cells of the uterus; Oxidative damage may be one of the mechanisms by which PFOA induces liver toxicity.	80
		3.5 mg/kg daily for 17 days		Altered expression levels of circadian rhythm-related genes mainly in the PPAR signaling pathway in kidneys of offspring mice; Oxidative stress in the kidneys.	81
NMRI pregnant mice	PFOA	1,10, and 20 mg/kg for 9 days	Intraperitoneal	↓ weight of fetus and placenta, ↓ length of fetus and ↓ diameter of placenta; Pathological abnormalities in liver, brain, heart, and placenta; Adverse effects on embryofetal development due to mitochondria dysfunction.	82
SPF pregnant mice		5 mg/kg bw daily for 28 days (E13 until delivery of pups)	Intragastric infusion	PFOA compromises oocyte and embryo development, and ovarian follicle growth; Gut microbiota dysbiosis may reshape host metabolism → fetal growth retardation; Restoration of gut microbiota, activation of AMPK improves fetus growth.	83
APOE*3-Leiden.CETP transgenic mice	Ammonium PFOA	10, 300, or 30 000 ng/g daily for 4 or 6 weeks	Oral (diet)	No alterations in plasma lipids in the 10 and 300 ng/g dose groups, while at 30 000 ng: ↓ TGs, ↓ TC, ↓ non-HDL-C, and ↑ HDL-C; Very low-density lipoprotein (VLDL) production and ↑VLDL clearance by the liver through ↑ lipoprotein lipase activity; Changes in lipoprotein metabolism mediated through activation of the PPARα.	84

Male and female humanized PPARα mice	PFOA and American diet (29 PFAS analyzed)	American diet and control drinking water or with PFOA (8μM) for 6-7 weeks	Oral (diet, water)	↑ serum lipoprotein cholesterol in male mice; hPPARα and CAR activated in liver at a human relevant exposure level; Altered expression of genes involved in cholesterol homeostasis; Females more sensitive to changes in gene expression; Both PPARα-dependent and -independent effects were induced by PFOA.	85
Male WT, PPARα- null and PPARα-humanized mice on Sv/129 genetic background	PFOS	5 mg/kg bw (0.003 %) for 28 days or 10 mg/kg bw (0.006 %) for 7 days	Oral (diet)	PFOS induced PPARα-dependent peroxisome proliferation and ↑ Acox1, Cyp4a10 in WT, but not in PPARα- null or PPARA- humanized mice; CAR (Cyp2b10) and PXR (Cyp3a11) targets ↑ in all genotypes; Hepatomegaly occurred independent of PPARα.	86
White-footed mice (*Peromyscus leucopus*)		0.2, 1, and 5 mg/kg daily for 7 days	Oral gavage	Prenatal exposure to PFOS caused mortality in neonatal mice; Adult mice exposed to PFOS had ↑ liver effects and ↓ serum thyroxine.	87
Deer mice (*Peromyscus maniculatus)*	PFHxS	0, 1.6, 3.5, 7 and 14 mg/kg daily for 7 days	Oral gavage	Maternal exposure to PFHxS increased stillbirths in deer mice; Adult mice exposed to PFHxS showed ↓ plaque forming cells; PFHxS functional immunity data are novel and valuable for risk estimation.	88
Mouse preimplantation embryo	PFOS and F-53B	10 or 100 nM and 2 or 20 nM (zygotes or embryonic stem cells, ESCs)	Injection	Zygotes exhibited higher reactive oxygen species (ROS) activity in 8-cell embryos; PFOS and F–53B significantly affected the proportion and aggregation of the inner cell mass in the blastocyst → disrupted development and differentiation.	89

Zebrafish eleutheroembryos	PFOS	0.03-1.0 mg/L from 48 to 120 hpf (transcriptome study); 0.10–100 mg/L (morphometric tests)	Static renewal waterborne	Transcriptomic changes at concentrations 1/10 to 1/100 of the macroscopic LOEC; alterations in lipid metabolism and the immune system. Some of the observed changes occurred at concentrations already detected in humans.	(Martínez et al., 2019)
Zebrafish embryos (AB strain)		0.37–3.26 mg/L from 7 to 120 hpf; with/without chorion	Static waterborne	↑ mortality, spinal curvature, uninflated swim bladder/abnormal orientation; earlier onset and higher sensitivity in dechorinated embryos; dysregulation of PPAR signaling; upregulation of PPAR coactivators; downregulation of multiple lipid metabolism genes.	[90]
		0, 0.1, 1, 5, 10, 20 µM (4-120 hpf)	Static waterborne in multi-well plates	↑ duration of bursting behavior in zebrafish larvae; seizures in the brain; alterations in expression of seizure-related transcripts; influence on the endogenous concentrations of some neurochemicals.	[91]
Zebrafish embryos (Tübingen strain)	PFOS	0, 100, 500, and 1000 µg/L (4-120 hpf)	Static renewal waterborne	Developmental malformations and neurotoxicity; ↓ locomotor movement, affecting behavior; microglial activation led to neuronal inflammation and apoptosis; dopaminergic neurotoxicity.	[92]
Zebrafish embryos	PFOS or PFOA	25 µg/mL, 250 µg/mL and 2.5 mg/mL within 2 hpf	Microinjection	Specific dysregulated lipid classes in PFAS-treated embryos; Lipidomic dysregulation varied, reflecting distinct toxic effects of PFOS and PFOA; Downregulated docosahexaenoic acid.	[93]

	PFOS or PFBS	PFOS: 0-200 μM. PFBS: 0-20,000 μM (developmental toxicity); Sublethal BMDL-based for behavior: PFOS (0.01-10 μM), PFBS (7-7000 μM), 4-120 hpf	Static waterborne	PFBS ↓ developmental toxicity 700 times more than PFOS in zebrafish embryos; PFBS neurotoxicity mechanism was associated with oxidative stress, lipid metabolism, and glycolysis/glucogenesis; similar neurotoxicity mechanisms between PFBS and PFOS.	[94]
	PFOA, PFOS or PFOSA	1 and 10 μg/L, <4 hpf - 120 hpf	Static renewal waterborne	PFAS-induced pericardial edema; Behavioral-level alterations and changes in cardiac output and abnormal cardiac morphology after PFAS exposure; PFOA and PFOSA induce more severe response compared to PFOS	(X. Liu et al., 2022)
Zebrafish embryos (AB strain and transgenic line for heart imaging)	PFOSA	0.1, 1, 10, 100 μg/L (1- 120 hpf)	Waterborne exposure in glass Petri dishes	Transcriptional changes; Abnormal cardiac morphology; Disordered heartbeat signals; ↓ heart rate and cardiac output.	[95]
Zebrafish embryos (AB strain)	PFOA, PFHxA, PFBA	Acute toxicity: PFOA 0-900 ppm, PFHxA 0-10,000 ppm, PFBA 0-10,000 ppm (1-72 hpf)	Static renewal waterborne	↓ LC_{50} of PFAA with ↑ in chain length; PFOA caused hyperactivity and PFBA hypoactivity; PFOA, PFHxA, and PFBA exposure caused morphological alterations specific to each.	[96]
	PFHxA	0, 0.48, 2.4, 12 mg/L for 96 h		↓ thyroid-stimulating hormone β (tshβ) contents in zebrafish larvae; Alterations of THs levels → thyroid endocrine disruption; Genes involved in HPT axis modulated after PFHxA exposure.	[97]

	PFHxS	0.3, 1, 3, 10 μM (exposures started <4 hpf → sampled at 4, 24, 48, 72, 120 hpf)	Static renewal waterborne	Dysregulation of lipid metabolism in developing zebrafish; Remodeling of glycerophospholipid composition in zebrafish embryos; Oxidative stress and inflammation in embryonic zebrafish.	98
	HFPO-DA (GenX) or PFBS	LC50 tests: GenX 0-10,000 ppm, PFBS 0-2000 ppm Sublethal assays: 0.4- 400 ppb (dopamine, 1- 72 hpf); 4- 4000 ppb (behavior, 1–72 hpf)		GenX: LC_{50} ~8617 ppm; Bioaccumulation in tissues; changes in locomotor activity (at 40–4000 ppb); ↑ dopamine at 40 ppb. PFBS: LC_{50} >2000 ppm; Higher bioaccumulation than GenX; Induced hyperactivity across light/dark phases (from 40 ppb); ↓ dopamine at 400 ppb.	99
	HFPO-DA (or GenX)	0.5- 20,000 mg/L, bioaccumulation assessed at 4000 mg/L (3- 4 hpf → 72 hpf)		↑ heart rate in embryos exposed to 2 mg/L and 10 mg/L; Spinal deformities and edema phenotypes in higher concentrations; Seven downregulated genes were associated with visual response, and seven upregulated genes associated with the cardiovascular system.	100
Zebrafish embryos (AB strain, WT and transgenic lines)	HFPO-TA	20- 400 mg/L for LC_{50} determination; sublethal exposures at 50, 100, 200 mg/L and 60, 120, 240 mg/L (S. Sun et al., 2024) from 6 hpf → 96- 120 hpf (in both studies)	Waterborne static renewal (in well plates)	↓ liver size of zebrafish larvae; Alterations in gene expression of PPAR signaling pathway → lipid metabolism disruption and inflammatory response; ↓ bile acid synthesis but promotes bile acid transport. Developmental toxicity, especially heart development; Sprouting angiogenesis, and disruption of gene expression of VEGF signaling pathway in vascular development; Oxidative stress → apoptosis.	101,102

Zebrafish embryos (AB strain)	6:2 Cl-PFESA (or F-53B)	0, 0.5, 20, 200 µg/L from 2 hpf to 120 hpf	Waterborne static renewal (in glass beakers)	↑ ROS levels in zebrafish exposed to 20 and 200 µg/L; F-53B acts as an inhibitor of PI3K in the activation of the Nrf2-ARE pathway; ↓ activity and protein levels associated to oxidative stress (Nrf2, SOD, CAT) in 200 µg/L group.	103
	Nafion BP2 or PFOS	20, 40, 60, 80, 100, 120, 140, 160 mg/L Nafion BP2 or 2, 3, 4, 5, 6, 9, 12 mg/L PFOS from 0.5 hpf to 120 hpf	Waterborne static (in E3 medium)	↓ survival and hatching rates, ↑ malformations; intestinal structure disturbance and activation of intestinal inflammatory responses (e.g., C4, IL-6).	104
	PFOS, PFBS, Nafion BP1, Nafion BP2, F-53B, OBS, PFOA, PFUnDA, PFO5DoDA, HFPO-TA	Various concentrations depending on the PFAS, ranging from 2 to 700 mg/L (lowest: PFOS-highest: PFBS) from 0.5 hpf to 120 hpf		Malformations of zebrafish embryo; Developmental toxicity with all PFAS; PFUnDA exhibited the highest toxicity with lowest EC_{50} (4.36 mg/L) Common DEGs and pathways were consistent with the observed phenotypes.	105
Zebrafish embryos (Tübingen strain)	PFOA (ammonium salt) PFO3OA, PFO4DA, PFO5DoDA (potassium salts)	0–2400 mg/L from 0.25→ 5 dpf (continuous exposure)	Waterborne static renewal (in 6-well plates)	All caused dose-dependent malformations (mainly uninflated swim bladder); toxicity rank: PFO5DoDA > PFO4DA > PFOA > PFO3OA; ↓ T3/T4, dysregulation of thyroid-related; T3/T4 supplementation partially rescued swim bladder defects.	106
Zebrafish embryos (AB strain, WT and transgenic lines)	PFOS or 6:2 FTS	1, 5, 10, 20, 50 µM PFOS; 10, 50, 200, 500, 1000 µM 6:2 FTS; from 4 → 144 hpf	Waterborne static renewal in embryo medium	6:2 FTS induces vascular hyperplasia, inhibits atrial development, and ↓ blood flow velocity; ↑ activity of CAT and GSH-Px, involved in calcium signal transduction and myocardial contraction.	107

Zebrafish embryos (AB strain and transgenic lines)	6:2 FTCA	0.08, 0.8, 8 µg/mL from 4 → 144 hpf	Waterborne static renewal	Developmental effects; abnormalities in thyroid function and motor neuron development in zebrafish; TRβ (thyroid receptor) potential target of 6:2 FTCA → neuroendocrine disrupting effects.	[108]
Zebrafish embryos (AB strain)	6:2 FTSA	0.5 mg/L , from 2 dpf →5 dpf for 72 h	Waterborne semi-static	↓ SOD and LZM activities; ↑ expression of immune-related proteins (IL-1β, TNF-α, NF-κB, TLR4)	[109]
Zebrafish embryos (5D strain)	8:8 PFPiA	0.1, 0.3, 1, 3, 10 µM (≈92.4, 227, 924, 2272, 9240 µg/L) from ≤4 hpf → 144 hpf	Waterborne semi-static renewal	Upregulated genes related to thyroid hormones l (for neurodevelopment); ↓ global DNA methylation at higher treatment levels → effects on epigenetic regulation.	[110]
Zebrafish embryos (AB strain)	PFECHS	0.01, 0.1, 80, 150, 300, 600, 1200 µg/L from <2 hpf → 96 hpf		↑ abundance of transcripts of peroxisome proliferator activated receptor alpha (PPARα), cytochrome p450 1a1 (cyp1a1), and apolipoprotein IV.	[111]
Zebrafish embryos (AB strain and ; transgenic lines)	AFFF mixture, PFOS, PFHxS, PFOS + PFHxS (mixture)	AFFF: 4.40×10^{-6} to 3.52×10^{-3} % (mortality); PFOS: 16, 32 µM; PFOS + PFHxS mixture: 282.3 mg/L + 45 mg/L, from 3 hpf → 96 hpf	Waterborne static renewal	LC_{50} of the AFFF mixture at 96 hpf was calculated to be 7.41×10^{-4}% AFFF; effects in development of larvae (e.g., shorter body length) after exposure to AFFF and PFOS/PFHxS; ↓ liver weight	[112]
Zebrafish embryos (AB strain)	9 PFAS (C_4-C_8 PFCAs,C_4, C_5, C_8 PFSAs and 6:2 FTSA) individuals and mixture of all nine PFAS	Individual PFAS: 0.0001–100 mg/L Mixture: 0.0001–30 mg/L from <2 hpf → 144 hpf	Waterborne static	PFASs significantly altered swimming behaviour in zebrafish embryos; short-chain PFASs caused behavioural toxicity; PFAS mixture was less potent than some individual PFASs; Toxicity was related to chain length	[113]

				and functional group.	
Zebrafish embryos (5D strain)	38 PFAS (screened) PFOSA (follow-up)	Screening: 50 μM for each of 38 PFAS. PFOSA concentration–response: 6.25, 12.5, 25, 50 μM (until 12 hpf); Liver/lipid assays: 0.78 μM (until 24 hpf)	Waterborne static	PFOSA induced developmental delays up to 12 h → embryotoxic; PFOSA-exposed embryos were deficient in liver development; mRNA-sequencing identified hepatotoxicity and lipid transport as affected pathways.	114
Zebrafish embryos (5D strain, dechorionated)	139 PFAS (diverse PFCAs, PFSAs, FTSA, fluorotelomers, precursors)	10-point concentration range 0.015- 100 μM (nominal) from 6 → 120 hpf		49 PFAS bioactive; PFOSA bioactive in all assays, while PFDA was the most potent teratogen; Low PFAS volatility is associated with developmental toxicity.	115
Zebrafish embryos and larvae	PFOS	16 or 32 μM at 3 hpf followed up to 30 dpf	Waterborne static renewal	↑ embryonic saturated fatty acids and ↓ PPAR gene expression; ↑ pancreatic islet areas; ↑ lipid staining and the number of lipid droplets.	116
		0, 0.032, 0.32 and 3.2 mg/L (2-120 hpf)		ROS generation in larvae head; alterations in mRNA and protein levels involved in dopamine pathway; neurobehavioral effects.	117
Zebrafish embryos and larvae (AB strain)	PFOS or PFOA or PFOA+PFOS	7, 70, 700 ng/L PFOA; 24, 240, 2400 ng/L PFOS; PFOA/PFOS half doses, 1:1; from ≤4 hpf to 5 dpf	Waterborne static renewal	F0: PFOA ↓ locomotion, PFOS ↓ activity (light only), mixture ↑ locomotion; No malformations. F1: PFOA ↑ locomotion, PFOS ↑ in dark↓ in light, mixture strong ↓ activity; fecundity ↓ at PFOA 70 ng/L; some sex ratio shifts. F2: PFOA ↓ locomotion (all doses), PFOS ↑ locomotion (24 ng/L); DEGs linked to lipid metabolism, xenobiotic	118

				signaling, immune and epigenetic pathways.	
Zebrafish embryos and larvae (AB strain)	PFOS or PFOA	0.01, 0.1, 1.0 μM or 0.1, 1, 10, 100 μM (5 hpf - adulthood)	Waterborne static renewal (Petri dishes)	Distinct behavioral changes in zebrafish; PFOA ↑ larval motility in the dark, while PFOS reversed light-dark response in the larval motility test; PFOS caused hypoactivity in adulthood and reduced acoustic startle magnitude in adolescence.	119
Zebrafish embryos and larvae (AB strain and transgenic lines)	PFOS or PFOA	PFOS: 17, 14, 28, 56 μM or PFOA: 64 μM (4 hpf-5 dpf)	Static waterborne Injection	Functional and morphological differences in exposed larvae; Upregulation of the microglia activation gene; ↑ microglia responses to brain injury in the absence of inflammation; PFOS-exposed larvae: neurochemical signatures of excitatory–inhibitory imbalance.	120
	PFOS or OBS	0, 20 mg/L PFOS or 20, 30 mg/L OBS for 96 h	Waterborne static renewal	OBS caused hatching delays, body axis curvature, neurobehavioral inhibition and abnormal cardiovascular development. Developmental effects induced by OBS milder than that of PFOS	121
Zebrafish embryos and larvae	PFOS or OBS	PFOS: 0 -20 mg/L or OBS: 0- 40 mg/L; from 6 hpf → 7 dpf (larval stage)	Waterborne (in embryo culture medium)	OBS disrupted folding of intestinal fold and PFOS led to muscle cell necrosis; Both PFAS caused oxidative stress and inflammatory responses; OBS had less effect than PFOS on immune related gene expression.	122
		PFOS: 20 mg/L or OBS: 20, 30 mg/L for 4 days	Waterborne static renewal	OBS induced less developmental toxicity on body growth than PFOS; Both PFAS trigger thyroid dysfunction (THs synthesis, conversion from T4 to T3) and osteoclast differentiation; OBS has more severe	123

				disruptive effects on thyroid function than PFOS.	
Zebrafish embryos and larvae (transgenic lines)	PFBS	8.25, 82.5, 825, or 8250 µM at 3 hpf (for survival, hatching, and swim bladder inflation) and 16 or 32 µM at 1 dpf	Waterborne static renewal	delayed swim bladder inflation, and impaired yolk utilization; ↑ truncated exocrine pancreas length; ↑ of severely hypomorphic islets and ↑ occurrence of fragmented islets; RNA-Seq data also identified disruptions in regulation of lipid homeostasis.	124
Zebrafish embryos and larvae (AB strain, transgenic line for GABAergic neurons)	PFNA	0.01, 0.1, 1, 10, 100 µg/L Embryos <4 hpf exposed until 120 hpf, larvae assessed at 96 and 120 hpf		↓ neurotransmitter levels (acetylcholine, glutamate, 5-hydroxytryptamine, γ-aminobutyric acid, dopamine, and noradrenaline) in zebrafish larvae and ↓ GABAergic neurons in transgenic line, accompanied by altered swimming behaviors and reduced activity.	125
Zebrafish embryos and larvae (AB strain and transgenic lines)	PFBA, PFOA, PFNA	0.1, 1, 10 µg/L (4-120 hpf)		↑ PFAS-induced immunotoxicity with longer carbon chain lengths; Pivotal role of toll-like receptor (TLR) in mediating PFAS immunomodulatory effects; MyD88 key modulator in PFAS-related immune effects.	126
Zebrafish embryos and larvae (AB strain)	PFHxA, PFHxS, 6:2 FTOH	LC_{50} assays: 0–1000 µM Sublethal: 0, 0.02, 0.2, 2, 20 µM (3 hpf - 120 hpf)	Waterborne static, non-renewed	PFHxA was the most acutely toxic, yet caused no behavioral or morphometric effects; PFHxS impacted morphometric and behavioral endpoints at high doses; 6:2 FTOH impacted gene expression and behavioral endpoints.	127

	PFHxS	0.1 -10 μM (1–4 cell stage)	Waterborne static renewal	Larvae malformations; ↑ glucose and lipid accumulation → oxidative stress; ROS by altering GSH balance; developmental toxicities; effects in cell cycle.	128
Zebrafish embryos and larvae (AB strain)	6:2 Cl-PFESA (or F-53B)	Acute tests**:** 0 - 30 mg/L (embryos, 4 hpf → 96 hpf); 0 - 6 mg/L (larvae, 72 hpf → 96 h exposure)	Waterborne static renewal	Zebrafish embryos were more resistant to acute exposure to F53B than larvae; Body growth of zebrafish larvae was inhibited by F53B; More severe immunotoxicity in larvae than embryos.	129
	PFOS, Cl-PFOS, 6:2 Cl-PFESA, 8:2 Cl-PFESA	1 μM From 6 hpf → 7 dpf (larval stage)		Hepatic steatosis in zebrafish larvae; Cl-PFOS, 6:2 Cl-PFESA and PFOS disrupted lipid metabolism similarly; 8:2 Cl-PFESA caused lipid alterations in different way.	130
	PFOS, Cl-PFOS, 6:2 Cl-PFESA, 6:2 H-PFESA	0.01, 0.1, 1 μM from 4 hpf → 6 dpf (free-swimming larvae stage)	Waterborne static (in embryo rearing water)	PFOS, Cl-PFOS, and 6:2 Cl-PFESA accumulated similarly and disrupted thyroid hormone homeostasis, altered HPT-axis genes; competitive binding to TTR and interference with Na^+/K^+ transport; 6:2 H-PFESA weaker effects.	131
Zebrafish embryos and larvae (strains AB/Tupfel Long Fin mixed)	ADONA, GenX, PFESA1, PFHxA, PFHxS, PFOA, PFOS, PFBS, PFPeS, PFHpS	0.04–80 μM (most PFAS); 0.2–3.1 μM PFOS; Sulfonic acids 1.7–100 μM (from 4–6 hpf → 6 dpf (larval stage)	Waterborne semi-static renewal	PFHxS or PFOS exposure caused failed swim bladder inflation, and hyperactivity; PFHxA caused a unique hyperactivity signature; Developmental toxicity.	132
Zebrafish embryos and larvae	PFBS, PFPeA, FBSA, 4:2 FTS	PFBS 0.6–57 μM; PFPeA 0.7–69 μM; FBSA 1.0–100 μM; 4:2 FTS 0.9–85 μM; from 8 hpf → 120	Waterborne static	All induced abnormal larval behavior; only FBSA caused morphological defects (craniofacial abnormalities) ; FBSA → transcriptomic disruption linked to lipid	133

(5D strain, pathogen-free)		hpf; behavioral endpoints at 24 hpf and 120 hpf		metabolism, PPARα, RXRα, and AHR signaling; Bioaccumulation rank: FBSA > PFBS > 4:2 FTS > PFPeA.	
Zebrafish larvae (AB strain)	PFOA, GenX, and HFPO-TA	PFOA 100 μM, GenX 200 μM and HFPO-TA 30 μM (72 hpf → 72 hpe)	Waterborne static renewal	PFOA & HFPO-TA caused malformations (pericardial edema, ↓swim bladder), altered heart rate, lipid accumulation (↑TC, TG, LDL-C), and neutrophil liver infiltration; GenX less severe but still altered lipids; broad DEGs, enriched in PPAR and lipid metabolism pathways.	134
Zebrafish larvae and adult (AB strain)	PFOS, 6:2 Cl-PFESA, and 8:2 Cl-PFESA	0.1, 0.5 μM for 7 days (larvae) and 28 days (adult, gonadal histopathology)	Waterborne static	↑ VTG, ↑ E2, ↑ testosterone after exposure to all PFAS; 6:2 Cl-PFESA exhibited stronger estrogenic activity with ↑ ERα and ERβ1, while PFOS and 8:2 Cl-PFESA, showed weaker activity and ↓ CYP19b; Histopathology in adult zebrafish showed reproductive toxicity: ↑ early oocytes, ↓ mature oocytes and ↓ spermatocytes.	135
Zebrafish larvae and adult (AB strain and larvae transgenic line for liver development)	PFOS, F-53B, and OBS	Adults: 1 μM for 21 days; Larvae: 5 μM at 72 hpf for 3 days	Waterborne semi-static renewal	Hepatotoxicity to adult zebrafish: F-53B > PFOS > OBS; Alterations in liver histopathology and liver function; PFOS and F-53B → aberrant hepatic lipid metabolism through PPARγ; Gut microbiota dysbiosis plays role in PFAS-induced toxicity through PPARs.	136

Adult male Zebrafish (AB strain)	PFOS	0, 0.02, 0.04, 0.08 mg/L dissolved in dechlorinated-tap water for 21 days	Waterborne static renewal	Disturbance in immunoregulatory function; Effects on the liver structure, enzyme activities and mRNA expression in immune system; Pro-inflammatory effect of hepatocytes; NF-κB signaling pathway involvement in the action mechanism.	137
Adult male Zebrafish (AB strain)	PFOS	0, 2, 20, 200 µg/L for 21 days	Waterborne semi-static	Sexual behaviors; Downregulation of the genes gonadotropin-releasing hormone (GnRH), gonadotropin-releasing hormone receptor (GnRHr); Zebrafish testes are more sensitive than brains and livers after exposure to PFOS; Reproductive toxicity.	138
		0, 0.1, 0.6, 3.2, 20, 100 µg/L; P: 7 hpf → 180 dpf; F1: 7 hpf → 180 dpf; F2: 7 hpf → 16 dpf	Waterborne continuous (renewal 5-30 dpf)	Adverse effects on body weight and length at the highest exposure treatment; Threshold for ecologically relevant adverse effects in zebrafish at 47 µg/L for all statistically significant negative effects.	139
Adult female Zebrafish	PFOA	Acute LC_{50} test: 250–500 mg/L (for 96 h); 100 mg/L for 15 days	Waterborne semi-static	↓ Fertilization & hatching rates; ↑ embryo malformations; ovarian damage, vacuolation, fewer mature oocytes; immune genes upregulated	140
Adult Zebrafish (AB strain)	PFOS	0.1 mg/L and 1 mg/L	Waterborne static renewal	PFOS bioaccumulation (highest in male liver); Altered in liver, intestine, ovary, heart; Sex- and tissue-specific patterns; Up-/down-regulation of neural genes (ChAT, AChE, hdac6, ngf, bdnf); ↓ ChAT in muscle; Sex-dependent bdnf/ngf responses in muscle.	141

	PFOA	0, 0.05, 0.1, 0.5, or 1 mg/L for 7, 14, and 21 d		Regulation of immunoglobulin levels by interfering with IFN and IL-1β expression. Effects on the NF-κB pathway by attacking TLRs; Disturbances in the secretion of cytokines and antibodies by affecting the TLR/myd88/NF-κB pathway.	142
Adult Zebrafish	PFOA, HFPO-DA, and HFPO-TA	5 μg/L and 500 μg/L for each PFAS, 14 days exposure	Waterborne static renewal	HFPO-DA and HFPO-TA induced comparable inflammation and apoptosis with PFOA; Correlations between gut microbiota and toxic effects.	143
	HFPO-TA	5, 50, 100 μg/L (I) fertilization: 21 dpf (early development), (II) 21–42 dpf (putative gonadal differentiation), (III) 42–63 dpf	Waterborne exposure (renewed every 48 h)	Reproductive toxicity, growth inhibition; ↓ condition factor, ↓ gonadosomatic index, and ↓ average number of eggs; Upregulation of the genes for cytochrome P450 A1A, vitellogenin 1, estrogen receptor alpha, and estrogen receptor 2b; HFPO-TA similar toxicity to PFOA.	144
	6:2 Cl-PFESA (or F-53B)	0, 5, 50 μg/L for 180 days; F1 embryos collected at final spawning	Waterborne flow-through	Sex-dependent accumulation of F-53B in liver; Hepatomegaly and ↓ liver triglyceride levels; Differences between male and female hepatic transcripts; F-53B interferes with the PPAR pathway in adults and their offspring.	145
	PFOS, F-53B, and OBS	1 μM for each PFAS, continuously exposed for 21 days	Waterborne semi-static renewal	Alterations in the histopathology of zebrafish intestine and head kidney; anti-inflammatory effects on zebrafish liver; immunotoxicity associated with intestinal microbiota dysbiosis in adult zebrafish.	122
Kras(v12) transgenic	PFOS	Larvae: 0, 50, 100, 200, 500, 1000 μg/L (4–8 dpf,	Waterborne static renewal	PFOS promoted DOX-induced liver enlargement and hepatocellular carcinoma progression in zebrafish liver; Metabolic	146

zebrafish Tg (larvae/adults)		with doxycycline, DOX); Adults: 500 μg/L PFOS		reprogramming of krasV12 transgenic zebrafish liver; ↓ vitamin D level and ↑ FA intake → responsible for tumor-promoting effects of PFOS.	
Zebrafish Nrf2a WT or Nrf2a mutant	PFBS	F0 females: 0.08, 0.14, 0.25 mg/L for 7 days. F1 embryos collected	Waterborne semi-static renewal	Maternal preconception exposure in zebrafish altered egg and embryo development. Alterations in nutrient profiles and fatty acid composition with unique patterns at different larval ages and in Nrf2a mutants.	[147]
C. elegans	PFOS	1 to 200 ppm (≈2–400 μM); mechanistic studies focused at 75 ppm (~150 μM) for 72h	Waterborne in plates with bacterial food	Dopamine-dependent functional deficits, without altering acetylcholine-dependent paralysis; Effects on mitochondrial content; ↑ oxidative stress; Mutation in mitochondrial SOD rendered animals more vulnerable.	[148]
		0.01, 0.1, 0.5, 1 μM for 72h		Expression of the fatty acid (FA) desaturation gene fat-3 was down-regulated → FA disorder is associated with decrease in mRNA expression of Δ6-desaturase genes in *C. elegans*; Disorders in FA metabolism lad to disruption of mitochondrial function with a reduction in ATP synthesis.	[149]
		0.001, 0.01, 0.1, 0.5, 1.0, 2.0 μM for 48h (acute and behavioral assays)	Waterborne in liquid K-medium with bacterial food	Trans-generational effects of PFOS in *C. elegans*; Effects on locomotion, reproduction, lifespan & chemotaxis behavior; PFOS ≥ 0.01 μM caused severe toxicity to locomotion and effect transferred to progeny; Neurobehavioral defects can transfer from P0 to F1.	[150]

C. elegans	PFOA or PFOS	0.5 and 2 mg/L, respectively for 24h at 20° C	Waterborne in liquid medium	Reproduction was impeded; PFAS triggered oxidative stress of *C. elegans*; Alterations in amino acid and lipid metabolism.	151
	PFOA or PFOS	0.1, 1, 10, 100 µM for 72h	Waterborne in liquid K-medium with bacterial food	Lipid accumulation independent of feeding behavior; Alterations in the fatty acid composition in *C. elegans*; PFOA or PFOS-induced obesity associated with fatty acid desaturation and triglycerides synthesis; Disruption of lipid metabolism.	152
	PFOA	Acute lethality: 0.25–500 µM; Other assays: 0.001–2 µM (for 48 h)		PFOA affected lifespan and reproduction at environmentally relevant concentrations. LC_{50} of PFOA in *C. elegans* was 1.83 mg/L.; PFOA contamination caused negative outcomes on the food-chain.	153
	PFOS or PFBS	PFOS 10, 20, 40 µM or PFBS 1000, 2000 µM for 1-2 days	Liquid exposure in S-complete medium	Reproductive toxicity; Preconception exposure of PFOS and PFBS led to offspring physiological dysfunctions; Abnormalities in growth rate, body size and locomotive activity in F1 offspring.	154
	PFBS	0.001–50 mM; Behavioral assays: 0.0005, 0.01, 0.1, 0.5, 1.0, 2.0 mM; parental-only exposures at 0.01, 0.1, 1.0 mM for 48h	Waterborne exposure in K-medium with bacterial food	↓ life span and brood size in P0 nematodes following exposure were not transferred to the progeny; retardance in the locomotion behavior of P0 worms and at higher concentrations (e.g.,1.0 mM) was transferred to F1; Chronic exposure to PFBS → behavioral toxicity.	155
	lab reconstituted PFAS mixtures from environmental water (PFBA, PFPeA, PFHxA,	$PFAS_H$ (high) and $PFAS_L$ (low): 1000 times diluted $PFAS_H$ for 48 h and		Effects in innate immune response gene expression; ↓ immune surveillance to Gram-positive bacteria; ↑ expression of hemolysin	156

C. elegans	PFHpA, PFOA, PFNA, PFDA, PFBS, PFHxS, PFOS, 6:2 FTS)	followed up to 7 days	Waterborne exposure in K-medium with bacterial food	A and virulence gene regulator in *S. aureus*; ↑ *S. aureus* mediated intestinal permeability and death in nematodes	
	PFBA, PFHxA, PFOA, PFBS, PFHxS, PFOS, NEtFOSAA, 6:2 FTS, PFOSA, HFPO-DA	0.1, 1,10, 100, 200 μM for 72 h		In the tested endpoints (e.g., growth, development, fecundity, and behavior) PFOS was found to be the most toxic among the 10 PFAS; larval stages were the most vulnerable to PFAS exposure.	157
Chicken embryos/ eggs	PFOA	0, 0.5, 1 or 2 mg/kg	Air cell injection	PPARα-silencing lentivirus was co-applied with PFOA exposure; Developmental exposure to PFOA resulted in persistent cardiotoxicity, but not hepatotoxicity; PPARα involved in both cardiotoxicity and hepatotoxicity.	158
		0, 0.5, 1 or 2 mg/kg		Developmental exposure to PFOA resulted in miRNA changes in hatchling chicken heart; miR-490-5p-SNAP91/LYPD6 is involved in PFOA-induced developmental cardiotoxicity.	159
		2 mg/kg		Alterations in primary cardiomyocyte viability, morphology, and ↑ calcium level; PPARα silencing may partially alleviate PFOA-induced cardiomyocyte toxicities.	160
	HFPO-DA	0, 1, 2, 4, 8 mg/kg		Developmental cardiotoxicity (thinned right ventricular wall and ↑ heart rates); Developmental hepatotoxicity in the form of steatosis; Silencing of PPARα alleviated such effects; PPARα participates in such toxicities, with some differential downstream gene (e.g., CD36. enoyl-CoA hydratase and 3-hydroxyacyl CoA) regulation in different	161

				organs (heart, liver).	

Legend: BW= bodyweight; dpf= days post-fertilization; F1= first generation; GD= gestational day; hpe/f= hours post-exposure/fertilization; KLH- keyhole limpet hemocyanin; P0= parent; PD= postnatal day; RfDs= reference doses SD= Sprague Dawley; SPF= specific pathogen-free; VTG= vitellogenin; WT= wildtype.

Table S4. Summary of in vitro PFAS toxicological studies, including the substances used in each study, exposure concentrations, cell lines, (human) tissue types, and main observed effects.

Cell line	Human Tissue Type	PFAS used	Concentration and Time of Exposure	Main Effects	Study
HepG2	Liver - Hepatocellular Carcinoma	PFOS, PFOA, PFNA, PFDA, PFHxS, PFHpA; plus binary/ternary/multi-component mixtures	Singles tested across ranges to derive IC_{50}; mixtures prepared at equipotent ratios for 24h	Singles IC_{50} :↑chain length ↑potency; sulfonates > carboxylates at same chain. Binary interactions: mostly synergistic (e.g., PFOS+PFOA PFOS+PFDA, PFNA+PFDA strong synergy); antagonism seen for PFOA+PFHxS, and for PFHxS+PFHpA & PFOA+PFHpA at low–mid effect levels. Ternary/multi-component: generally synergy at low–mid effects, trending to additivity/antagonism at high effects; PFOS-containing mixes more consistently synergistic than PFOA-containing ones.	162
		PFOA, GenX	PFOA: 10–600 µM (MTT), 20–200 µM for gene expression; GenX: 20–1000 µM (MTT), 100–600 µM for gene expression for 48h	PFOA caused dose-dependent global DNA hypomethylation with ↓TET1 and ↑TET2/TET3; dysregulated lipid genes; cyclins ↓ at higher doses; viability fell at ≥400–600 µM. GenX produced weaker effects: modest/non-monotonic methylation changes, smaller shifts in lipid/cell-cycle genes, viability peaked ~200 µM then declined at higher doses.	163
		PFOA	100 & 300 µM for 24 h	Metabolic flux disruption: ^13C incorporation ↓ into TCA intermediates and serine; de novo lipogenesis from glucose ↓. Mitochondrial respiration inhibited at 300 µM. ROS ↑. G0/G1 arrest ≥100 µM; viability ~80% at 100–300 µM, ~10% at 600 µM.	164

HepG2 WT; stable HepG2 HaloTag vs HaloTag-SPNS1 (Spinster-1)	Liver - Hepatocellular Carcinoma	PFOS	200 μM for 12 h	SPNS1 overexpression localized to lysosomes and increased lysosomal acidification. PFOS caused lysosomal membrane permeabilization (↑ cytosolic cathepsin D) and impaired autophagosome degradation. Overall: PFOS → autophagy activation → ↓ Tyr-α-tubulin → weakened SPNS1–tubulin coupling → lysosomal membrane destabilization.	[165]
HepG2	Liver - Hepatocellular Carcinoma	PFOA	0 to 1000 ng/mL (0–2.4 μM) for 24h	Insulin-stimulated glycogen synthesis reduced even at 0.1 ng/mL PFOA; glucose uptake blunted ≥10 ng/mL; Total GLUT4 unchanged, but insulin-stimulated membrane translocation impaired; InsR protein unchanged; basal p-InsR increased ≥10 ng/mL; Insulin-stimulated p-Akt and p-GSK3β significantly reduced ≥1 ng/mL.	[166]
		PFOA, PFOS, PFDA, PFNA, PFHxS and binary mixtures: PFOS + PFOA, PFOS + PFDA, PFOS + PFNA, PFOS + PFHxS, PFOA + PFDA, PFOA + PFNA, PFOA + PFHxS	0.2–20 μM (singles and pairs) for 24 h	Cytotoxicity and GSH depletion increased with chain length (PFDA > PFNA > PFOS > PFOA ≫ PFHxS); ROS largely unchanged (PFDA 20 μM lowered ROS); binary mixtures were additive with strongest effects at 20 μM+20 μM.	[167]

HepG2	Liver - Hepatocellular Carcinoma	PFAS library (150 samples; 142 unique parent structures), incl. PFOA, PFOS, PFNA, PFDA, GenX (ammonium salt)	8-point series up to 300 µM for 20–24 h exposure	Frequent activation of PPARα, PXR, and RXRβ; weaker/partial ER activity for a subset; minimal/weak AhR activity; NRF2 (ARE) stress responses observed for several PFAS; strongest from 3,3-bis(trifluoromethyl)-2-propenoic acid; PFOA's PPARα/γ activation blocked by selective antagonists; radioligand assays confirmed PPARγ and RXRβ binding for representative PFAS. GenX: ammonium salt in H_2O active (PPARα agonist), free acid inactive in DMSO/H_2O.	[168]
		PFOA	0–600 µM for 24 h (MTT); 10, 25, 50 µM for 24 h (apoptosis/necrosis, ROS, cytokines)	Concentration-dependent cytotoxicity; ~80% cell death at ≥400 µM. Low–mid doses shifted death mode from apoptosis (higher at 10 µM) toward necrosis as dose increased (more necrosis at 50 µM). ROS increased at all doses; GSH and CAT increased only at 10 µM; SOD unchanged. Immune effects: IL-6 up to ~1.8-fold↑; IL-8 suppressed at 25–50 µM.	[169]
		PFOS, PFOA, PFNA, PFDA, PFHxS and their binary mixtures	0–400 µM for 24 h Individuals) and mixtures tested at equipotent 1:1 ratios for 24 h; Comet assay: singles at serial two-fold dilutions starting at 1/10 EC50	All five PFAS decreased viability concentration-dependently; Mixtures: PFOS+PFNA, PFOS+PFDA, PFOS+PFOA showed synergistic cytotoxicity; PFOS+PFHxS, PFOA+PFNA, PFOA+PFDA, PFOA+PFHxS were additive; PFOS-containing mixes generally more potent; Comet assay: singles and binaries produced modest but significant increases in %Tail DNA at several concentrations without clear dose-response; PFOA, PFDA, PFHxS significant at all tested doses; PFOS and PFNA significant at some doses. Overall, mixtures enhanced cytotoxicity more than genotoxicity	[170]

HepG2	Liver - Hepatocellular Carcinoma	PFOS	0, 50, 100 μM PFOS for ~16 h (overnight) for ROS/SECM assays; 0 and 100 μM PFOS for 48 h for time-course SECM (scanning electrochemical microscopy)	↑ intracellular ROS and electrochemical redox signal in a dose-dependent manner after 16 h; 48 h PFOS led to higher and time-increasing electrochemical response; ↓ extracellular GSH with PFOS; SOD1 inhibition further amplified the SECM signal, implicating superoxide anion–mediated electron transfer as the driver of PFOS-induced redox/cytotoxic effects (cells remained >95% viable at 16 h).	[171]
ARE reporter–HepG2	Liver - Hepatocellular Carcinoma	PFHxS, PFHpA, PFOS, PFOA, PFNA, PFDA (inviduals and/or mixtures)	3.1–400 μM for 24 h (cytotoxic screen), PFOS/PFOA/PFNA 0–5 μM; PFDA 0–2 μM; PFHxS/PFHpA 0–15 μM, for 24 h, and mixtures 1:1 equitoxic ratio based on concentration that induced an induction ratio (IR) of 1.5 (ECIR1.5) (Nrf2-ARE reporter)	All compounds activated Nrf2-ARE with potency trending by chain length and functional group (sulfonates > carboxylates at same length); Mixtures often exceeded concentration-addition (CA) predictions: binary synergism for PFOS+PFHxS, PFOS+PFOA, PFOS+PFNA, PFOS+PFDA; PFOA binaries mostly additive except PFOA+PFDA (synergistic). Ternary PFOS+PFNA+PFDA synergistic; others additive; Experimental ECIR1.5 generally lower than CA predictions, indicating additivity→ synergism dependent on composition.	[172]
HepG2 and HaCaT	Liver - Hepatocellular Carcinoma and Skin (keratinocyte)	PFOA	0.1 μM–1 mM for 6–72 h (MTT)	PFOA reduced viability at 10 μM/24 h and further at higher doses; ↑iNOS mRNA/protein (both lines); slight ↓eNOS mRNA (HepG2) and ↓nNOS mRNA (HaCaT); ↑ROS and ↑NOx; ↑MnSOD protein (both) and ↑cyt c mRNA/protein; baseline mitochondrial membrane potential (MMP) not decreased at 24 h, but ionophore test showed mitochondrial hyperpolarization with PFOA. Overall, PFOA induced nitro-oxidative stress and mitochondrial dysfunction.	[173]

HepaRG and HepG2	Liver - Hepatocellular Carcinoma	PFOA, HFBA, PFTA	10–1000 nM for 48h	Increase in ROS production and in expression of TNFα and IL-6; Upregulation of steatosis and fibrosis by activation of UPR pathway.	[174]
HepaRG	Liver - Hepatocellular Carcinoma	PFOA, PFOS, PFNA	up to 400 µM for 6, 24, or 72 h	BMC50 values; PFOA most potent in activating the PPARα response genes, followed by PFNA.	[175]
HepaRG (differentiated) and HepG2 (reporter)	Liver - Hepatocellular Carcinoma	PFOA, PFOS	PFOA: ≤500 µM (24 h), ≤250 µM (48 h); PFOS: ≤100 µM (24 h), ≤50 µM (48 h)	Broad downregulation of cholesterol/bile-acid genes; Strong CYP7A1 suppression at mRNA, promoter activity, and protein levels. At 24 h: PFOS (100 µM) ↑ cholesterol in supernatant; PFOA ↓ intracellular cholesterol; effects largely normalized by 48 h. Conjugated bile acids generally ↓ (cells & media) with biphasic rebound at highest doses; PFOA also altered unconjugated CA/CDCA (PFOS did not).	[176]
HepaRG and HEK293	Liver - Hepatocellular Carcinoma and Embryonic Kidney	PFOS, PFOA, PFNA, PFDA, PFHxA, PFBA, PFHxS, PFBS, HFPO-DA, ADONA	5mM for 72h, except PFOS, PFOA, PFNA, PFDA (up to 250µM)	Increased triglyceride accumulation after individual PFAS and PFAS mixtures exposure; steatosis; PPARα activation and up- or downregulation of PPARα target genes (e.g., PLINK2, ADH4, respectively).	[177]
SMMC-7721	Liver- Hepatocellular Carcinoma	PFOA	50–80 µg/mL for 48 h; MTT: 5, 10, 50, 200, 2000 µg/mL for 24/48/72 h	Cytotoxic & pro-apoptotic: time- and dose-dependent ↓viability; apoptotic morphology; Apoptosis ↑ with dose; Bax ↑, Bcl-2 ↓ (protein & mRNA) → reduced Bcl-2/Bax ratio consistent with apoptosis induction in SMMC-7721 cells.	[178]

Hep3B and SK-Hep1	Liver-Hepatocellular Carcinoma and Liver – sinusoidal endothelial-like	PFHxS	0–500 μM for 24/48 h; Colony formation: 0–500 μM for 12–14 days	Cytotoxic ≥400 μM in both lines. Clonogenicity: colonies ↑ ≤300 μM; ↓ at 500 μM. Cell-cycle signaling: Cyclin E, Cyclin D1, CDK2, CDK4, and PCNA ↑ (10–100 μM, 48 h). Interpretation: PFHxS can enhance proliferation of human liver tumours (HCC) and liver sinusoidal endothelial-like cells at 10–200 μM, with high-dose toxicity.	[179]
HEK293, HPA-s human preadipocytes (primary, subcutaneous fat), and Recombinant PPARγ-ligand binding domains (LBDs) (human & mouse)	Embryonic Kidney, Adipose, and Biochemical target (no tissue)	HFPO-DA, HFPO-TA, PFOA	HFPO-TA 12 μM, HFPO-DA 50 μM, PFOA 25 μM for 24h (HEK-293); Max test concentrations 25–50 μM	Robust lipid accumulation & adipogenic gene up-regulation; potency HFPO-TA > PFOA > HFPO-DA in human HPA-s; transactivation: all three agonize PPARγ (human > mouse). Overall: HFPO-TA is a more potent human PPARγ binder/agonist and adipogenic inducer than PFOA; HFPO-DA is weaker.	[180]
HEK 293T and hTERT RPE-1	Embryonic Kidney and Eye (retina pigment epithelium)	PFOA, PFNA, PFDoA, PFUnA, PFHxS, PFOS, PFDS, 4:2 FTS, 6:2 FTS, 8:2 FTS	1–100 μM for 24 h or mixtures: 100 μM total (10 μM each for 24 h) and 10 μM total (1 μM each for 24 h)	Carboxylates largely non-toxic ≤100 μM; PFNA ~30% ↓ viability; PFOS/PFOA/PFDoA slightly ↑ viability; sulfonate & fluorotelomer toxicity ↑ with chain length; 8:2 FTS > PFDS; in mixtures, short-chain species (4:2 FTS, PFHxS, PFOA, PFNA) often < LOD; Subcellular distribution: long-chain PFAS mostly insoluble (membrane/nuclear) fraction, while 6:2 FTS mainly soluble; PFHxS detectable only soluble. Transporter mechanism: CD36 overexpression increases cellular PFAS levels.	[181]

Huh-7 and HEK293	Hepatocellular Carcinoma and Embryonic Kidney	6:2 Cl-PFESA	3–10 μM for 72 h and for 24h (luciferase reporter assys)	Dose-dependent lipid accumulation and increased triglycerides; Upregulation of ACOX1 expression and peroxisome fatty acid β-oxidation; Suppression of hsa-miR-532-3p	[182]
hNIS-HEK293T-EPA	Kidney (engineered with human sodium/iodide symporter, NIS)	149 PFAS (including PFOS, PFHxS, PFNA, PFDA, PFUnDA, PFOS precursors, novel sulfonic PFAS, etc.)	up to 100 μM for 2 h	High-throughput radioactive iodide uptake (RAIU) assay identified 38 PFAS as NIS inhibitors; PFHxS and PFOS among the most potent; several novel PFAS (e.g., PFHpS, PFESA analogs) inhibited NIS with minimal cytotoxicity; ranked potency scores generated for thyroid disruption prioritization	[183]
HK-2	Kidney (proximal tubular cells)	PFOS	0–250 μM (12 h) for range-finding; 200 μM (12 h) used for mechanistic tests	PFOS can lead to the onset of ferroptosis in HK-2 cells and reduce HK-2 cell viability in a dose-dependent manner, ↑ ROS, produce toxic effects on cells (ferroptosis and apoptosis in HK-2 cells). Damage to the kidney.	[184]
RTCs	Renal Tubular Epithelium	PFOS	100 μM for 24h	Increased cytosolic ROS and PFOS- mediated apoptosis oxidative stress and renal toxicity.	[185]
Caki-1 (2D or 3D culture)	Kidney (Renal carcinoma, organoid model)	PFBA, PFPeA, PFHpA, PFOA, PFDA	20 μM for 16–72 h or 10 μM for 21 days	PFHpA, PFOA, PFDA showed stronger cytotoxicity; 20 μM PFACs stimulated cell migration; EMT activation evidenced by reduced E-cadherin, increased vimentin/N-cadherin, disrupted actin cytoskeleton, altered Na^+/K^+-ATPase; ↓ E-cadherin	[186]
HTR-8/SVneo and HEK 293T	Immortalized Trophoblast cells and Embryonic Kidney	PFOS	0–10 mM PFOS for 24 or 48 h (growth); 48 h (apoptosis)	PFOS inhibits trophoblast cell growth (dose-dependent) without significant apoptosis or cell-cycle change; H19 upregulated, miR-19a/b downregulated (cells & placentas); H19 promoter hypomethylation in PFOS-exposed placentas.	[187]

HTR-8/Svneo	Placenta (trophoblast, immortalized cells)	PFOS	1 nM–100 μM, 24–48 h	Higher doses ↓ cell viability; PFOS ↑ ROS generation, upregulated miR-29b, downregulated DNMT1/3A/3B and SIRT1/3, ↓ global DNA methylation, and induced protein hyperacetylation.	188
		PFOS, PFOA, GenX	Migration/invasion: 1000 ng/mL, 24 h pretreatl; Gene/secreted factors: 10–10 000 ng/mL for 24 h	Migration ↓ to 72.1% (PFOS), 80.7% (PFOA), 68.8% (GenX) of control at 1000 ng/mL; invasion significantly ↓ only with GenX (to 63.7%); PFOS/PFOA invasion ↓ but NS. Broad inflammatory transcript ↓; PFOS most potent; GenX minimal.	189
		PFOA	0, 100, and 200 μM for 72h	PFOA affects the balance between proliferation and apoptosis of trophoblasts; PFOA disrupts the response of trophoblasts to environmental stress; ROS and UPR signalings mediate the disruption effect of PFOA; Both the ROS and UPR signalings are triggered by endoplasmic reticulum stress.	190
JEG3	Placenta Choriocarcinoma (trophoblast)	PFOA, PFOS, HFPO-DA (detected at moderate levels: PFNA, PFUdA, PFTrDA, 6:2 FTS)	Cytotox screen: 0.001–100 μg/mL for 24 h. Non-cytotoxic doses: 0.01–10 μg/mL for 24 h	PFOS cytotoxic only in serum-free media (SFM) ≥50 μg/mL; PFOA/HFPO-DA non-cytotoxic across range. Cellular accumulation: PFOS > PFOA > HFPO-DA; PFOS & PFOA accumulate far more in SFM than SSM (dose-dependent), HFPO-DA similar in both.	191
JEG3	Placenta Choriocarcinoma (trophoblast)	PFOA, PFOS, GenX	50– 500 μM the duration of exposure depending on the endpoint evaluated	The placenta may be a direct target of PFAS exposure, with trophoblast cell gene expression and function being disrupted at PFAS levels significantly lower than the calculated cytotoxicity threshold (EC_{50}).	192

BeWo cells	Immortalized Choriocarcinoma cells	PFOS, PFBS	10 μM of PFOS and 100 μM of PFBS for 24h	PFOS but not PFBS decreased the gene expression of PlGF and increased the gene expression of FLT1; PFOS or PFBS significantly ↓ CGB7 ↓ hCG in syncytiotrophoblast; PFOS (but not PFBS) ↑ gene expression of CRH; Reproductive toxicity of PFOS and PFBS.	193
L-02 cells	Embryonic Liver	PFOS	50–250 μM for 12–48 h	↓ Viability (≥150 μM), ↑ ROS, ↓ mitochondrial membrane potential (MMP), ↑ autophagic vacuoles, ↑ apoptosis; Oxidative stress → mitochondrial dysfunction → autophagy contributing to apoptosis	194
hiPSC BiONi010-C; hiPSC IMR90-1	Pluripotent, no fixed tissue); differentiated into cardiomyocytes (heart)	PFOA, PFOS, GenX	Viability: 25–200 μM for 3 days; Differentiation: PFOS 3.13–50 μM; PFOA 6.25–100 μM; GenX 6.25–100 μM for 6 days	PFOS and PFOA significantly reduced embryoid body (EB) beat score and EB size, disrupted cardiomyocyte differentiation, altered cardiac gene expression (↑ISL1, ↓MYH7); GenX: no significant effects in BiONi010-C; mild concentration-dependent reduction in contractility in IMR90-1.	195
hiPSCs and hPP	Pluripotent stem cells → pancreatic progenitors → endocrine lineage	PFOS, PFOA, PFHxA, PFHxS, PFBS, PFBA	hPSCs: 50 nM PFASs, continuous exposure during 14-day pancreatic differentiation and hPPs: 50 nM PFASs, exposure for 7 days during endocrine differentiation	PFOS and PFOA disrupted pancreatic and endocrine differentiation; ↓ PDX1 expression in pancreatic progenitors; ↓ NGN3 expression in endocrine induction stage; hyperactivated NOTCH signaling; Short-chain PFASs (PFHxA, PFHxS, PFBS, PFBA) did not significantly disrupt hPP generation or SOX9 expression; PFOS/PFOA impair endocrine lineage commitment and pancreatic development.	196

hiPSCs differentiated toward dopaminergic neurons (DP1–DP3; NSC → neural progenitors)	Brain — dopaminergic neurons (iPSC-derived)	PFOA	1, 10, 100 ng/mL PFOA for 24 h at DP1-DP3 or functional assays: 10 ng/mL PFOA for 24 h at DP1-DP3	Highest accumulation at DP1; progressively lower at DP2/DP3 (stage-dependent); ↑ membrane fluidity) at DP1 and DP2; no significant change at DP3; Cytotoxicity: No significant effect; Overall, short-term PFOA (10 ng/mL, 24 h) is associated with impaired dopaminergic differentiation/ function without overt cytotoxicity.	197
hMSCs	Mesenchymal stem cells (human; multipotent stromal, bone marrow)	PFOA, PFOS	environmental and human-relevant concentrations (ng/mL to low µM range)	Acute cytotoxicity observed for both PFOS and PFOA; ↓ CD90 surface marker expression in undifferentiated hMSCs; ↑ Adipogenesis (promotion via PPARγ activation, ↑ lipid accumulation); PFOA partly impaired osteogenesis; PFOS/PFOA affect stem cell self-renewal.	198
hMSCs	Mesenchymal (human; multipotent stromal, adipose/ bone marrow)	PFOS	0.1 µM and higher	PFOS at 0.1 µM → induce cellular ROS, enhance the PPARγ and ap2 mRNA expression => promoting adipogenic differentiation of hMSCs even at low, environmentally relevant concentrations	199
hMSCs → adipocytes or hMSCs → osteoblasts	Mesenchymal (undifferentiated/ adipocyte or osteoblast lineage	PFBS, PFHxS, PFBA, PFHxA	50–300 µM acute; 1 nM–10 µM chronic for 6–24 h (acute) or 21-28 days (adipogenesis)	PFHxS cytotoxic at high µM; others not. All ↑ROS (PFHxS strongest); PFBS/PFHxS ↑Ca^{2+}. Low dose: ↓MSC markers, ↑adipogenesis; no osteogenesis change.	200
hESCs → cardiomyocytes	Embryonic (pluripotent; cardiac/epicardial lineage)	PFOS, F-53B (6:2/8:2 Cl-PFESA mix)	0.1–60 µM until differentiation; cytotox. at ≥120 µM (F-53B) or ≥180 µM (PFOS) over 48 h	≤60 µM: ↓cardiomyocyte differentiation/markers, ↓beating; ↑epicardial fate with ↑fibroblast/ ECM genes; F–53B > PFOS; WNT signaling ↑; WNT inhibitor partially rescues.	201

hESC (H9); hESC-derived hepatocyte-like cells	Embryonic stem cells; Liver	HFPO-TA	up to ~600 μM for 24-72h; hepatocyte-like cells: 0.01-50 μM for 14 days; 10 μM used for RNA-seq/validation	hESCs: dose-dependent cytotoxicity; mitochondrial depolarization; oxidative stress; apoptosis/necrosis. Hepatocyte-like cells: impaired glycogen storage ; hepatic genes dysregulated; PPARα ↑ at 10 μM, ↓ at 50 μM; 417 DEGs with enrichment in PPAR signaling, lipid/fatty-acid metabolism and solute carriers; hub liver proteins downregulated → hepatotoxicity/ metabolic transport disruption.	202
MCF-10A	Breast epithelium	PFOA, PFOS	10 μM, for 72h 100 μM for 72h	Promotion of cell migration & invasion and increased global DNA methylation and epigenetic mechanisms implicated in tumorigenesis.	203
MCF-10A	Breast epithelium	PFHxS, PFHxA, PFBS, HFBA, PFO2OA	500 pM to 500 μM for 72h	PFHxS induced epithelial cell proliferation, and promoted the migration and invasion potential, by altering cell-cycle regulators, adhesion proteins and histone modifications; PFHxA, HFBA, PFBS and PFO2OA all seem to be safer alternatives.	204
MCF-10A	Breast epithelium	PFOA, PFOS	100 pM–100 μM for 72h	Cell proliferation mediated by PXR activation, an increase in cyclin proteins levels, decrease in p21 and p53 levels. Alterations in histone modifications. Exposure to higher concentrations of the mixture caused cell death.	205
MCF-7	Breast Tumor	HFPO-DA, HFPOA-TA, HFPO-TeA, PFOA	PFOA 0–800 μM; HFPO-DA 0–800 μM; HFPO-TA 0–25 μM; HFPO-TeA 0–25 μM (MVLN assay 12h & 24h)	PFOA acted as ER agonist (↑ luciferase; competitively reduced E2-induced signal). HFPO-TA & HFPO-TeA acted as ER antagonists (↓ luciferase; inhibited E2 response); much stronger ER binding than PFOA; Docking/ MD: PFOA binds (agonist like) with ERs (similar as 17β-estradiol) while HFPOs disrupted helix-12 (antagonist-like).	206

MCF-7, HepG2	Breast Tumor and Hepatocellular Carcinoma	PFOA	100 and 200 μM 200 and 400 μM	DNA methylation, histone modification, DNMT activity, and heterochromatin reorganization — relevant to epigenetic toxicity and cancer risk.	207
MDA-MB-231	ER-Negative Breast Tumor	PFOA	10–200 μM PFOA	↑ Cell migration and invasion of MDA-MB-231 cells upon PFOA exposure; Effect linked to upregulation of fatty acid 2-hydroxylase (FA2H); Silencing FA2H attenuated PFOA-stimulated migration, supporting mechanistic involvement; ER-independent effects.	208
MCF-7 and DU145	Breast Tumor and Prostate Tumor	PFOA	dilutions from 10 μM to 10 pM	Increase proliferation of cancer cells in both tumor cell lines; PFOA possible carcinogen.	209
T47D-Kbluc and hPPARα/PPARγ reporter assay	Breast Tumor and Engineered Human Receptor System	PFHxA, PFOA, PFNA, PFDA, PFBS, PFHxS, PFOS, HFPO-DA, HFPO-DA-AS, NBP2, PFMOAA, PFOSA, EtFOSA, 4:2 FTOH, 6:2 FTOH, 8:2 FTOH	1–1000 μM for 24 h	Estrogen receptor (ERα) activation by 8:2 FTOH, 6:2 FTOH, PFHxS, PFOSA; weaker activity by PFOA, PFNA, PFOS; overall weak to moderate estrogenicity; Multiple PFAS acted as PPARα and PPARγ agonists; HFPO-DA and HFPO-DA-AS were the most potent activators.	210
KGN	Ovarian Granulosa cells	PFOA	0.03–300 μM (0-48 h)	Reproductive toxicity: PFOA decreased viability and increased apoptosis; downregulated Cx37 (mRNA) and Cx43 (mRNA/protein) and inhibited GJIC; RA mitigated these effects (↑GJIC/viability, ↓apoptosis), while CBX worsened them.	211
HGrC1	Ovarian Granulosa (immortalized) cells	PFOA	1, 10, 100 μM (MTT); 1–10 μM (proliferation/ migration) for 24, 48, 72, 96 h	10 μM ↑ MTT at 24–48 h; 1–10 μM ↑ metabolic activity at 72–96 h; 100 μM ↓ viability; 1–10 μM ↑ proliferation/migration with no change in apoptosis; ↑ CCND1/CCNA2/CCNB1 mRNA and YAP1/CTGF proteins.	212

OVCAR-3and Caov-3	Epithelial Ovarian Adenocarcinoma	PFOA, PFHpA, PFPeA and mixtures: PFOA + PFHpA, PFOA + PFPA, PFOA + PFHpA + PFPeA	PFAS (individual): 25 nM–2 μM, total 48 h and PFAS mixtures: totals 2.0–2.25 μM for 48 h	At sub-cytotoxic doses, PFAS mixtures increased survival fraction; individual PFHpA/PFPeA (not PFOA) increased survival after carboplatin → induced platinum resistance; OVCAR-3 generally more sensitive than Caov-3.	213
IOSE-80	Ovary— epithelial (non-tumorigenic)	PFDA	0.01–100 μM PFDA for 12 h (γ-H2AX) and 0–16 μM for 12/24/48 h (viability test)	↑ DNA double-strand breaks in a dose-dependent manner;↑ cGAS expression and nuclear accumulation; PFDA effects persisted in controls after withdrawal but resolved with cGAS KD/Importazole; nuclear cGAS impairs homologous recombination, interacts with PARP; Cytotoxicity only at higher doses/longer times.	214
HUVECs	Vascular endothelium	PFOS (± HSA; ± FBS), PFOA (comparative)	PFOS 0–100 μM, 24 h; HSA prebound 50 μM; FBS 0/2/10%	Albumin/FBS binding sequesters PFOS → ↓cellular uptake and cytosolic/mitochondrial/nuclear accumulation; ↓ROS, ↓caspase-3, ↓LDH leakage; ↑viability. Toxicity stronger in serum-free media; largely abrogated with 10% FBS. Similar mitigation observed for PFOA.	215
Ishikawa endometrial epithelial cells	Endometrium (epithelial)	PFOA with or without progesterone (P4)	1 μM PFOA co-incubated 48 h with P4 10^{-8} M after E2 10^{-7} M priming (PFOA alone also tested)	Antagonizes progesterone signaling: ↓P4-induced genes; effect blunted by higher P4 (consistent with competitive interaction); no change in proliferation. Epidemiology: higher PFOA exposure associated with later menarche (~+164 days) and more irregular cycles.	216

PrECs grown as prostaspheres (progenitor 3D spheroids)	Prostate	PFOS, PFOA	10 nM (3–4 weeks)	Chronic low-dose exposure increased prostasphere number and size, indicating enhanced stem/progenitor self-renewal and proliferation; PPARα, RXRα/β expression and aberrant luminal progenitor differentiation; Transcriptomic enrichment of cell cycle, NF-κB/TNFα, and KRAS signaling pathways; Glycolytic reprogramming.	217
Human Semen Samples	Reproductive — mature sperm	PFOA	0.1, 1, 10 ng/mL PFOA for 1–2 h	↓ Progressive motility from 0.1 ng/mL at 1 h; PFOA accumulates in sperm membranes; ↑ Membrane fluidity and ↓ plasma-membrane potential; ΔΨ Mitochondrial respiration efficiency ↓; β-cyclodextrin removes membrane PFOA and restores ΔΨp & motility.	218
Human ejaculated spermatozoa	Reproductive — spermatozoa	PFOA with or without progesterone (P4)	PFOA 0.25, 2.5, 25 µg/mL (~0.6, 6, 60 µM) for 30 min–4 h	CatSper/$[Ca^{2+}]i$: Rapid extracellular Ca^{2+} influx via CatSper; CatSper currents ↑; Progesterone signaling: PFOA pre-exposure blunted P4-elicited Ca^{2+} entry, reduced P4-induced sperm penetration and acrosome reaction. Function/oxidative stress: High-dose PFOA (25 µg/mL) reduced penetration and ↑ ROS at 4 h.	219
NHT (normal human thyroid)	Endocrine — Thyroid Gland	C6O4, PFOA, PFOS	Viability: 0–100 ng/mL for 24, 48, 72, 144 h. Apoptosis: 100 ng/mL, 144 h. Proliferation : 0–100 ng/mL, 6 days.	C6O4: no change in viability, apoptosis/necrosis, proliferation, or ROS at any dose in NHT cells at any time points. **Safe alternative!** PFOA: no significant change in viability or proliferation across doses/times; PFOS: ↓ viability at all timepoints; ↓ proliferation (6 d); ↑ apoptosis/necrosis at 100 ng/mL/144 h.	220

A549 (cancer cell line)	Lung — alveolar epithelial (adenocarcinoma)	PFOA, PFOS, GenX	10–1000 μM for 24 & 48 h	Viability: GenX ↑ proliferation ≥50 μM; PFOA/PFOS biphasic (↑ at 100–200 μM, ↓ ≥200–600 μM; PFOS > PFOA toxicity); Apoptosis: BAX ↑, BCL2/BCL2L1 ↓ (PFOS > PFOA > GenX); CASP3/9 ↑ → intrinsic apoptosis; Uptake: PFOS > PFOA; GenX ND.	[221]
HBEC3-KT	Bronchial epithelium	PFBS, PFHxS, PFOS, PFOA, 8:2 FTOH	0.13–10 μM for 48 h (PFOS up to 30 μM)	No cytotoxicity ≤10 μM; PFOS: ↓ CXCL8 & CXCL10 (Poly I:C-primed), ↑ IL-1α and IL-1β; PFOA: ↑ IL-1β; PFBS, PFHxS, 8:2 FTOH: no effect on cytokines.	[222]
16HBE14o	Bronchial epithelium	PFOS	5–25 μM for 24–72 h	↓ transepithelial electrical resistance and ↑ paracellular permeability; ↓ IL-8 release; downregulated and disrupted organization of tight junction proteins ZO-1 and occludin; ↑ PKD phosphorylation. PKD inhibition attenuated PFOS-induced barrier dysfunction and restored occluding expression/localization.	[223]
BEAS-2B	Bronchial Epithelium	PFOA, PFOS, GenX	1–500 μM for 24h 1–1000 μM for 24h	PFOA and PFOS alone or in a mixture activate the NLRP3 inflammasome; Combined with in vivo study in mice: inflammation- and immune-related genes differentially expressed; Asthma/airway hyper-responsiveness.	[224]
Human Primary Hepatocytes (hPP)	Liver	GenX	0.1, 10, 100 μM for 48–96 h	Dose-dependent induction of fibro-inflammatory genes; activation of STAT3, TNF/IL-6→STAT1/NF-κB; TGFB1/SMAD4 fibrosis signaling ↑; Proliferation: PCNA, Ki67, CDK4, Cyclins ↑ (notably at 100 μM); Metabolism: Lipid metabolism pathways altered; PPARα activation.	[225]

Human Primary Hepatocytes (hPP)	Liver	PFHxS, PFOS, PFOA, PFBA, PFPeA, PFHxA, PFHpA, PFBS, PFNA, PFDA, PFUnDA, PFDoA, PFTrDA, PFTeDA, 6:2 FTS, FOSA, MeFOSA, EtFOSA	Legacy PFAS (PFOA, PFHxS, PFOS): up to 25 μM; Short-chain PFAAs (PFBA, PFPeA, PFHxA, PFHpA, PFBS), alternative PFAS (HFPO-DA, 6:2 FTS) & precursors (FOSA, MeFOSA, EtFOSA): tested at 0.25, 2.5, 25 μM	Legacy PFAS induced expression of genes related to xenobiotic metabolism, such as CYP2B6 and SULT2A1(strongest effects at 25 μM); Shorter chain PFAS (C4-C5), 6:2 FTS, and MeFOSA, induced significant liver lipid accumulation, and gene activation at lower concentrations than legacy PFAS.	[226]
hBMSCs → osteoblasts; hBMSCs → adipocytes	Mesenchymal (Bone marrow)	PFOS	0.2–200 nM during differentiation (Day 0–14)	Osteogenesis↓: mineralization (Alizarin Red) ↓ dose-dependently ≤100 nM; OCN protein ↓; OPN/ON mRNA ↓; β-catenin ↓; RANKL↑/OPG↓. Adipogenesis↑: lipid accumulation↑; PPARγ, leptin mRNA ↑. Pathway endpoints: WNT/β-catenin suppressed (LiCl rescues; DKK1 antagonizes); TGF-β signaling enriched in transcriptomics.	[227]
hBMSCs → osteogenic differentiation	Mesenchymal (Bone marrow)	Cl-PFESA, PFOS, PFHxS, PFOA	100 nM pre-exposure for 7 days then PFAS removed	261 genes were affected by all 4 compounds, enriching osteoblast differentiation: common DEGs across PFAS; osteoblast differentiation and lipid/cholesterol metabolism pathways perturbed; calcium-signaling gene sets ↑ (PFOS/PFHxS/PFOA); TGFβ/TGFβ-response enriched; Cl-PFESA posed stronger effects on calcium transients than PFOS and PFHxS; Cl-PFESA repressed RUNX2 in osteogenic differentiation following exposure.	[228]

Primary Human Lymphocytes	Blood	PFOS	50 μM for 72 h	Altered production of interleukins (↑IL-1, ↑IL-4, ↑IL-6, ↑IL-8); DEGs affecting immune system processes identified: cytokine and GPCR signaling, PPAR and PI3K-Akt pathways; Dysregulation of glycerophospholipid, sphingolipid, and glycerolipid metabolism linked to immune dysfunction.	[229]
Primary human BMCs and primary human basophils	Blood	PFBA, PFHxA, PFHxS, PFOA, PFOS, PFNA (single or mixtures)	0.02–2 ng/mL for 20 h or 2 ng/mL for 1h	PFAS mixtures reduced T cell activation; short-chain PFAS ↓CD71, ↓TNF-α in $CD8^+$/NKT cells; long-chain PFAS ↓CD71 in $CD8^+$ and MAIT cells; mixtures exerted strongest suppression across all T cell subsets; MAIT cells showed ↓IFN-γ, ↓TNF-α, ↓CD69 co-expression; Broad downregulation of immune-related genes.	[230]
hBMSCs	Bone marrow	HFPO-DA, HFPO-TA	0.1–10 μM, 7–21 d (undifferentiated and induced differentiation)	Transcriptomics revealed >1000 DEGs per chemical at 0.1 μM; enriched pathways included cell cycle regulation, pluripotency, and multiple cancer-related pathways. HFPO-TA promoted abnormal proliferation at 1 μM, while higher doses downregulated CDK and CASP expression. Both HFPOs repressed MSC stemness markers and impaired osteogenic differentiation (reduced calcium deposition); adipogenic effects minimal → multipotency impairment and carcinogenesis-related signaling disruption.	[231]
Namalwa cells (lymphoma B cell line)	Blood	PFOS, PFOA, PFHxS, PFNA	up to 100 μM (PFNA ≤33 μM) for 6–48 h	All PFAS ↓ RAG1/2 expression in a time- and concentration-dependent manner; PFOA RNA-seq altered 574 genes, affecting B cell development and primary immunodeficiency pathways; BMD modeling showed PFNA most potent, PFHxS least potent.	[232]

HCT116	Colorectal	PFOS	0.001–10 μM for 48h	Increased proliferation and migration of colorectal cancer cells (CRC) via PI3K/Akt-NF-κB activation and EMT; Upregulation of VEGF, IL-8.	[233]
DLD-1	Colorectal Carcinoma (colon)	PFOA	10–600 μM for	↑ contributions of glutamine and fatty acids to tricarboxylic acid (TCA) under PFOA stress; Production of palmitate from glucose and glutamine was both inhibited by PFOA; Reductive metabolism of glutamine in intestinal cells was activated by PFOA.	[234]
SW48 KRAS WT and SW48 KRAS G12A	Colorectal Carcinoma (colon)	PFOS, PFOA	0–100 μM 24–48 h (migration); 1–7 days (viability)	10 μM PFOS/PFOA ↑ migration in both lines (WT & G12A); short exposures didn't change cell counts, but prolonged high doses ↓ proliferation. Metabolomics at 10 μM showed ↓ free amino acids (e.g., alanine, phenylalanine, proline) and ↓ fatty acids/monoglycerides, ↑ fatty alcohols (1-dodecanol/1-octanol), and ↓ uridine.	[235]
Pancreatic 1.1B4 β-cells	Pancreas	PFOS	100 μM for 36 h	PFOS induced mitochondrial ROS, loss of mitochondrial membrane potential, caspase-3 activation, and β-cell apoptosis; PFOS impaired glucose-stimulated insulin secretion; Protective interventions: RAC1-NOX2 inhibition, PTX (cAMP/PKA activator), and NAC (antioxidant) attenuated ROS generation, preserved PKA-CREB signaling, and improved β-cell survival and insulin secretion.	[236]

SH-SY5Y	Brain¾ Neuroblastoma	PFOS	0, 25, 50, 100, 200 μM for 48 h; 200 μM for 0–48 h	Dose- and time-dependent ↑ JNK phosphorylation; apoptosis from 50 μM (↑ Bax, ↑ cleaved caspase-3, ↓ Bcl-2); ↑ ROS in dose-dependent manner; activated p-JNK accumulated in mitochondria and nucleus; apoptosis and ROS mitigated by NAC (ROS inhibitor) and SP600125 (JNK inhibitor).	237
		PFOA	10, 100, 250, 500 μM for 4/24/48 h (iability/cytotoxicity/apoptosis) and 0.01, 0.1, 1, 10, 100, 250, 400 μM for 24/48/72 h (metabolically active cells)	No cytotoxicity at ≤100 μM (≤48 h); cytotoxicity & caspase-3/7 apoptosis at ≥250 μM (24–48 h); ATP ↓ at 400 μM (24–48 h) and at ≥100 μM; ATP synthase activity inhibited at 250 μM; Mitochondrial membrane potential ↓ ~40–60% at 100–250 μM. ROS: modest early increase at 100–250 μM; Overall: mitochondrial dysfunction emerges ≥100 μM, preceding overt cytotoxicity at higher doses.	238
		PFOA	4 days at 0.4 or 4 μg/L (~1–10 nM), then PFOA removed → differentiation: 14 days PFOA-free	No overt cytotoxicity (MTT) but faster proliferation during exposure; nuclear size ↑ and roundness changes; Neuronal morphology: neurite branches↓; process number ↓ at 4 μg/L; neurite length ~unchanged. Mitochondria: volume ↓ post-diff; more fragmented networks; ↓ DA markers; low-ppb developmental-like PFOA exposure imprints persistent epigenetic hypomethylation & mitochondrial deficits.	239
Astroglia SVG p12	Fetal Brain	PFOA	10-200 μM for 24, 48 or 72h	Alterations in the DA-like neurons, differentiated from SH-SY5Y cells; altered network connectivity and mitochondrial volume; 10 μM PFOA: elevated glutamate levels, while 80 μM PFOA: ↑ expression of S100B, tachykinin, and CYP1A1 → neurotoxicity.	240

A549, DLD-1 and L-02	Lung Epithelial (adenocarcinoma), Colon Epithelial and Liver	PFOA	0, 100, 300 μM for 24 h & 48 h for cytokines and for 48 h for metabolomics	A549: Strong, dose-dependent metabolic disruption (67 DMs at 300 μM), especially amino-acid pathways; Linoleic/arachidonic-pathway metabolites ↓. DLD-1: Purine metabolism suppressed (adenine & GDP ↓); enrichment in ammonia recycling/methyl-histidine and aminoacyl-tRNA biosynthesis.	[241]
CaCo-2, HepaRG, HEK293, HMC-3, MRC-5, and RMS-13	Colon Epithelial, Liver, Embryonic Kidney, Brain microglia, Lung fibroblast, and Skeletal muscle (rhabdomyosarcoma)	HFPO-DA, PFHxA, 6:2 FTOH, PFOA, PFBS, PFHxS, PFOS	10 pM–100 μM for 48h	HepaRG, HMC-3, and RMS-13 most sensitive—clear viability loss to PFOS/PFOA and to PFHxS and 6:2 FTOH (HFPO-DA notably active in HepaRG); CaCo-2 and HEK293-hTLR2 were largely resistant to short-chain PFAS and to PFOA, with PFOS the only consistent reducer of viability (at higher doses); Overall potency: PFOS ≳ PFOA ≫ PFHxA/PFBS; PFHxS and 6:2 FTOH affected mainly the sensitive lines; no consistent dependence on chain length or headgroup across cell types.	[242]
HEK293-hTLR2, HepaRG, HMC-3, and RMS-13	Embryonic Kidney, Liver, Microglia (brain), and Skeletal muscle	PFBS, PFHxA, HFPO-DA, 6:2 FTOH, PFHxS	24 h at 1 nM & 1 μM in FBS-free media; ROS imaged only in HepaRG	ROS (HepaRG): none at 1 nM; ↑ at 1 μM PFBS and PFHxA; CAT: ↑ mainly with HFPO-DA in HEK293, HepaRG, RMS-13; HEK293 also ↑ with PFBS; SOD: HEK293 ↑ with PFBS & 6:2 FTOH; HepaRG ↑ with PFHxA; HMC-3 ↑ with 6:2 FTOH and PFHxA; Effects observed below cytotoxic EC50s; compound- and tissue-specific redox responses.	[243]
Mouse ES cell D3 differentiated into cardiomyocytes	Mouse Embryonic stem cells	PFOS	40 μM during cardiomyocyte differentiation (days 3–10)	Autophagosome accumulation, impaired autophagic flux, disrupted mitophagy and mitochondrial biogenesis, and impaired autophagy–lysosome pathway → ↓ATP, mitochondrial membrane potential, and cardiogenesis.	[244]

(1) Xenopus laevis oocytes expressing human α1β2γ2L $GABA_A$; (2) Rat primary cortical neurons; (3) hiPSC Glutaneurons + Astrocytes; (4) hiPSC co-culture: Glutaneurons, GABAergic, Astrocytes	(1) Xenopus ovary—oocytes; (2) Rat brain; (3) and (4) Human brain (iPSC-derived) — glutamatergic+ GABAergic neurons with astrocytes	PFOA, PFOS	Oocytes: 0.01–100 μM with GABA; MEA networks: 0.1–100 μM acute	LOECs for PFAS on GABA(A) receptor and neuronal activity reported non-competitive GABA_A antagonists and clear neurotoxic risk; Networks: Rat cortex—PFOS ↑MSR/MBR at 100 μM, otherwise minor. Human iPSC (Glut+ Astro): PFOS LOEC 0.1 μM; PFOA LOEC 1 μM → ↓MSR, ↓MNBR, ↑burst duration; PFOS > PFOA potency; human models more sensitive.	245
Mouse liver organoids (3D)	Mouse liver epithelium—hepatic progenitors + mature hepatocyte-like cells within organoids	PFOS, PFOA, PFPA (PFPeA), HFBA (PFBA)	Viability (EC50): 0.1 μM–2 mM for 96 h; Histology: 100, 500, 1000 μM for 6/24/48 h; Caspase-3/7: 100–1000 μM at 1/3 h	Cytotoxicity: PFOS EC50 ≈ 670 μM; PFOA EC50 ≈ 895 μM; PFPA & HFBA NC (no EC50 ≤1000 μM). Morphology: PFOS/PFOA cause early loss of epithelial cells, nuclear pyknosis, architectural collapse (visible by 6 h at 1000 μM; milder at 100–500 μM). HFBA: mild-moderate disorganization; PFPA: minimal; Apoptosis: Caspase-3/7 ↑ only for PFOS/PFOA at 1000 μM; Overall: long-chain > short-chain for cytotoxicity; short-chains still trigger early hepatotoxic stress.	246
3D primary mouse liver spheroids (hanging drop) co-cultured 28 d	Mouse liver epithelium	PFOA, HFPO-TA	25, 50, 100 μM for 28 d; RNA-seq (100 μM); LPS 10 μg/mL for 24 h on day 27 for inflammation tests	HFPO-TA in spheroids > 2× PFOA. Cytotoxicity: ATP ↓ at 100 μM HFPO-TA; LDH ↑ at mid/high doses; albumin ↓ at all HFPO-TA doses and mid/high PFOA; urea ↓ at high dose; Total bile acids ↑ in spheroids; 1603 DEGs (HFPO-TA) vs 772 (PFOA); acute inflammatory response; disease links to fatty liver, cholestasis, carcinoma. With LPS: TNFα & IL-1β ↑ dose-dependently; HFPO-TA generally stronger than PFOA.	102

3D co-culture angiogenesis model: Human primary HUVECs + normal human colonic fibroblasts (NCFs); Monocultures: HUVECs, NCFs, HCT116, HEK-293	Endothelium + Fibroblasts; Embryonic kidney, Colon epithelium/ carcinoma	PFOS	0–120 µg/mL: up to 120 h (viability/metabolic, for 24h (apoptosis), for 6h (VEGFR2 bioassay), for 7 days (angiogenesis/ co-culture)	Sensitivity: HUVECs >> NCFs/HCT116; VEGFR2 signaling: PFOS dose-dependently suppresses VEGFR2 without cytotoxicity in HEK cells; Angiogenesis (3D): PFOS inhibits sprouting dose-dependently; Co-culture viability over 7 d unchanged up to 96 µg/mL, supporting anti-angiogenic action rather than general cytotoxicity. Overall: antiangiogenic effects via VEGFR2 inhibition, mechanistically related to developmental toxicity (AOP43) including reduced fetal growth.	[247]
liver-on-a-chip (HepaRG + HMEC-1 + THP-1)	Liver (hepatocytes, endothelial, macrophage co-culture)	PFBS, PFHxA, HFPO-DA, PFHxS, 6:2 FTOH	1 nM and 1 µM for 48 h	Viability largely unaffected (except ↓ at 1 µM 6:2 FTOH); Gene expression changes: ↓ABCA1 (PFBS, HFPO-DA, 6:2 FTOH), ↑ABCG2 (all PFAS except HFPO-DA), ↑CYP1A1, ↓CYP1A2 , ↑CYP2B6, ↓CYP2C19; Suggests PFAS-induced oxidative stress and AHR/CAR pathway activation	[248]
3D InSight Human Liver Microtissues	Liver	Multiple single PFAS and mixtures (PFBA, PFPeA, PFHxA, PFHpA, PFOA, PFNA, PFDA, PFUnA, PFBS, PFHxS, PFOS, PFOSA, 6:2 FtS, 8:2 FtS; mixtures 1–7)	0.02–100 µM for 24 h or 10 days	Longer-chain PFCAs (PFNA, PFDA, PFUnA) showed higher potency (lower BMCs) than short-chain PFAS; Mixture effects generally followed concentration addition predictions; Cytotoxicity occurred at high concentrations (≥20–100 µM) for some long-chain PFAS and complex mixtures.	[249]

Legend: hBMSCs= human bone marrow-derived mesenchymal stem cells; hESCs= human embryonic stem cells; hiPSCs= human induced pluripotent stem cells; hMSCs= human mesenchymal stem cells; HUVECs= human umbilical vein endothelial cells. 3D InSight Human Liver Microtissues: spheroids from 10 human liver donors, co-culture of hepatocytes + Kupffer cells.

Table S5. Comparative toxicological evidence for alternative PFAS relative to legacy PFAS (PFOS, PFOA). The table summarizes key toxicological endpoints, highlights similarities and differences in potency and bioaccumulation, and provides supporting references.

Alternative PFAS	Key Toxicological Effects	Comparison with legacy PFAS	References
PFBS	Developmental toxicity, Neurotoxicity, Liver injury, Reproductive toxicity, Metabolic disturbances	Less bioaccumulative but still exhibits metabolic alterations and developmental toxicity; Significant reproductive and liver toxicity as PFOS; Less neurotoxic than PFOS, but exhibits similar molecular mechanisms	42,94,154,155,157,193,200,250
HFPO- DA	Liver damage – Hepatotoxicity; Metabolic effects- Lipid metabolism alteration; Neurotoxicity, Developmental and Reproductive (e.g., trophoblast effects) toxicity, Cardiotoxicity	Similar hepatotoxicity, and developmental effects as PFOA	3,24,28,56,132,163,189,251
PFHxA	Developmental, Liver damage and immunological disruption	Less bioaccumulative but exhibits developmental toxicity; Similar gene expression changes (with PPAR isoforms)	72,96,252
6:2 Cl-PFESA	Thyroid disruption, Liver damage, Metabolic syndrome and blood lipids alterations, Hepatotoxicity, Developmental and cardiovascular effects, Endocrine disruption	Not a safe alternative; shows comparable toxicity to PFOS; greater disruption of thyroid homeostasis compared to PFOS (human study)	12,103,182,201,253
HFPO-TA	Liver toxicity, Developmental (general, heart) toxicity, Reproductive toxicity; Carcinogenicity	Short-chain carboxylate; stronger agonistic activity towards PPARγ * than PFOA, potential liver damage and developmental toxicity, further research needed	53,180,202
6:2 FTOH	Behavioral alterations, morphometric changes, gene expression disruption, Developmental toxicity	Neurodevelopmental and gene expression effects similar to PFOS and PFOA; Limited data on bioaccumulation	127,243,254

OBS	Hepatotoxicity, microbiota dysbiosis, Endocrine toxicity Developmental toxicity, Neurotoxicity	Limited data, potential developmental and neurotoxic effects as PFOS; Less impact on the liver than PFOS.	[122,123,136,255]
6:2 FTSA	Immunotoxicity	Immunotoxic and developmental effects, similar to PFOS	[109]
Nafion-BP2	Hepatotoxicity, Metabolic effects, Immunotoxicity Developmental toxicity	Bioaccumulative; Similar hepatotoxicity as PFOS. Less developmental toxicity than PFOS, but more potent than GenX.	[2,105]
PFECAs (e.g., PFO4DA, PFO5DoDA, PFMO2HpA)	Liver damage, Metabolic effects	Lower accumulation in liver compared to PFOA; Similar hepatotoxicity with PFOA, GenX, except PFMO2HpA → potential alternative	[57–60]

Note: PPARγ: peroxisome proliferator-activated receptor gamma; The order of agonistic activity toward PPARγ signaling pathway in HEK 293 cells: HFPO-TA > PFOA > HFPO-DA. From molecular dynamics simulation, HFPO-TA was found to form more hydrogen bonds than PFOA; Water solubility of PFOA >500 g/L and PFECAs >1000 g/L.

Table S6. Full inventory of included human epidemiological studies (n = 69) identified in the ScR. Health outcome/endpoint was extracted verbatim from study titles using a rule-based parsing approach. Table 7 in the main manuscript presents an illustrative subset of these studies. Two studies appear in 2024 journal issues but were published online in 2023 and retained per the eligibility rule.

First author	Title	Health outcome / endpoint	DOI	PFAS investigated
Donat-Vargas (2019)[256]	Perfluoroalkyl substances and risk of type II diabetes: A prospective nested case-control study.	Risk of type 2 diabetes	10.1016/j.envint.2018.12.026	PFOA, PFOS, PFNA, PFHxS, PFDA, PFUnDA
Girardi (2019)[257]	A mortality study on male subjects exposed to polyfluoroalkyl acids with high internal dose of perfluorooctanoic acid.	Mortality	10.1016/j.envres.2019.108743	PFOA
Jain (2019)[258]	Concentration of selected liver enzymes across the stages of glomerular function: the associations with PFOA and PFOS.	Concentration levels of liver enzymes	10.1016/j.heliyon.2019.e02168	PFOA, PFOS
Jain (2019)[259]	Selective Associations of Recent Low Concentrations of Perfluoroalkyl Substances With Liver Function Biomarkers: NHANES 2011 to 2014 Data on US Adults Aged ≥20 Years.	Liver Function Biomarkers levels	10.1097/JOM.0000000000001532	PFOA, PFOS, PFDA, PFHxS, PFNA
Nian (2019)[252]	Liver function biomarkers disorder is associated with exposure to perfluoroalkyl acids in adults: Isomers of C8 Health Project in China.	Liver function biomarkers levels	10.1016/j.envres.2019.02.013	PFOS, PFPeA, PFHxA, PFNA, PFDoDA, PFTrDA, PFTeDA
Wang (2019)[260]	Perfluoroalkyl substances exposure and risk of polycystic ovarian syndrome related infertility in	Risk of polycystic ovarian syndrome related infertility in chinese	10.1016/j.envpol.2019.01.039	PFOA, PFOS, PFDoA

	Chinese women	women		
Wang (2019)[261]	Renal function and isomers of perfluorooctanoate (PFOA) and perfluorooctanesulfonate (PFOS): Isomers of C8 Health Project in China	Renal function impairment	10.1016/j.chemosphere.2018.11.191	PFOA, PFOS
Wen (2019)[262]	Prenatal perfluorooctanoic acid exposure is associated with early onset atopic dermatitis in 5-year-old children.	Early onset atopic dermatitis	10.1016/j.chemosphere.2019.05.100	PFOA, PFOS
Ait Bamai (2020)[263]	Effect of prenatal exposure to per- and polyfluoroalkyl substances on childhood allergies and common infectious diseases in children up to age 7 years: The Hokkaido study on environment and children's health.	Potential of allergic reaction common infectious diseases susceptibility	10.1016/j.envint.2020.105979	PFHxA, PFHpA, PFOA, PFNA, PFDA, PFUnDA, PFDoDA, PFTrDA, PFTeDA, PFHxS, PFOS
Canova (2020)[264]	Associations between perfluoroalkyl substances and lipid profile in a highly exposed young adult population in the Veneto Region.	Alterations in lipid profiles of young adults	10.1016/j.envint.2020.106117	PFOA, PFOA, PFHxS
Cohn (2020)[265]	In utero exposure to poly- and perfluoroalkyl substances (PFASs) and subsequent breast cancer.	Subsequent breast cancer development risk	10.1016/j.reprotox.2019.06.012	EtFOSAA, PFOS
De Toni (2020)[266]	Increased Cardiovascular Risk Associated with Chemical Sensitivity to Perfluoro-Octanoic Acid: Role of Impaired Platelet Aggregation.	Increased Cardiovascular Risk	10.3390/ijms21020399	PFOA
Dreyer (2020)[267]	Perfluoroalkyl Substance Exposure Early in Pregnancy Was Negatively Associated with Late Pregnancy Cortisone Levels	Late Pregnancy Cortisone Levels association	10.1210/clinem/dgaa292	PFOS, PFOA, PFHxS, PFNA, PFDA

Kashino (2020)[268]	Prenatal exposure to 11 perfluoroalkyl substances and fetal growth: A large-scale, prospective birth cohort study.	Fetal growth development risks	10.1016/j.envint.2019.105355	PFHxS, PFHxA, PFHpA, PFOS, PFOA, PFNA, PFDA, PFUnDA, PFDoDA, PFTrDA, PFTeDA
Liu (2020)[269]	Does Low Maternal Exposure to Per- and Polyfluoroalkyl Substances Elevate the Risk of Spontaneous Preterm Birth? A Nested Case-Control Study in China.	Risk of spontaneous preterm birth	10.1021/acs.est.0c01930	PFOS, PFOA, 6:2 Cl-PFESA
Martinsson (2020)[270]	Intrauterine exposure to perfluorinated compounds and overweight at age 4: A case-control study.	Risk of obesity	10.1371/journal.pone.0230137	PFOS, PFOA, PFHxS, PFNA
Salihovic (2020)[271]	Plasma perfluoroalkyls are associated with decreased levels of proteomic inflammatory markers in a cross-sectional study of an elderly population	Decreased levels of proteomic inflammatory markers	10.1016/j.envint.2020.106099	PFNA, PFDA, PFUnDA, PFHxS, PFOS, PFOA, PFOSA
Shin (2020)[272]	Modeled prenatal exposure to per- and polyfluoroalkyl substances in association with child autism spectrum disorder: A case-control study.	Child autism spectrum disorder	10.1016/j.envres.2020.109514	PFHxS, PFOS, PFOA, PFNA, PFDA, PFUA, PFDoDA, Me-FOSAA, Et-FOSAA
Sinisalu (2020)[273]	Early-life exposure to perfluorinated alkyl substances modulates lipid metabolism in progression to celiac disease.	Celiac disease	10.1016/j.envres.2020.109864	PFHpA, PFHxS, PFOA, PFNA, PFOS, PFDA, PFUnDA
Souza (2020)[274]	Exposure to per- and polyfluorinated alkyl substances in pregnant Brazilian women and its association with fetal growth.	Fetal development risks	10.1016/j.envres.2020.109585	PFOS, PFOA

Timmermann (2020)[275]	Serum Perfluoroalkyl Substances, Vaccine Responses, and Morbidity in a Cohort of Guinea-Bissau Children	Morbidity issues and vaccine responses in young children	10.1289/EHP6517	PFHxS, PFOS, PFOA, PFNA, PFDA, PFUnDA
Tsai (2020)[276]	A case-control study of perfluoroalkyl substances and the risk of breast cancer in Taiwanese women.	Risk of breast cancer development	10.1016/j.envint.2020.105850	PFHxS, PFHpA, PFDA, PFUnDA, PFDoDA, PFHxA, PFOA, PFOS, PFNA,PFTrDA
Xu (2020)[277]	Exposure to elevated per- and polyfluoroalkyl substances in early pregnancy is related to increased risk of gestational diabetes mellitus: A nested case-control study in Shanghai, China	Risk of gestational diabetes mellitus	10.1016/j.envint.2020.105952	PFOA, PFOS, PFHpA, PFDS, PFOSA, PFBS, PFDoA
Bulka (2021)[278]	Associations of exposure to perfluoroalkyl substances individually and in mixtures with persistent infections: Recent findings from NHANES 1999-2016.	Increased risk of presistent infections	10.1016/j.envpol.2021.116619	PFOS, PFOA, PFHxS, PFNA
Clarity (2021)[279]	Associations between polyfluoroalkyl substance and organophosphate flame retardant exposures and telomere length in a cohort of women firefighters and office workers in San Francisco.	Cellular growth issues based on telomere length	10.1186/s12940-021-00778-z	PFOA, PFOA, PFDA, PFNA
Duan (2021)[280]	Serum concentrations of per-/polyfluoroalkyl substances and risk of type 2 diabetes: A case-control study.	Risk of type 2 diabetes	10.1016/j.scitotenv.2021.147476	PFPeA, PFHxA, PFHpA, PFOA, PFNA, PFDA, PFBS, PFHxS, L-PFDS, PFUnDA, PFDoDA, 6:2 Cl-PFESA, 8:2 Cl-PFESA
Fart (2021)[281]	Perfluoroalkyl substances are increased in patients with late-onset ulcerative colitis and	Increased risk of late-onset ulcerative colitis	10.1080/00365521.2021.1961306	PFOA, PFOS, PFNA, PFDA, PFHpA, PFUnDA, PFTrDA

	induce intestinal barrier defects ex vivo in murine intestinal tissue.			
Han (2021)[282]	Exposure to novel and legacy per- and polyfluoroalkyl substances (PFASs) and associations with type 2 diabetes: A case-control study in East China.	Associations with type 2 diabetes risk	10.1016/j.envint.2021.106637	PFOA, PFNA, PFUnDA, 6:2 Cl-PFESA
Luo (2021)[283]	Environmental exposure to per- and polyfluoroalkyl substances mixture and male reproductive hormones.	Issues in male reproductive hormone levels	10.1016/j.envint.2021.106496	PFOS, PFBS, F-53B, PFUnDA, PFDoA, PFTrDA
Oh (2021)[284]	Prenatal exposure to per- and polyfluoroalkyl substances in association with autism spectrum disorder in the MARBLES study.	Potentially increased risk of Autism spectrum disorders	10.1016/j.envint.2020.106328	PFOA, PFNA, PFOS, PFHxS, PFDA, PFUnDA, PFDoDA, MeFOSAA, EtFOSAA
Omoike (2021)[285]	A cross-sectional study of the association between perfluorinated chemical exposure and cancers related to deregulation of estrogen receptors.	Increased risk for cancer development dut to to deregulation of estrogen receptor expression	10.1016/j.envres.2020.110329	PFOA, PFOS, PFNA, PFHxS, PFDA
Ou (2021)[286]	Gestational exposure to perfluoroalkyl substances and congenital heart defects: A nested case-control pilot study.	Risk of congenital heart defects	10.1016/j.envint.2021.106567	PFOS, PFDA, PFDoA
Papadopoulou (2021)[287]	Prenatal and postnatal exposure to PFAS and cardiometabolic factors and inflammation status in children from six European cohorts.	Risk of cardiometabolic issues related to prenatal and postnatal exposure	10.1016/j.envint.2021.106853	PFHxS, PFOS, PFOA, PFNA, PFUnDA
Schillemans (2021)[288]	Plasma metabolites associated with exposure to perfluoroalkyl substances and risk of type 2	Risk of type 2 diabetes and levels of plasma	10.1016/j.envint.2020.106180	PFOA, PFOS, PFHxS, PFNA, PFDA, PFUnDA

	diabetes - A nested case-control study.	metabolites		
Skogheim (2021)[289]	Prenatal exposure to per- and polyfluoroalkyl substances (PFAS) and associations with attention-deficit/hyperactivity disorder and autism spectrum disorder in children.	Risk of autism spectrum disorder	10.1016/j.envres.2021.111692	PFOA, PFNA, PFDA, PFUnDA, PFHxS, PFHpS, PFOS
Yu (2021)[290]	Environmental exposure to perfluoroalkyl substances in early pregnancy, maternal glucose homeostasis and the risk of gestational diabetes: A prospective cohort study.	Risk of gestational diabetes	10.1016/j.envint.2021.106621	PFBS, PFHpA, PFOS, PFNA, PFDA, PFHxS
Yu (2021)[291]	Perfluorooctane sulfonate alternatives and metabolic syndrome in adults: New evidence from the Isomers of C8 Health Project in China.	Increased risk of metabolic syndrome	10.1016/j.envpol.2021.117078	6:2 Cl-PFESA, 8:2 Cl-PFESA
Batzella (2022)[292]	Perfluoroalkyl substance mixtures and cardio-metabolic outcomes in highly exposed male workers in the Veneto Region: A mixture-based approach.	Risk of developing cardio-metabolic issues	10.1016/j.envres.2022.113225	PFOS, PFOA, PFHxS, PFNA (Mixture modeling applied via WQS regression)
Gao (2022)[293]	Prenatal Exposure to Per- and Polyfluoroalkyl Substances and Child Growth Trajectories in the First Two Years.	Child Growth and developmental risks	10.1289/EHP9875	PFOA, PFOS, PFNA, PFUA, PFDA, PFHxS, PFBS, PFDoA, PFHpA, PFOSA
Ku (2022)[294]	Associations between prenatal exposure to perfluoroalkyl substances, hypomethylation of MEST imprinted gene and birth outcomes.	Increased hypomethylation of MEST imprinted gene and birth outcomes	10.1016/j.envpol.2022.119183	PFOS, PFOA
Li (2022)[295]	Cancer incidence in a Swedish cohort with high exposure to perfluoroalkyl substances in	Increased Cancer incidence	10.1016/j.envres.2021.112217	PFPeA, PFHxA, PFHpA, PFOA, PFBS, PFHxS,

	drinking water.			PFHpS, PFOS
Li (2022)[296]	Chlorinated Polyfluorinated Ether Sulfonates and Thyroid Hormone Levels in Adults: Isomers of C8 Health Project in China	Thyroid Hormone Levels	10.1021/acs.est.1c03757	6:2 Cl-PFESA, 8:2 Cl-PFESA, PFOS, PFOA
Liao (2022)[250]	Association of single and multiple prefluoroalkyl substances exposure with preterm birth: Results from a Chinese birth cohort study.	Preterm birth risk	10.1016/j.chemosphere.2022.135741	PFOS, PFHpA, PFBS, PFHxS, PFOA, PFUnA
Lin (2022)[297]	Association of maternal perfluoroalkyl substance exposure with postpartum haemorrhage in Guangxi, China.	Postpartum haemorrhage	10.1016/j.ecoenv.2022.114078	PFOS, PFHxS, PFDoA, PFUnA, PFNA
Liu (2022)[298]	Exposure to perfluoroalkyl substances in early pregnancy and the risk of hypertensive disorders of pregnancy: A nested case-control study in Guangxi, China.	Risk of hypertensive disorders of pregnancy	10.1016/j.chemosphere.2021.132468	PFOS, PFNA, PFBS, PFUnA, PFHxS, PFHpA, PFDoA
Lochhead (2022)[299]	Plasma concentrations of perfluoroalkyl substances and risk of inflammatory bowel diseases in women: A nested case control analysis in the Nurses' Health Study cohorts.	Risk of inflammatory bowel disease	10.1016/j.envres.2021.112222	PFOA, PFHxS, PFNA, PFDA, PFOS
Nian (2022)[300]	Association of emerging and legacy per- and polyfluoroalkyl substances with unexplained recurrent spontaneous abortion.	Unexplained recurrent spontaneous abortion risk	10.1016/j.ecoenv.2022.113691	6:2 Cl-PFESA, 8:2 Cl-PFESA, HFPO-DA, PFOA, PFOS, PFNA, PFDA, PFHxS, PFHpS, PFBS, PFBA, PFHxA, PFHpA, PFDoA, PFUnDA
Oh	Childhood exposure to per- and polyfluoroalkyl substances and neurodevelopment in the	Neurodevelopmental risks	10.1016/j.envres.2022.1	PFOA, PFHpA, PFDA,

(2022)[301]	CHARGE case-control study.		14322	PFUnDA
Qu (2022)[302]	Evaluated serum perfluoroalkyl acids and their relationships with the incidence of rheumatoid arthritis in the general population in Hangzhou, China	Incidence of rheumatoid arthritis	10.1016/j.envpol.2022.119505	PFOS, PFOA, PFNA, PFDA, PFUnA, PFDoA, PFTrA
Xu (2022)[303]	Perfluoroalkyl substances influence DNA methylation in school-age children highly exposed through drinking water contaminated from firefighting foam: a cohort study in Ronneby, Sweden.	Effect on DNA methylation levels	10.1093/eep/dvac004	PFOS, PFHxS, PFOA
Yao (2022)[304]	Prenatal exposure to per- and polyfluoroalkyl substances, fetal thyroid hormones, and infant neurodevelopment.	Neurodevelopmental risk	10.1016/j.envres.2021.112561	PFBS, PFHxS, PFOS, PFOA, PFHpA, PFNA, PFDA, PFDoA, PFUA, PFOSA
Yu (2022)[305]	Associations of prenatal exposure to perfluoroalkyl substances with preterm birth: A family-based birth cohort study.	Preterm birth risk	10.1016/j.envres.2022.113803	PFBA, PFHxA, PFOA, PFNA, PFDA, PFHxS, PFOS
Zhang (2022)[306]	Association between serum per- and polyfluoroalkyl substances concentrations and common cold among children and adolescents in the United States.	Risk of increased susceptibility in infectious agents	10.1016/j.envint.2022.107239	PFHxS , PFNA, PFOA, PFOS
Zhao (2022)[307]	The influences of perfluoroalkyl substances on the rheumatoid arthritis clinic	Rheumatoid arthritis risk	10.1186/s12865-022-00483-7	PFOA, PFNA, PFTrA, PFOS, 8:2 Cl-PFESA, PFDA, PFUnA
Bailey (2023)[308]	Immune response to COVID-19 vaccination in a population with a history of elevated exposure to per- and polyfluoroalkyl substances (PFAS)	Effect on Immune responses to COVID-19	10.1038/s41370-023-00564-8	39 PFAS

	through drinking water.	vaccination		
Dai (2023)[309]	Per- and polyfluoroalkyl substances in umbilical cord serum and body mass index trajectories from birth to age 10 years: Findings from a longitudinal birth cohort (SMBCS).	Effects on body mass index and development	10.1016/j.envint.2023.108238	PFBS, PFHxS, PFHpA, PFHpS, PFOA, PFOS, PFNA, PFDA, PFUnDA, PFDoA, PFDS, PFOSA
He (2023)[310]	Novel Insights into the Adverse Health Effects of per- and Polyfluoroalkyl Substances on the Kidney via Human Urine Metabolomics	Increased risk of kidney issues	10.1021/acs.est.3c06480	23 PFAS
Li (2023)[311]	Association of exposure to perfluoroalkyl substances and risk of the acute coronary syndrome: A case-control study in Shijiazhuang Hebei Province.	Risk of the acute coronary syndrome	10.1016/j.chemosphere.2022.137464	PFOS, PFOS, PFHxS, HFPO-DA
Li (2023)[312]	Per- and polyfluoroalkyl substances and the associated thyroid cancer risk: A case-control study in China.	Thyroid cancer development risk	10.1016/j.chemosphere.2023.139411	PFOA, PFNA, PFHxS, PFDA, PFUnDA
Liang (2023)[313]	Joint effects of per- and polyfluoroalkyl substance alternatives and heavy metals on renal health: A community-based population study in China.	Effects on renal health	10.1016/j.envres.2022.115057	6:2 Cl-PFESA, 8:2 Cl-PFESA
Rhee (2023)[314]	Serum concentrations of per- and polyfluoroalkyl substances and risk of renal cell carcinoma in the Multiethnic Cohort Study.	Risk of renal cell carcinoma development	10.1016/j.envint.2023.108197	PFHxS, n-PFOS, Sm-PFOS, PFOA, PFNA, PFDA, PFUnDA, FOSA, MeFOSAA, EtFOSAA
Siwakoti (2023)[315]	Prenatal per- and polyfluoroalkyl substances (PFAS) exposure in relation to preterm birth subtypes and size-for-gestational age in the	Preterm birth risk	10.1016/j.envres.2023.116967	PFHP, PFHxS, PFNA, PFOA, PFOS, PFOSA,

	LIFECODES cohort 2006-2008.			PFUA
Tian (2023)[253]	In utero exposure to per-/polyfluoroalkyl substances (PFASs): Preeclampsia in pregnancy and low birth weight for neonates.	Low birth weight	10.1016/j.chemosphere.2022.137490	PFOA, 6:2 Cl-PFESA, PFUnDA, 4:2 FTS, ADONA
Xu (2023)[316]	Exposure to high levels of PFAS through drinking water is associated with increased risk of type 2 diabetes-findings from a register-based study in Ronneby, Sweden.	Risk of type 2 diabetes	10.1016/j.envres.2023.115525	PFAS
Zang (2023)[317]	Exposure to per- and polyfluoroalkyl substances in early pregnancy, risk of gestational diabetes mellitus, potential pathways, and influencing factors in pregnant women: A nested case-control study.	Risk of gestational diabetes mellitus	10.1016/j.envpol.2023.121504	PFBA, PFPeA, PFHxA, PFNA, PFHpA, PFOA, PFUnDA, PFDoDA, PFBS, PFPeS, PFHxS, PFHpS, PFHxS, PFOS, 6:2 Cl-PFESA, 8:2 Cl-PFESA
Zhang (2023)[318]	Prenatal perfluoroalkyl substances exposure and neurodevelopment in toddlers: Findings from SMBCS.	Neurodevelopmental issues	10.1016/j.chemosphere.2022.137587	PFOA, PFOS, PFHpA, PFBS, PFNA, PFDA, PFHxS, PFUnDA, PFDoDA, PFHpS, PFDS, PFOSA
van Gerwen (2023)[319]	Per- and polyfluoroalkyl substances (PFAS) exposure and thyroid cancer risk	Risk of thyroid cancer development	10.1016/j.ebiom.2023.104831	PFOS, PFOA, PFNA, PFHxS, PFHpS
Ao (2024)[320]	Environmental exposure to legacy and emerging per- and polyfluoroalkyl substances and endometriosis in women of childbearing age.	Endometriosis	10.1016/j.scitotenv.2023.167838	PFOA, PFOS
Chen	Neonatal per- and polyfluoroalkyl substance	Retinoblastoma	10.1016/j.envres.2023.1	PFOA, PFOS, PFNA

(2024)[321]	exposure in relation to retinoblastoma.	development	17435	

Abbreviations

6:2 Cl-PFESA (or F-53B)	Perfluoro(2-(6-chlorohexyl) oxy) ethanesulfonic acid)
6:2 FTCA	2-(Perfluorohexyl)ethanoic acid
6:2 FTOH	2-(Perfluorohexyl)ethanol
6:2 FTSA	6:2 Fluorotelomer sulfonic acid
8:2 Cl-PFESA	11-chloroeicosafluoro-3-oxaundecane-1-sulfonic acid
8:8 PFPiA	8:8 Perfluoroalkyl phosphonic acid
ACE2	Angiotensin-converting enzyme 2
ALT	Alanine transaminase (liver damage marker)
AMPA	α-amino-3-hydroxy-5-methyl-4-isoxazole propionic acid
BBB	Blood–brain barrier
CAR	Constitutive androstane receptor
DEGs	Differentially expressed genes
E2	17β- estradiol
EC_{50}	Half- maximal effective concentration
ER	Estrogen receptor
FA	Fatty acid
HFPO-DA	Hexafluoropropylene oxide dimer acid
HFPO-TA	Hexafluoropropylene oxide trimer acid
HFPO-TeA	Hexafluoropropylene oxide tetramer acid
IgM	Immunoglobulin M
IL	Interleukin
LC_{50}	Median lethal concentration
LOEC	Lowest observed effect concentration
LXR	Liver X receptor
LYPD6	Ly6/PLAUR Domain Containing 6 (gene coding brain related protein)
MyD88	Myeloid differentiation factor 88
NBP2	Nafion by-product 2
NK	Natural killer
OBS	Sodium p-perfluorous nonenoxybenzene sulfonate
PFAS	Per- and Polyfluoroalkyl Substances
PFBS	Perfluorobutane sulfonic acid
PFCAs	Perfluoroalkyl carboxylic acids
PFDA	Perfluorodecanoic acid
PFECAs	Perfluoroalkyl ether carboxylic acids
PFECHS	Perfluoro ethyl cyclohexane sulfonate
PFHpA	Perfluoroheptanoic acid
PFHxA	Perfluorohexanoic acid
PFHxS	Perfluorohexane sulfonic acid
PFMOAA	2-methoxyacetic acid
PFMOBA	Perfluoro4-methoxybutanioc acid
PFMOPrA	Perfluoro-2-methoxypropanoic acid
PFNA	Perfluorononanoic acid
PFO2OA	Perfluoro diEther octananoic acid
PFOA	Perfluorooctanoic acid
PFOS	Perfluorooctane sulfonic acid
PFOSA	Perfluorooctanesulfonamide
PFPeA	Perfluoropentanoic acid
PFSAs	Perfluoroalkyl sulfonic acids
PFTA	Perfluorotetradecanoic acid
PFUnDA	Perfluoroundecanoic acid
PPARα	Peroxisome proliferator-activated receptor α
PXR	Pregnane X receptor

ROS	Reactive oxygen species
SNAP91	Synaptosome associated protein 91
SOD	Superoxide dismutase
TNF	Tumor necrosis factor

References

1 S. Daugherty, V. Mulabagal, J. Hayworth and B. T. Akingbemi, Legacy and Emerging Perfluoroalkyl and Polyfluoroalkyl Substances Regulate Steroidogenesis in the Male Gonad., *Endocrinology*, 2023, **164**, bqad142.

2 J. M. Conley, C. S. Lambright, N. Evans, E. Medlock-Kakaley, A. Dixon, D. Hill, J. McCord, M. J. Strynar, J. Ford and L. E. J. Gray, Cumulative maternal and neonatal effects of combined exposure to a mixture of perfluorooctanoic acid (PFOA) and perfluorooctane sulfonic acid (PFOS) during pregnancy in the Sprague-Dawley rat., *Environment international*, 2022, **170**, 107631.

3 J. M. Conley, C. S. Lambright, N. Evans, J. McCord, M. J. Strynar, D. Hill, E. Medlock-Kakaley, V. S. Wilson and L. E. J. Gray, Hexafluoropropylene oxide-dimer acid (HFPO-DA or GenX) alters maternal and fetal glucose and lipid metabolism and produces neonatal mortality, low birthweight, and hepatomegaly in the Sprague-Dawley rat., *Environment international*, 2021, **146**, 106204.

4 J. M. Conley, C. S. Lambright, N. Evans, A. K. Farraj, J. Smoot, R. D. Grindstaff, D. Hill, J. McCord, E. Medlock-Kakaley, A. Dixon, E. Hines and L. E. J. Gray, Dose additive maternal and offspring effects of oral maternal exposure to a mixture of three PFAS (HFPO-DA, NBP2, PFOS) during pregnancy in the Sprague-Dawley rat., *The Science of the total environment*, 2023, **892**, 164609.

5 Q. Zhang, W. Liu, H. Zhao, Z. Zhang, H. Qin, F. Luo and Q. Niu, Developmental perfluorooctane sulfonate exposure inhibits long-term potentiation by affecting AMPA receptor trafficking., *Toxicology*, 2019, **412**, 55–62.

6 H. Zhang, H. Lu, L. Yu, J. Yuan, S. Qin, C. Li, R.-S. Ge, H. Chen and L. Ye, Effects of gestational exposure to perfluorooctane sulfonate on the lung development of offspring rats., *Environmental pollution (Barking, Essex : 1987)*, 2021, **272**, 115535.

7 R. Zhang, G. Yu, T. Luo, X. Zeng, Y. Sun, B. Huang, Y. Liu and J. Zhang, Transcriptomic and metabolomic profile changes in the liver of Sprague Dawley rat offspring after maternal PFOS exposure during gestation and lactation., *Ecotoxicology and environmental safety*, 2024, **270**, 115862.

8 Y. Liu, G. Yu, R. Zhang, L. Feng and J. Zhang, Early life exposure to low-dose perfluorooctane sulfonate disturbs gut barrier homeostasis and increases the risk of intestinal inflammation in offspring., *Environmental pollution (Barking, Essex : 1987)*, 2023, **329**, 121708.

9 S. V. Dangudubiyyam, J. S. Mishra, H. Zhao and S. Kumar, Perfluorooctane sulfonic acid (PFOS) exposure during pregnancy increases blood pressure and impairs vascular relaxation mechanisms in the adult offspring., *Reproductive toxicology (Elmsford, N.Y.)*, 2020, **98**, 165–173.

10 S. V. Dangudubiyyam, J. S. Mishra and S. Kumar, Perfluorooctane sulfonic acid modulates expression of placental steroidogenesis-associated genes and hormone levels in pregnant rats., *Reproductive toxicology (Elmsford, N.Y.)*, 2023, **118**, 108390.

11 D. Xu, L. Li, L. Tang, M. Guo and J. Yang, Perfluorooctane sulfonate induces heart toxicity involving cardiac apoptosis and inflammation in rats., *Experimental and therapeutic medicine*, 2022, **23**, 14.
12 N. Zhao, Y. Kong, Q. Yuan, Z. Wei, J. Gu, C. Ji, H. Jin and M. Zhao, The toxic mechanism of 6:2 Cl-PFESA in adolescent male rats: Endocrine disorders and liver inflammation regulated by the gut microbiota-gut-testis/liver axis., *Journal of hazardous materials*, 2023, **459**, 132155.
13 G. Du, J. Hu, Z. Huang, M. Yu, C. Lu, X. Wang and D. Wu, Neonatal and juvenile exposure to perfluorooctanoate (PFOA) and perfluorooctane sulfonate (PFOS): Advance puberty onset and kisspeptin system disturbance in female rats., *Ecotoxicology and environmental safety*, 2019, **167**, 412–421.
14 J.-S. Han, S. Jang, H.-Y. Son, Y.-B. Kim, Y. Kim, J.-H. Noh, M.-J. Kim and B.-S. Lee, Subacute dermal toxicity of perfluoroalkyl carboxylic acids: comparison with different carbon-chain lengths in human skin equivalents and systemic effects of perfluoroheptanoic acid in Sprague Dawley rats., *Archives of toxicology*, 2020, **94**, 523–539.
15 L. Tang, J. Yu, S. Zhuge, H. Chen, L. Zhang and G. Jiang, Oxidative stress and Cx43-mediated apoptosis are involved in PFOS-induced nephrotoxicity., *Toxicology*, 2022, **478**, 153283.
16 H. Liang, M. Yang, C. Zeng, W. Wu, L. Zhao and Y. Wang, Perfluorooctane sulfonate exerts inflammatory bowel disease-like intestinal injury in rats., *PeerJ*, 2021, **9**, e10644.
17 X. Zhang, X. Ren, W. Sun, N. Griffin, L. Wang and H. Liu, PFOA exposure induces aberrant glucose and lipid metabolism in the rat liver through the AMPK/mTOR pathway., *Toxicology*, 2023, **493**, 153551.
18 S.-H. Hong, S. H. Lee, J.-Y. Yang, J. H. Lee, K. K. Jung, J. H. Seok, S.-H. Kim, K. T. Nam, J. Jeong, J. K. Lee and J.-H. Oh, Orally Administered 6:2 Chlorinated Polyfluorinated Ether Sulfonate (F-53B) Causes Thyroid Dysfunction in Rats., *Toxics*, DOI:10.3390/toxics8030054.
19 A. Renyer, K. Ravindra, B. A. Wetmore, J. L. Ford, M. DeVito, M. F. Hughes, L. C. Wehmas and D. K. MacMillan, Dose Response, Dosimetric, and Metabolic Evaluations of Replacement PFAS Perfluoro-(2,5,8-trimethyl-3,6,9-trioxadodecanoic) Acid (HFPO-TeA)., *Toxics*, DOI:10.3390/toxics11120951.
20 V. Basaly, J. Hill, S. W. Bihaqi, E. Marques, A. L. Slitt and N. H. Zawia, Developmental Perfluorooctanesulfonic acid (PFOS) exposure as a potential risk factor for late-onset Alzheimer's disease in CD-1 mice and SH-SY5Y cells., *Neurotoxicology*, 2021, **86**, 26–36.
21 H. T. Wan, L. Y. Cheung, T. F. Chan, M. Li, K. P. Lai and C. K. C. Wong, Characterization of PFOS toxicity on in-vivo and ex-vivo mouse pancreatic islets., *Environmental pollution (Barking, Essex : 1987)*, 2021, **289**, 117857.
22 Z. Li, Z. Lin, S. Ji, K.-P. Lai, H.-T. Wan, C. K. C. Wong and L. Li, Perfluorooctanesulfonic acid exposure altered hypothalamic metabolism and disturbed male fecundity, *Science of the Total Environment*, DOI:10.1016/j.scitotenv.2022.156881.
23 L. Zhao, M. Teng, D. Shi, J. Sun, Y. Li, Z. Zhang, W. Zhu and F. Wu, Adverse impacts of environmentally relevant PFOS alternatives on mice pancreatic tissues, *Sci. Total Environ.*, 2024, **909**, 168649.

24 F. Rashid, V. Dubinkina, S. Ahmad, S. Maslov and J. M. K. Irudayaraj, Gut Microbiome-Host Metabolome Homeostasis upon Exposure to PFOS and GenX in Male Mice., *Toxics*, DOI:10.3390/toxics11030281.
25 F. Rashid, S. Ahmad and J. M. K. Irudayaraj, Effect of Perfluorooctanoic Acid on the Epigenetic and Tight Junction Genes of the Mouse Intestine., *Toxics*, DOI:10.3390/toxics8030064.
26 M. Yang, Y. Lee, L. Gao, K. Chiu, D. D. Meling, J. A. Flaws and G. R. Warner, Perfluorooctanoic Acid Disrupts Ovarian Steroidogenesis and Folliculogenesis in Adult Mice., *Toxicological sciences : an official journal of the Society of Toxicology*, 2022, **186**, 260–268.
27 B. E. Blake, H. A. Cope, S. M. Hall, R. D. Keys, B. W. Mahler, J. McCord, B. Scott, H. M. Stapleton, M. J. Strynar, S. A. Elmore and S. E. Fenton, Evaluation of Maternal, Embryo, and Placental Effects in CD-1 Mice following Gestational Exposure to Perfluorooctanoic Acid (PFOA) or Hexafluoropropylene Oxide Dimer Acid (HFPO-DA or GenX)., *Environmental health perspectives*, 2020, **128**, 27006.
28 B. E. Blake, C. N. Miller, H. Nguyen, V. A. Chappell, T. P. Phan, D. P. Phadke, M. R. Balik-Meisner, D. Mav, R. R. Shah and S. E. Fenton, Transcriptional pathways linked to fetal and maternal hepatic dysfunction caused by gestational exposure to perfluorooctanoic acid (PFOA) or hexafluoropropylene oxide-dimer acid (HFPO-DA or GenX) in CD-1 mice., *Ecotoxicology and environmental safety*, 2022, **248**, 114314.
29 H. A. Cope, B. E. Blake, C. Love, J. McCord, S. A. Elmore, J. B. Harvey, V. A. Chappell and S. E. Fenton, Latent, sex-specific metabolic health effects in CD-1 mouse offspring exposed to PFOA or HFPO-DA (GenX) during gestation., *Emerging contaminants*, 2021, **7**, 219–235.
30 B. Zhu, N. Sheng and J. Dai, Adverse effects of gestational exposure to hexafluoropropylene oxide trimer acid (HFPO-TA) homologs on maternal, fetal, and placental health in mice, *Sci. Total Environ.*, 2024, **912**, 169151.
31 E. S. Marques, J. Agudelo, E. M. Kaye, S. M. S. Modaresi, M. Pfohl, J. Bečanová, W. Wei, M. Polunas, M. Goedken and A. L. Slitt, The role of maternal high fat diet on mouse pup metabolic endpoints following perinatal PFAS and PFAS mixture exposure., *Toxicology*, 2021, **462**, 152921.
32 L. Zhang, B. Rimal, R. G. Nichols, Y. Tian, P. B. Smith, E. Hatzakis, S.-C. Chang, J. L. Butenhoff, J. M. Peters and A. D. Patterson, Perfluorooctane sulfonate alters gut microbiota-host metabolic homeostasis in mice., *Toxicology*, 2020, **431**, 152365.
33 E. Marques, M. Pfohl, A. Auclair, R. Jamwal, B. J. Barlock, F. M. Sammoura, M. Goedken, F. Akhlaghi and A. L. Slitt, Perfluorooctanesulfonic acid (PFOS) administration shifts the hepatic proteome and augments dietary outcomes related to hepatic steatosis in mice., *Toxicology and applied pharmacology*, 2020, **408**, 115250.
34 D. Luo, J. Zhang, H. Yin, S. Li, S. Xu and S. Li, Cannabidiol alleviates perfluorooctane sulfonate-induced macrophage extracellular trap mediate inflammation and fibrosis in mice liver, *Ecotoxicology and Environmental Safety*, DOI:10.1016/j.ecoenv.2023.115374.
35 T. Li, L. Jiang, S. Zheng, C. Qiu, N. Zhao, X. Lin, H. Ren, J. Huang, H. Wang and L. Qiu, Organic anion transporting polypeptide 3a1 is a novel influx pump for

Perfluorooctane sulfonate in Sertoli cells and contributes to its reproductive toxicity., *Chemosphere*, 2023, **345**, 140428.
36 A. Mshaty, A. Haijima, Y. Takatsuru, A. Ninomiya, H. Yajima, M. Kokubo, M. A. Khairinisa, W. Miyazaki, I. Amano and N. Koibuchi, Neurotoxic effects of lactational exposure to perfluorooctane sulfonate on learning and memory in adult male mouse., *Food and chemical toxicology : an international journal published for the British Industrial Biological Research Association*, 2020, **145**, 111710.
37 A. Ninomiya, A. Mshaty, A. Haijima, H. Yajima, M. Kokubo, M. A. Khairinisa, W. Ariyani, Y. Fujiwara, S. Ishii, N. Hosoi, H. Hirai, I. Amano and N. Koibuchi, The neurotoxic effect of lactational PFOS exposure on cerebellar functional development in male mice., *Food and chemical toxicology : an international journal published for the British Industrial Biological Research Association*, 2022, **159**, 112751.
38 Z. Pan, W. Miao, C. Wang, W. Tu, C. Jin and Y. Jin, 6:2 Cl-PFESA has the potential to cause liver damage and induce lipid metabolism disorders in female mice through the action of PPAR-γ, *Environ. Pollut.*, 2021, **287**, 117329.
39 C. A. McDonough, C. Ward, Q. Hu, S. Vance, C. P. Higgins and J. C. DeWitt, Immunotoxicity of an Electrochemically Fluorinated Aqueous Film-Forming Foam., *Toxicological sciences : an official journal of the Society of Toxicology*, 2020, **178**, 104–114.
40 M. Yang, L.-Y. Li, X.-D. Qin, X.-Y. Ye, S. Yu, Q. Bao, L. Sun, Z.-B. Wang, M. S. Bloom, P. Jalava, L.-W. Hu, H.-Y. Yu, X.-W. Zeng, B.-Y. Yang, G.-H. Dong and C.-W. Li, Perfluorooctanesulfonate and perfluorooctanoate exacerbate airway inflammation in asthmatic mice and in vitro, *The Science of the Total Environment*, 2021, **766**, 142365.
41 L. Chen, Y. Liu, H. Mu, H. Li, S. Liu, M. Zhu, Y. Bu and B. Wu, Effects of perfluorobutane sulfonate and perfluorooctane sulfonate on lipid homeostasis in mouse liver., *Environmental pollution (Barking, Essex : 1987)*, 2022, **315**, 120403.
42 L. Chen, M. Zhu, Y. Liu, Z. Yang, H. Li, H. Mu, S. Liu and B. Wu, Perfluorobutanesulfonate exposure induces metabolic disturbances in different regions of mouse gut., *The Science of the total environment*, 2023, **866**, 161261.
43 K. Roth, Z. Yang, M. Agarwal, W. Liu, Z. Peng, Z. Long, J. Birbeck, J. Westrick, W. Liu and M. C. Petriello, Exposure to a mixture of legacy, alternative, and replacement per- and polyfluoroalkyl substances (PFAS) results in sex-dependent modulation of cholesterol metabolism and liver injury., *Environment international*, 2021, **157**, 106843.
44 Z. Yang, K. Roth, J. Ding, C. D. Kassotis, G. Mor and M. C. Petriello, Exposure to a mixture of per-and polyfluoroalkyl substances modulates pulmonary expression of ACE2 and circulating hormones and cytokines., *Toxicology and applied pharmacology*, 2022, **456**, 116284.
45 M. Pfohl, L. Ingram, E. Marques, A. Auclair, B. Barlock, R. Jamwal, D. Anderson, B. S. Cummings and A. L. Slitt, Perfluorooctanesulfonic Acid and Perfluorohexanesulfonic Acid Alter the Blood Lipidome and the Hepatic Proteome in a Murine Model of Diet-Induced Obesity., *Toxicological sciences : an official journal of the Society of Toxicology*, 2020, **178**, 311–324.
46 X. Li, Z. Wang and J. E. Klaunig, The effects of perfluorooctanoate on high fat diet induced non-alcoholic fatty liver disease in mice., *Toxicology*, 2019, **416**, 1–14.

47 M. Soltani, M. Pourfarzam, A. H. Sharifabad, A. K. Neisiani, M. K. Mousavi and M. Aliomrani, Effect of pretreatment with a synbiotic on Perfluorooctanoic acid-induced liver damage after sub-acute oral exposure in C57BL/6J mice., *Toxicology and applied pharmacology*, 2023, **459**, 116360.
48 K. D. Taylor, T. L. Woodlief, A. Ahmed, Q. Hu, P. C. Duncker and J. C. DeWitt, Quantifying the impact of PFOA exposure on B-cell development and antibody production., *Toxicological sciences : an official journal of the Society of Toxicology*, 2023, **194**, 101–108.
49 Z. Weng, C. Xu, X. Zhang, L. Pang, J. Xu, Q. Liu, L. Zhang, S. Xu and A. Gu, Autophagy mediates perfluorooctanoic acid-induced lipid metabolism disorder and NLRP3 inflammasome activation in hepatocytes., *Environmental pollution (Barking, Essex : 1987)*, 2020, **267**, 115655.
50 X. Wen, A. A. Baker, C. D. Klaassen, J. C. Corton, J. R. Richardson and L. M. Aleksunes, Hepatic carboxylesterases are differentially regulated in PPARα-null mice treated with perfluorooctanoic acid., *Toxicology*, 2019, **416**, 15–22.
51 R. Crebelli, S. Caiola, L. Conti, E. Cordelli, G. De Luca, E. Dellatte, P. Eleuteri, N. Iacovella, P. Leopardi, F. Marcon, M. Sanchez, P. Sestili, E. Siniscalchi and P. Villani, Can sustained exposure to PFAS trigger a genotoxic response? A comprehensive genotoxicity assessment in mice after subacute oral administration of PFOA and PFBA., *Regulatory toxicology and pharmacology : RTP*, 2019, **106**, 169–177.
52 T. Woodlief, S. Vance, Q. Hu and J. DeWitt, Immunotoxicity of Per- and Polyfluoroalkyl Substances: Insights into Short-Chain PFAS Exposure., *Toxics*, DOI:10.3390/toxics9050100.
53 L. Hu, L. Sun, J. Zhou, F. Wu, Z. Fu and X. Xie, Impact of a hexafluoropropylene oxide trimer acid (HFPO-TA) exposure on impairing the gut microbiota in mice., *Chemosphere*, 2022, **303**, 134951.
54 W.-P. Qin, L.-Y. Cao, C.-H. Li, L.-H. Guo, J. Colbourne and X.-M. Ren, Perfluoroalkyl Substances Stimulate Insulin Secretion by Islet β Cells via G Protein-Coupled Receptor 40., *Environmental science & technology*, 2020, **54**, 3428–3436.
55 H. Guo, H. Zhang, N. Sheng, J. Wang, J. Chen and J. Dai, Perfluorooctanoic acid (PFOA) exposure induces splenic atrophy via overactivation of macrophages in male mice., *Journal of hazardous materials*, 2021, **407**, 124862.
56 B.-X. Peng, F. Li, M. Mortimer, X. Xiao, Y. Ni, Y. Lei, M. Li and L.-H. Guo, Perfluorooctanoic acid alternatives hexafluoropropylene oxides exert male reproductive toxicity by disrupting blood-testis barrier., *The Science of the total environment*, 2022, **846**, 157313.
57 H. Guo, J. Wang, J. Yao, S. Sun, N. Sheng, X. Zhang, X. Guo, Y. Guo, Y. Sun and J. Dai, Comparative Hepatotoxicity of Novel PFOA Alternatives (Perfluoropolyether Carboxylic Acids) on Male Mice, *Environmental Science and Technology*, 2019, **53**, 3929–3937.
58 H. Guo, J. Chen, H. Zhang, J. Yao, N. Sheng, Q. Li, Y. Guo, C. Wu, W. Xie and J. Dai, Exposure to GenX and Its Novel Analogs Disrupts Hepatic Bile Acid Metabolism in Male Mice., *Environmental science & technology*, 2022, **56**, 6133–6143.
59 C. Wang, H. Fu, J. Yang, L. Liu, F. Zhang, C. Yang, H. Li, J. Chen, Q. Li, X. Wang, Y. Ye, N. Sheng, Y. Guo, J. Dai, G. Xu, X. Liu and J. Wang, PFO5DoDA disrupts hepatic homeostasis primarily through glucocorticoid signaling inhibition., *Journal of hazardous materials*, 2023, **447**, 130831.

60 F. Zhang, L. Liu, J. Hu, H. Fu, H. Li, J. Chen, C. Yang, Q. Guo, X. Liang, L. Wang, Y. Guo, J. Dai, N. Sheng and J. Wang, Accumulation and glucocorticoid signaling suppression by four emerging perfluoroethercarboxylic acids based on animal exposure and cell testing, *Environ. Int.*, 2023, **178**, 108092.
61 B. Ünlü Endirlik, A. Eken, F. Öztürk and A. Gürbay, Assessing perfluorooctanoic acid toxicity in lung, heart, and testis tissues of mice: Evaluation of protective effects of taurine and coenzyme Q10, *Journal of Research in Pharmacy*, 2023, **27**, 441–453.
62 L.-L. Xu, Y.-K. Chen, Q.-Y. Zhang, L.-J. Chen, K.-K. Zhang, J.-H. Li, J.-L. Liu, Q. Wang and X.-L. Xie, Gestational exposure to GenX induces hepatic alterations by the gut-liver axis in maternal mice: A similar mechanism as PFOA., *The Science of the total environment*, 2022, **820**, 153281.
63 P. Zhang, C. Qi, Z. Ma, Y. Wang, L. Zhang and X. Hou, Perfluorooctanoic acid exposure in vivo perturbs mitochondrial metabolic during oocyte maturation., *Environmental toxicology*, 2022, **37**, 2965–2976.
64 Y.-T. Zhou, R. Li, S.-H. Li, X. Ma, L. Liu, D. Niu and X. Duan, Perfluorooctanoic acid (PFOA) exposure affects early embryonic development and offspring oocyte quality via inducing mitochondrial dysfunction., *Environment international*, 2022, **167**, 107413.
65 Y. Zhao, H. Zhao, H. Xu, P. An, B. Ma, H. Lu, Q. Zhou, X. Li and Y. Xiong, Perfluorooctane sulfonate exposure induces preeclampsia-like syndromes by damaging trophoblast mitochondria in pregnant mice, *Ecotoxicology and Environmental Safety*, DOI:10.1016/j.ecoenv.2022.114256.
66 Y. Yu, C. Wang, X. Zhang, J. Zhu, L. Wang, M. Ji, Z. Zhang, X.-M. Ji and S.-L. Wang, Perfluorooctane sulfonate disrupts the blood brain barrier through the crosstalk between endothelial cells and astrocytes in mice., *Environmental pollution (Barking, Essex : 1987)*, 2020, **256**, 113429.
67 R. Grønnestad, S. M. Johanson, M. H. B. Müller, D. Schlenk, P. Tanabe, Å. Krøkje, V. L. B. Jaspers, B. M. Jenssen, E. M. Ræder, J. L. Lyche, Q. Shi and A. Arukwe, Effects of an environmentally relevant PFAS mixture on dopamine and steroid hormone levels in exposed mice., *Toxicology and applied pharmacology*, 2021, **428**, 115670.
68 E. A. Khan, R. Grønnestad, Å. Krøkje, Z. Bartosov, S. M. Johanson, M. H. B. Müller and A. Arukwe, Alteration of hepato-lipidomic homeostasis in A/J mice fed an environmentally relevant PFAS mixture, *Environ. Int.*, 2023, **173**, 107838.
69 S. De Guise and M. Levin, Suppression of Th2 cytokines as a potential mechanism for reduced antibody response following PFOA exposure in female B6C3F1 mice, *Toxicology letters*, 2021, **351**, 155–162.
70 H. L. Shane, R. Baur, E. Lukomska, L. Weatherly and S. E. Anderson, Immunotoxicity and allergenic potential induced by topical application of perfluorooctanoic acid (PFOA) in a murine model., *Food and chemical toxicology : an international journal published for the British Industrial Biological Research Association*, 2020, **136**, 111114.
71 L. M. Weatherly, H. L. Shane, E. Lukomska, R. Baur and S. E. Anderson, Systemic toxicity induced by topical application of heptafluorobutyric acid (PFBA) in a murine model., *Food and chemical toxicology : an international journal published for the British Industrial Biological Research Association*, 2021, **156**, 112528.
72 L. M. Weatherly, H. L. Shane, E. Lukomska, R. Baur and S. E. Anderson, Systemic toxicity induced by topical application of perfluoroheptanoic acid (PFHpA),

perfluorohexanoic acid (PFHxA), and perfluoropentanoic acid (PFPeA) in a murine model., *Food and chemical toxicology : an international journal published for the British Industrial Biological Research Association*, 2023, **171**, 113515.
73 D. Wang, Z. Tan, J. Yang, L. Li, H. Li, H. Zhang, H. Liu, Y. Liu, L. Wang, Q. Li and H. Guo, Perfluorooctane sulfonate promotes atherosclerosis by modulating M1 polarization of macrophages through the NF-κB pathway., *Ecotoxicology and environmental safety*, 2023, **249**, 114384.
74 H. J. Yoo, M. C. Pyo, K. H. Rhee, J.-M. Lim, S.-A. Yang, M. K. Yoo and K.-W. Lee, Perfluorooctanoic acid (PFOA) and hexafluoropropylene oxide-dimer acid (GenX): Hepatic stress and bile acid metabolism with different pathways., *Ecotoxicology and environmental safety*, 2023, **259**, 115001.
75 M. Estefanía González-Alvarez, A. Severin, M. Sayadi and A. F. Keating, PFOA-Induced Ovotoxicity Differs Between Lean and Obese Mice With Impacts on Ovarian Reproductive and DNA Damage Sensing and Repair Proteins., *Toxicological sciences : an official journal of the Society of Toxicology*, 2022, **190**, 173–188.
76 M. E. González-Alvarez and A. F. Keating, Hepatic and ovarian effects of perfluorooctanoic acid exposure differ in lean and obese adult female mice., *Toxicology and applied pharmacology*, 2023, **474**, 116614.
77 L. Shi, X. Deng, X. Liu, S. Yan, Y. Wang, Y. Li, J. Zheng, L. Jiang, X. Liu and C. Xiao, The effect of chronic exposure to a low concentration of perfluorooctanoic acid on cognitive function and intestinal health of obese mice induced by a high-fat diet., *Food and chemical toxicology : an international journal published for the British Industrial Biological Research Association*, 2022, **168**, 113395.
78 D. Li, L. Zhang, Y. Zhang, S. Guan, X. Gong and X. Wang, Maternal exposure to perfluorooctanoic acid (PFOA) causes liver toxicity through PPAR-α pathway and lowered histone acetylation in female offspring mice., *Environmental science and pollution research international*, 2019, **26**, 18866–18875.
79 W. Jiang, Y. Deng, Z. Song, Y. Xie, L. Gong, Y. Chen and H. Kuang, Gestational Perfluorooctanoic Acid Exposure Inhibits Placental Development by Dysregulation of Labyrinth Vessels and uNK Cells and Apoptosis in Mice., *Frontiers in physiology*, 2020, **11**, 51.
80 Y. Zhang, L. Zhang, J. Bao, L. Liu and X. Wang, Perfluorooctanoic acid exposure in early pregnancy induces oxidative stress in mice uterus and liver., *Environmental science and pollution research international*, 2021, **28**, 66355–66365.
81 Y. Zhang, Y. Li, N. Gao, Y. Gong, W. Shi and X. Wang, Transcriptome and Metabolome Analyses Reveal Perfluorooctanoic Acid-Induced Kidney Injury by Interfering with PPAR Signaling Pathway., *International journal of molecular sciences*, DOI:10.3390/ijms241411503.
82 A. Salimi, M. Nikoosiar Jahromi and J. Pourahmad, Maternal exposure causes mitochondrial dysfunction in brain, liver, and heart of mouse fetus: An explanation for perfluorooctanoic acid induced abortion and developmental toxicity., *Environmental toxicology*, 2019, **34**, 878–885.
83 C. Huang, D. Wu, K. Zhang, F. A. Khan, N. S. Pandupuspitasari, Y. Wang, L. Huo and F. Sun, Perfluorooctanoic acid alters the developmental trajectory of female germ cells and embryos in rodents and its potential mechanism., *Ecotoxicology and environmental safety*, 2022, **236**, 113467.

84 M. G. Pouwer, E. J. Pieterman, S.-C. Chang, G. W. Olsen, M. P. M. Caspers, L. Verschuren, J. W. Jukema and H. M. G. Princen, Dose Effects of Ammonium Perfluorooctanoate on Lipoprotein Metabolism in APOE*3-Leiden.CETP Mice., *Toxicological sciences : an official journal of the Society of Toxicology*, 2019, **168**, 519–534.
85 J. J. Schlezinger, H. Puckett, J. Oliver, G. Nielsen, W. Heiger-Bernays and T. F. Webster, Perfluorooctanoic acid activates multiple nuclear receptor pathways and skews expression of genes regulating cholesterol homeostasis in liver of humanized PPARα mice fed an American diet., *Toxicology and applied pharmacology*, 2020, **405**, 115204.
86 S. Su, L. J. Billy, S. Chang, F. J. Gonzalez, A. D. Patterson and J. M. Peters, The role of mouse and human peroxisome proliferator-activated receptor-α in modulating the hepatic effects of perfluorooctane sulfonate in mice., *Toxicology*, 2022, **465**, 153056.
87 A. M. Narizzano, E. M. Lent, J. M. Hanson, A. G. East, M. E. Bohannon and M. J. J. Quinn, Reproductive and developmental toxicity of perfluorooctane sulfonate (PFOS) in the white-footed mouse (Peromyscus leucopus)., *Reproductive toxicology (Elmsford, N.Y.)*, 2022, **113**, 120–127.
88 A. M. Narizzano, M. E. Bohannon, A. G. East, B. A. Guigni and M. J. J. Quinn, Reproductive and immune effects emerge at similar thresholds of PFHxS in deer mice., *Reproductive toxicology (Elmsford, N.Y.)*, 2023, **120**, 108421.
89 Y. Qiu, M. Gao, T. Cao, J. Wang, M. Luo, S. Liu, X. Zeng and J. Huang, PFOS and F-53B disrupted inner cell mass development in mouse preimplantation embryo., *Chemosphere*, 2024, **349**, 140948.
90 J. E. Mylroie, M. S. Wilbanks, A. N. Kimble, K. T. To, C. S. Cox, S. J. McLeod, K. A. Gust, D. W. Moore, E. J. Perkins and N. Garcia-Reyero, Perfluorooctanesulfonic Acid–Induced Toxicity on Zebrafish Embryos in the Presence or Absence of the Chorion, *Environmental Toxicology and Chemistry*, 2021, **40**, 780–791.
91 H. Lee, C. M. Tran, S. Jeong, S. S. Kim, M. A. Bae and K.-T. Kim, Seizurogenic effect of perfluorooctane sulfonate in zebrafish larvae., *Neurotoxicology*, 2022, **93**, 257–264.
92 A. Mahapatra, P. Gupta, A. Suman, S. S. Ray, G. Malafaia and R. K. Singh, Unraveling the mechanisms of perfluorooctanesulfonic acid-induced dopaminergic neurotoxicity and microglial activation in developing zebrafish., *The Science of the total environment*, 2023, **887**, 164030.
93 Z. Yang, L. Fu, M. Cao, F. Li, J. Li, Z. Chen, A. Guo, H. Zhong, W. Li, Y. Liang and Q. Luo, PFAS-induced lipidomic dysregulations and their associations with developmental toxicity in zebrafish embryos., *The Science of the total environment*, 2023, **861**, 160691.
94 E. K. Min, H. Lee, E. J. Sung, S. W. Seo, M. Song, S. Wang, S. S. Kim, M. A. Bae, T.-Y. Kim, S. Lee and K.-T. Kim, Integrative multi-omics reveals analogous developmental neurotoxicity mechanisms between perfluorobutanesulfonic acid and perfluorooctanesulfonic acid in zebrafish., *Journal of hazardous materials*, 2023, **457**, 131714.
95 H. Chen, W. Qiu, X. Yang, F. Chen, J. Chen, L. Tang, H. Zhong, J. T. Magnuson, C. Zheng and E. G. Xu, Perfluorooctane Sulfonamide (PFOSA) Induces Cardiotoxicity

via Aryl Hydrocarbon Receptor Activation in Zebrafish., *Environmental science & technology*, 2022, **56**, 8438–8448.
96 O. Wasel, K. M. Thompson and J. L. Freeman, Assessment of unique behavioral, morphological, and molecular alterations in the comparative developmental toxicity profiles of PFOA, PFHxA, and PFBA using the zebrafish model system, *Environ. Int.*, 2022, **170**, 107642.
97 S. Zhang, X. Guo, S. Lu, J. He, Q. Wu, X. Liu, Z. Han and P. Xie, Perfluorohexanoic acid caused disruption of the hypothalamus-pituitary-thyroid axis in zebrafish larvae, *Ecotoxicology and Environmental Safety*, DOI:10.1016/j.ecoenv.2022.113283.
98 M. Xu, J. Legradi and P. Leonards, Using comprehensive lipid profiling to study effects of PFHxS during different stages of early zebrafish development., *The Science of the total environment*, 2022, **808**, 151739.
99 O. Wasel, H. King, Y. J. Choi, L. S. Lee and J. L. Freeman, Differential Developmental Neurotoxicity and Tissue Uptake of the Per- and Polyfluoroalkyl Substance Alternatives, GenX and PFBS., *Environmental science & technology*, 2023, **57**, 19274–19284.
100 S. Gong, F. McLamb, D. Shea, J. P. Vu, M. F. Vasquez, Z. Feng, K. Bozinovic, K. K. Hirata, R. M. Gersberg and G. Bozinovic, Toxicity assessment of hexafluoropropylene oxide-dimer acid on morphology, heart physiology, and gene expression during zebrafish (Danio rerio) development., *Environmental science and pollution research international*, 2023, **30**, 32320–32336.
101 S. Sun, X. Li, L. Zhang, Z. Zhong, C. Chen, Y. Zuo, Y. Chen, H. Hu, F. Liu, G. Xiong, H. Lu, J. Chen and J. Dai, Hexafluoropropylene oxide trimer acid (HFPO-TA) disturbs embryonic liver and biliary system development in zebrafish., *The Science of the total environment*, 2023, **859**, 160087.
102 S. Sun, J. Wang, J. Yao, H. Guo and J. Dai, Transcriptome analysis of 3D primary mouse liver spheroids shows that long-term exposure to hexafluoropropylene oxide trimer acid disrupts hepatic bile acid metabolism., *The Science of the total environment*, 2022, **812**, 151509.
103 Y. Wu, J. Huang, M. Deng, Y. Jin, H. Yang, Y. Liu, Q. Cao, J. A. Mennigen and W. Tu, Acute exposure to environmentally relevant concentrations of Chinese PFOS alternative F-53B induces oxidative stress in early developing zebrafish, *Chemosphere*, 2019, **235**, 945–951.
104 W. Gui, H. Guo, X. Chen, J. Wang, Y. Guo, H. Zhang, X. Zhou, Y. Zhao and J. Dai, Emerging polyfluorinated compound Nafion by-product 2 disturbs intestinal homeostasis in zebrafish (Danio rerio)., *Ecotoxicology and environmental safety*, 2023, **249**, 114368.
105 W. Gui, H. Guo, C. Wang, M. Li, Y. Jin, K. Zhang, J. Dai and Y. Zhao, Comparative developmental toxicities of zebrafish towards structurally diverse per- and polyfluoroalkyl substances., *The Science of the total environment*, 2023, **902**, 166569.
106 J. Wang, G. Shi, J. Yao, N. Sheng, R. Cui, Z. Su, Y. Guo and J. Dai, Perfluoropolyether carboxylic acids (novel alternatives to PFOA) impair zebrafish posterior swim bladder development via thyroid hormone disruption., *Environment international*, 2020, **134**, 105317.

107 L. Wang, S. Chen, H. Hou, M. Cao, Z. Zhou, H. Cao, M. Chen, Y. Liang and Y. Wang, Early Stage Exposure of 1H,1H,2H,2H-Perfluorooctanesulfonate-Induced Cardiovascular Abnormality in Zebrafish Embryos and Larvae, *ACS ES and T Water*, 2023, **3**, 106–117.
108 L. Wu, J. Gu, X. Duan, F. Ge, H. Ye, L. Kong, W. Liu, R. Gao, J. Jiao, H. Chen and G. Ji, Insight into the mechanisms of neuroendocrine toxicity induced by 6:2FTCA via thyroid hormone disruption., *Chemosphere*, 2023, **341**, 140031.
109 J. Zhang, Z. Ren and M. Chen, Immunotoxicity and Transcriptome Analyses of Zebrafish (Danio rerio) Embryos Exposed to 6:2 FTSA., *Toxics*, DOI:10.3390/toxics11050459.
110 S. Kim, K. M. Stroski, G. Killeen, C. Smitherman, M. F. Simcik and B. W. Brooks, 8:8 Perfluoroalkyl phosphinic acid affects neurobehavioral development, thyroid disruption, and DNA methylation in developing zebrafish, *Sci. Total Environ.*, 2020, **736**, 139600.
111 H. Mahoney, J. Cantin, J. Rybchuk, Y. Xie, J. P. Giesy and M. Brinkmann, Acute exposure of zebrafish (Danio rerio) to the next-generation perfluoroalkyl substance, perfluoroethylcyclohexanesulfonate, shows similar effects as legacy substances, *Environ. Sci. Technol.*, 2023, **57**, 4199–4207.
112 K. M. Annunziato, J. Doherty, J. Lee, J. M. Clark, W. Liang, C. W. Clark, M. Nguyen, M. A. Roy and A. R. Timme-Laragy, Chemical Characterization of a Legacy Aqueous Film-Forming Foam Sample and Developmental Toxicity in Zebrafish (Danio rerio)., *Environmental health perspectives*, 2020, **128**, 97006.
113 F. Menger, J. Pohl, L. Ahrens, G. Carlsson and S. Örn, Behavioural effects and bioconcentration of per- and polyfluoroalkyl substances (PFASs) in zebrafish (Danio rerio) embryos., *Chemosphere*, 2020, **245**, 125573.
114 S. Dasgupta, A. Reddam, Z. Liu, J. Liu and D. C. Volz, High-content screening in zebrafish identifies perfluorooctanesulfonamide as a potent developmental toxicant., *Environmental pollution (Barking, Essex : 1987)*, 2020, **256**, 113550.
115 L. Truong, Y. Rericha, P. Thunga, S. Marvel, D. Wallis, M. T. Simonich, J. A. Field, D. Cao, D. M. Reif and R. L. Tanguay, Systematic developmental toxicity assessment of a structurally diverse library of PFAS in zebrafish., *Journal of hazardous materials*, 2022, **431**, 128615.
116 K. E. Sant, K. Annunziato, S. Conlin, G. Teicher, P. Chen, O. Venezia, G. B. Downes, Y. Park and A. R. Timme-Laragy, Developmental exposures to perfluorooctanesulfonic acid (PFOS) impact embryonic nutrition, pancreatic morphology, and adiposity in the zebrafish, Danio rerio., *Environmental pollution (Barking, Essex : 1987)*, 2021, **275**, 116644.
117 L. Wu, Y. Dang, L.-X. Liang, Y.-C. Gong, M. Zeeshan, Z. Qian, S. D. Geiger, M. G. Vaughn, Y. Zhou, Q.-Q. Li, C. Chu, Y.-W. Tan, L.-Z. Lin, R.-Q. Liu, L.-W. Hu, B.-Y. Yang, X.-W. Zeng, Y. Yu and G.-H. Dong, Perfluorooctane sulfonates induces neurobehavioral changes and increases dopamine neurotransmitter levels in zebrafish larvae., *Chemosphere*, 2022, **297**, 134234.
118 A. Haimbaugh, C.-C. Wu, C. Akemann, D. N. Meyer, M. Connell, M. Abdi, A. Khalaf, D. Johnson and T. R. Baker, Multi-and Transgenerational Effects of Developmental Exposure to Environmental Levels of PFAS and PFAS Mixture in Zebrafish (Danio rerio), *Toxics*, DOI:10.3390/toxics10060334.

119 A. B. Hawkey, M. Mead, S. Natarajan, A. Gondal, O. Jarrett and E. D. Levin, Embryonic exposure to PFAS causes long-term, compound-specific behavioral alterations in zebrafish., *Neurotoxicology and teratology*, 2023, **97**, 107165.
120 S. E. Paquette, N. R. Martin, A. Rodd, K. E. Manz, E. Allen, M. Camarillo, H. I. Weller, K. Pennell and J. S. Plavicki, Evaluation of Neural Regulation and Microglial Responses to Brain Injury in Larval Zebrafish Exposed to Perfluorooctane Sulfonate., *Environmental health perspectives*, 2023, **131**, 117008.
121 J. Huang, L. Sun, J. A. Mennigen, Y. Liu, S. Liu, M. Zhang, Q. Wang and W. Tu, Developmental toxicity of the novel PFOS alternative OBS in developing zebrafish: An emphasis on cilia disruption., *Journal of hazardous materials*, 2021, **409**, 124491.
122 J. Huang, Q. Wang, S. Liu, H. Lai and W. Tu, Comparative chronic toxicities of PFOS and its novel alternatives on the immune system associated with intestinal microbiota dysbiosis in adult zebrafish., *Journal of hazardous materials*, 2022, **425**, 127950.
123 Q. Wang, Y. Liu, M. Zhang, S. Liu, N. Wan, M. Li and W. Tu, Novel PFOS alternative OBS inhibits body growth of developing zebrafish by triggering thyroid function disorder and osteoclast differentiation., *Chemosphere*, 2023, **341**, 140068.
124 K. E. Sant, O. L. Venezia, P. P. Sinno and A. R. Timme-Laragy, Perfluorobutanesulfonic Acid Disrupts Pancreatic Organogenesis and Regulation of Lipid Metabolism in the Zebrafish, Danio rerio., *Toxicological sciences : an official journal of the Society of Toxicology*, 2019, **167**, 258–268.
125 S. Liu, W. Qiu, R. Li, B. Chen, X. Wu, J. T. Magnuson, B. Xu, S. Luo, E. G. Xu and C. Zheng, Perfluorononanoic Acid Induces Neurotoxicity via Synaptogenesis Signaling in Zebrafish., *Environmental science & technology*, 2023, **57**, 3783–3793.
126 L. Tang, W. Qiu, S. Zhang, J. Wang, X. Yang, B. Xu, J. T. Magnuson, E. G. Xu, M. Wu and C. Zheng, Poly- and Perfluoroalkyl Substances Induce Immunotoxicity via the TLR Pathway in Zebrafish: Links to Carbon Chain Length., *Environmental science & technology*, 2023, **57**, 6139–6149.
127 K. M. Annunziato, C. E. Jantzen, M. C. Gronske and K. R. Cooper, Subtle morphometric, behavioral and gene expression effects in larval zebrafish exposed to PFHxA, PFHxS and 6:2 FTOH., *Aquatic toxicology (Amsterdam, Netherlands)*, 2019, **208**, 126–137.
128 Z. S. Ulhaq and W. K. F. Tse, Perfluorohexanesulfonic acid (PFHxS) induces oxidative stress and causes developmental toxicities in zebrafish embryos., *Journal of hazardous materials*, 2023, **457**, 131722.
129 S. Liu, H. Lai, Q. Wang, R. Martínez, M. Zhang, Y. Liu, J. Huang, M. Deng and W. Tu, Immunotoxicity of F53B, an alternative to PFOS, on zebrafish (Danio rerio) at different early life stages., *The Science of the total environment*, 2021, **790**, 148165.
130 S. Yi, P. Chen, L. Yang and L. Zhu, Probing the hepatotoxicity mechanisms of novel chlorinated polyfluoroalkyl sulfonates to zebrafish larvae: Implication of structural specificity., *Environment international*, 2019, **133**, 105262.
131 S. Yi, J. Wang, R. Wang, M. Liu, W. Zhong, L. Zhu and G. Jiang, Structure-Related Thyroid Disrupting Effect of Perfluorooctanesulfonate-like Substances in Zebrafish Larvae., *Environmental science & technology*, 2024, **58**, 182–193.
132 S. Gaballah, A. Swank, J. R. Sobus, X. M. Howey, J. Schmid, T. Catron, J. McCord, E. Hines, M. Strynar and T. Tal, Evaluation of Developmental Toxicity,

Developmental Neurotoxicity, and Tissue Dose in Zebrafish Exposed to GenX and Other PFAS., *Environmental health perspectives*, 2020, **128**, 47005.
133 Y. Rericha, D. Cao, L. Truong, M. T. Simonich, J. A. Field and R. L. Tanguay, Sulfonamide functional head on short-chain perfluorinated substance drives developmental toxicity., *iScience*, 2022, **25**, 103789.
134 W. Sun, X. Zhang, Y. Qiao, N. Griffin, H. Zhang, L. Wang and H. Liu, Exposure to PFOA and its novel analogs disrupts lipid metabolism in zebrafish., *Ecotoxicology and environmental safety*, 2023, **259**, 115020.
135 Y. Xin, B. Wan, B. Yu, Y. Fan, D. Chen and L.-H. Guo, Chlorinated Polyfluoroalkylether Sulfonic Acids Exhibit Stronger Estrogenic Effects than Perfluorooctane Sulfonate by Activating Nuclear Estrogen Receptor Pathways, *Environmental Science & Technology*, 2020, **54**, 3455–3464.
136 Q. Wang, J. Huang, S. Liu, C. Wang, Y. Jin, H. Lai and W. Tu, Aberrant hepatic lipid metabolism associated with gut microbiota dysbiosis triggers hepatotoxicity of novel PFOS alternatives in adult zebrafish, *Environ. Int.*, 2022, **166**, 107351.
137 J. Guo, P. Wu, J. Cao, Y. Luo, J. Chen, G. Wang, W. Guo, T. Wang and X. He, The PFOS disturbed immunomodulatory functions via nuclear Factor-κB signaling in liver of zebrafish (Danio rerio)., *Fish & shellfish immunology*, 2019, **91**, 87–98.
138 M. Bao, S. Zheng, C. Liu, W. Huang, J. Xiao and K. Wu, Perfluorooctane sulfonate exposure alters sexual behaviors and transcriptions of genes in hypothalamic–pituitary–gonadal–liver axis of male zebrafish (Danio rerio), *Environmental Pollution*, DOI:10.1016/j.envpol.2020.115585.
139 K. A. Gust, J. E. Mylroie, A. N. Kimble, M. S. Wilbanks, C. S. C. Steward, K. A. Chapman, K. M. Jensen, A. J. Kennedy, P. M. Krupa, S. A. Waisner, Z. Pandelides, N. Garcia-Reyero, R. J. Erickson, G. T. Ankley, J. Conder and D. W. Moore, Survival, Growth, and Reproduction Responses in a Three-Generation Exposure of the Zebrafish (Danio rerio) to Perfluorooctane Sulfonate., *Environmental toxicology and chemistry*, 2024, **43**, 115–131.
140 H. Zhang, L. Han, L. Qiu, B. Zhao, Y. Gao, Z. Chu and X. Dai, Perfluorooctanoic Acid (PFOA) Exposure Compromises Fertility by Affecting Ovarian and Oocyte Development., *International journal of molecular sciences*, DOI:10.3390/ijms25010136.
141 M. Khazaee, M. G. E. Guardian, D. S. Aga and C. A. Ng, Impacts of Sex and Exposure Duration on Gene Expression in Zebrafish Following Perfluorooctane Sulfonate Exposure., *Environmental toxicology and chemistry*, 2020, **39**, 437–449.
142 H. Zhang, L. Shen, W. Fang, X. Zhang and Y. Zhong, Perfluorooctanoic acid-induced immunotoxicity via NF-kappa B pathway in zebrafish (Danio rerio) kidney., *Fish & shellfish immunology*, 2021, **113**, 9–19.
143 Y. Wang, X. Chen, B. Wang, G. Lu, J. Liu, D. Wu and Z. Yan, Toxicity comparison of perfluorooctanoic acid (PFOA), hexafluoropropylene oxide dimer acid (HFPO-DA), and hexafluoropropylene oxide trimer acid (HFPO-TA) in zebrafish gut., *Aquatic toxicology (Amsterdam, Netherlands)*, 2023, **262**, 106655.
144 S. Dong, J. Xu, D. Yang, X. Zhao, X. Li, D. Chen, J. Xing, Y. Shi, Y. Sun and G. Ding, Different Life-Stage Exposure to Hexafluoropropylene Oxide Trimer Acid Induces Reproductive Toxicity in Adult Zebrafish (Danio rerio)., *Environmental toxicology and chemistry*, 2023, **42**, 2490–2500.

145 G. Shi, Q. Cui, J. Wang, H. Guo, Y. Pan, N. Sheng, Y. Guo and J. Dai, Chronic exposure to 6:2 chlorinated polyfluorinated ether sulfonate acid (F-53B) induced hepatotoxic effects in adult zebrafish and disrupted the PPAR signaling pathway in their offspring., *Environmental pollution (Barking, Essex : 1987)*, 2019, **249**, 550–559.
146 Y. Zhu, D. Yang, X. Duan, Y. Zhang, D. Chen, Z. Gong and C. Liu, Perfluorooctane sulfonate promotes doxycycline-induced liver tumor progression in male Kras(v12) transgenic zebrafish., *Environmental research*, 2021, **196**, 110962.
147 K. M. Annunziato, M. Marin, W. Liang, S. M. Conlin, W. Qi, J. Doherty, J. Lee, J. M. Clark, Y. Park and A. R. Timme-Laragy, The Nrf2a pathway impacts zebrafish offspring development with maternal preconception exposure to perfluorobutanesulfonic acid., *Chemosphere*, 2022, **287**, 132121.
148 S. R. Sammi, R. M. Foguth, C. S. Nieves, C. De Perre, P. Wipf, C. T. McMurray, L. S. Lee and J. R. Cannon, Perfluorooctane Sulfonate (PFOS) Produces Dopaminergic Neuropathology in Caenorhabditis elegans., *Toxicological sciences : an official journal of the Society of Toxicology*, 2019, **172**, 417–434.
149 C. Wei, Z. Zhou, L. Wang, Z. Huang, Y. Liang and J. Zhang, Perfluorooctane sulfonate (PFOS) disturbs fatty acid metabolism in Caenorhabditis elegans: Evidence from chemical analysis and molecular mechanism exploration., *Chemosphere*, 2021, **277**, 130359.
150 M. I. Chowdhury, T. Sana, L. Panneerselvan, A. K. Sivaram and M. Megharaj, Perfluorooctane sulfonate (PFOS) induces several behavioural defects in Caenorhabditis elegans that can also be transferred to the next generations., *Chemosphere*, 2022, **291**, 132896.
151 H. M. Kim, N. P. Long, S. J. Yoon, N. H. Anh, S. J. Kim, J. H. Park and S. W. Kwon, Omics approach reveals perturbation of metabolism and phenotype in Caenorhabditis elegans triggered by perfluorinated compounds., *The Science of the total environment*, 2020, **703**, 135500.
152 T.-A. Lin, C.-W. Huang and C.-C. Wei, Early-life perfluorooctanoic acid (PFOA) and perfluorooctane sulfonic acid (PFOS) exposure cause obesity by disrupting fatty acids metabolism and enhancing triglyceride synthesis in Caenorhabditis elegans., *Aquatic toxicology (Amsterdam, Netherlands)*, 2022, **251**, 106274.
153 T. Sana, M. I. Chowdhury, P. Logeshwaran, R. Dharmarajan and M. Megharaj, Perfluorooctanoic acid (PFOA) induces behavioural, reproductive and developmental toxicological impacts in Caenorhabditis elegans at concentrations relevant to the contaminated areas, *Environmental Advances*, DOI:10.1016/j.envadv.2021.100053.
154 Y. Yue, S. Li, Z. Qian, R. F. Pereira, J. Lee, J. J. Doherty, Z. Zhang, Y. Peng, J. M. Clark, A. R. Timme-Laragy and Y. Park, Perfluorooctanesulfonic acid (PFOS) and perfluorobutanesulfonic acid (PFBS) impaired reproduction and altered offspring physiological functions in Caenorhabditis elegans., *Food and chemical toxicology : an international journal published for the British Industrial Biological Research Association*, 2020, **145**, 111695.
155 M. I. Chowdhury, T. Sana, L. Panneerselvan, R. Dharmarajan and M. Megharaj, Acute toxicity and transgenerational effects of perfluorobutane sulfonate on Caenorhabditis elegans, *Environ. Toxicol. Chem.*, 2021, **40**, 1973–1982.

156 J. C. K. Mangu, M. Stylianou, P.-E. Olsson and J. Jass, Per- and polyfluoroalkyl substances enhance Staphylococcus aureus pathogenicity and impair host immune response., *Environmental pollution (Barking, Essex : 1987)*, 2022, **314**, 120294.
157 S. D. Currie, J. P. Doherty, K. S. Xue, J.-S. Wang and L. Tang, The stage-specific toxicity of per- and polyfluoroalkyl substances (PFAS) in nematode Caenorhabditis elegans., *Environmental pollution (Barking, Essex : 1987)*, 2023, **336**, 122429.
158 H. Ni, J. Yuan, J. Ji, Y. Guo, S. Zhong, Y. Lin, Y. Zheng and Q. Jiang, Long term toxicities following developmental exposure to perfluorooctanoic acid: Roles of peroxisome proliferation activated receptor alpha., *Environmental pollution (Barking, Essex : 1987)*, 2023, **317**, 120722.
159 Y. Guo, J. Yuan, H. Ni, J. Ji, S. Zhong, Y. Zheng and Q. Jiang, Perfluorooctanoic acid-induced developmental cardiotoxicity in chicken embryo: Roles of miR-490-5p., *Environmental pollution (Barking, Essex : 1987)*, 2022, **312**, 120022.
160 N. Lv, J. Yuan, A. Ji, L. Shi, M. Gao, L. Cui and Q. Jiang, Perfluorooctanoic acid-induced toxicities in chicken embryo primary cardiomyocytes: Roles of PPAR alpha and Wnt5a/Frizzled2., *Toxicology and applied pharmacology*, 2019, **381**, 114716.
161 X. Xu, H. Ni, Y. Guo, Y. Lin, J. Ji, C. Jin, F. Yuan, M. Feng, N. Ji, Y. Zheng and Q. Jiang, Hexafluoropropylene oxide dimer acid (HFPO-DA) induced developmental cardiotoxicity and hepatotoxicity in hatchling chickens: Roles of peroxisome proliferator activated receptor alpha., *Environmental pollution (Barking, Essex : 1987)*, 2021, **290**, 118112.
162 A. F. Ojo, C. Peng and J. C. Ng, Combined effects and toxicological interactions of perfluoroalkyl and polyfluoroalkyl substances mixtures in human liver cells (HepG2)., *Environmental pollution (Barking, Essex : 1987)*, 2020, **263**, 114182.
163 Y. Wen, N. Mirji and J. Irudayaraj, Epigenetic toxicity of PFOA and GenX in HepG2 cells and their role in lipid metabolism., *Toxicology in vitro : an international journal published in association with BIBRA*, 2020, **65**, 104797.
164 R. Zhang, B. Chen, L. Lin, H. Zhang and T. Luan, 13C isotope-based metabolic flux analysis revealing cellular landscape of glucose metabolism in human liver cells exposed to perfluorooctanoic acid, *Sci. Total Environ.*, 2021, **770**, 145329.
165 Z. Dong, T. Qiu, J. Zhang, S. Sha, X. Han, J. Kang, X. Shi, X. Sun, L. Jiang, G. Yang, X. Yao and Y. Ma, Perfluorooctane sulfonate induces autophagy-dependent lysosomal membrane permeabilization by weakened interaction between tyrosinated alpha-tubulin and spinster 1, *Food and Chemical Toxicology*, DOI:10.1016/j.fct.2021.112540.
166 L. De Toni, A. Di Nisio, M. S. Rocca, D. Guidolin, A. Della Marina, L. Bertazza, S. Sut, E. Purpura, M. Pannella, A. Garolla and C. Foresta, Exposure to Perfluoro-Octanoic Acid Associated With Upstream Uncoupling of the Insulin Signaling in Human Hepatocyte Cell Line, *Frontiers in Endocrinology*, 2021, **12**, 632927.
167 A. F. Ojo, Q. Xia, C. Peng and J. C. Ng, Evaluation of the individual and combined toxicity of perfluoroalkyl substances to human liver cells using biomarkers of oxidative stress., *Chemosphere*, 2021, **281**, 130808.
168 K. A. Houck, G. Patlewicz, A. M. Richard, A. J. Williams, M. A. Shobair, M. Smeltz, M. S. Clifton, B. Wetmore, A. Medvedev and S. Makarov, Bioactivity profiling of per- and polyfluoroalkyl substances (PFAS) identifies potential toxicity pathways related to molecular structure., *Toxicology*, 2021, **457**, 152789.

169 M. Abudayyak, E. Öztaş and G. Özhan, Determination of Perflourooctanoic Acid Toxicity in a Human Hepatocarcinoma Cell Line., *Journal of health & pollution*, 2021, **11**, 210909.
170 A. F. Ojo, C. Peng and J. C. Ng, Genotoxicity assessment of per- and polyfluoroalkyl substances mixtures in human liver cells (HepG2)., *Toxicology*, 2022, **482**, 153359.
171 S. Goines and J. E. Dick, Investigating the cytotoxic redox mechanism of PFOS within Hep G2 by hyperspectral-assisted scanning electrochemical microscopy., *The Analyst*, 2022, **147**, 4356–4364.
172 A. F. Ojo, C. Peng and J. C. Ng, Combined effects of mixed per- and polyfluoroalkyl substances on the Nrf2-ARE pathway in ARE reporter-HepG2 cells., *Journal of hazardous materials*, 2022, **421**, 126827.
173 M. C. Magnifico, M. Xhani, B. Sprovera, B. Buttari, G. Abballe, F. Desideri, E. Panieri, L. Saso and M. Arese, Nitro-Oxidative Stress and Mitochondrial Dysfunction in Human Cell Lines Exposed to the Environmental Contaminants PFOA and BPA., *Frontiers in bioscience (Landmark edition)*, 2022, **27**, 292.
174 Q. Qi, S. Niture, S. Gadi, E. Arthur, J. Moore, K. E. Levine and D. Kumar, Per- and polyfluoroalkyl substances activate UPR pathway, induce steatosis and fibrosis in liver cells., *Environmental toxicology*, 2023, **38**, 225–242.
175 J. Louisse, D. Rijkers, G. Stoopen, A. Janssen, M. Staats, R. Hoogenboom, S. Kersten and A. Peijnenburg, Perfluorooctanoic acid (PFOA), perfluorooctane sulfonic acid (PFOS), and perfluorononanoic acid (PFNA) increase triglyceride levels and decrease cholesterogenic gene expression in human HepaRG liver cells., *Archives of toxicology*, 2020, **94**, 3137–3155.
176 A.-C. Behr, A. Kwiatkowski, M. Ståhlman, F. F. Schmidt, C. Luckert, A. Braeuning and T. Buhrke, Impairment of bile acid metabolism by perfluorooctanoic acid (PFOA) and perfluorooctanesulfonic acid (PFOS) in human HepaRG hepatoma cells., *Archives of toxicology*, 2020, **94**, 1673–1686.
177 F. Sadrabadi, J. Alarcan, H. Sprenger, A. Braeuning and T. Buhrke, Impact of perfluoroalkyl substances (PFAS) and PFAS mixtures on lipid metabolism in differentiated HepaRG cells as a model for human hepatocytes., *Archives of toxicology*, 2024, **98**, 507–524.
178 D. Lv, Y. Gu, M. Guo, P. Hou, Y. Li and R. Wu, Perfluorooctanoic acid exposure induces apoptosis in SMMC-7721 hepatocellular cancer cells., *Environmental pollution (Barking, Essex : 1987)*, 2019, **247**, 509–514.
179 K. H. Sim, H. S. Oh, C. Lee, H. Eun and Y. J. Lee, Evaluation of the Effect of Perfluorohexane Sulfonate on the Proliferation of Human Liver Cells., *International journal of environmental research and public health*, DOI:10.3390/ijerph20196868.
180 C.-H. Li, X.-M. Ren and L.-H. Guo, Adipogenic activity of oligomeric hexafluoropropylene oxide (perfluorooctanoic acid alternative) through peroxisome proliferator-activated receptor γ pathway, *Environ. Sci. Technol.*, 2019, **53**, 3287–3295.
181 M. Camdzic, D. S. Aga and G. E. Atilla-Gokcumen, Cellular Interactions and Fatty Acid Transporter CD36-Mediated Uptake of Per- and Polyfluorinated Alkyl Substances (PFAS)., *Chemical research in toxicology*, 2022, **35**, 694–702.
182 C. Li, L. Jiang, Y. Jin, D. Zhang, J. Chen, Y. Qi, R. Fan, J. Luo, L. Xu, W. Ma, K. Zhao and D. Yu, Lipid metabolism disorders effects of 6:2 chlorinated polyfluorinated

ether sulfonate through Hsa-miRNA-532-3p/Acyl-CoA oxidase 1(ACOX1) pathway., *Ecotoxicology and environmental safety*, 2021, **228**, 113011.
183 T. E. Stoker, J. Wang, A. S. Murr, J. R. Bailey and A. R. Buckalew, High-Throughput Screening of ToxCast PFAS Chemical Library for Potential Inhibitors of the Human Sodium Iodide Symporter., *Chemical research in toxicology*, 2023, **36**, 380–389.
184 P. Wang, D. Liu, S. Yan, Y. Liang, J. Cui, L. Guo, S. Ren and P. Chen, The Role of Ferroptosis in the Damage of Human Proximal Tubule Epithelial Cells Caused by Perfluorooctane Sulfonate., *Toxics*, DOI:10.3390/toxics10080436.
185 L.-L. Wen, Y.-T. Chen, Y.-C. G. Lee, T.-L. Ko, H.-C. Chou and S.-H. Juan, Perfluorooctane sulfonate induces autophagy-associated apoptosis through oxidative stress and the activation of extracellular signal-regulated kinases in renal tubular cells., *PloS one*, 2021, **16**, e0245442.
186 B.-S. Lee, Y. Kim, H. Park, W.-J. Im, H.-Y. Han, Y.-B. Kim, S. Lim and M. H. Yoo, Long-chain perfluoroalkyl carboxylates induce cytoskeletal abnormalities and activate epithelial-mesenchymal transition in both renal cell carcinoma 3D cultures and Caki-1 xenografted mouse model., *Environment international*, 2023, **178**, 108093.
187 J. Li, X.-J. Quan, G. Chen, J.-W. Hong, Q. Wang, L.-L. Xu, B.-H. Wang, Z.-H. Yu and H.-M. Yu, PFOS-induced placental cell growth inhibition is partially mediated by lncRNA H19 through interacting with miR-19a and miR-19b., *Chemosphere*, 2020, **261**, 127640.
188 R. Sonkar, M. K. Kay and M. Choudhury, PFOS Modulates Interactive Epigenetic Regulation in First-Trimester Human Trophoblast Cell Line HTR-8/SV(neo)., *Chemical research in toxicology*, 2019, **32**, 2016–2027.
189 J. T. Szilagyi, A. N. Freedman, S. L. Kepper, A. M. Keshava, J. T. Bangma and R. C. Fry, Per- and Polyfluoroalkyl Substances Differentially Inhibit Placental Trophoblast Migration and Invasion In Vitro., *Toxicological sciences : an official journal of the Society of Toxicology*, 2020, **175**, 210–219.
190 Y. Du, Z. Cai, G. Zhou, W. Liang, Q. Man and W. Wang, Perfluorooctanoic acid exposure increases both proliferation and apoptosis of human placental trophoblast cells mediated by ER stress-induced ROS or UPR pathways., *Ecotoxicology and environmental safety*, 2022, **236**, 113508.
191 J. Bangma, J. Szilagyi, B. E. Blake, C. Plazas, S. Kepper, S. E. Fenton and R. C Fry, An assessment of serum-dependent impacts on intracellular accumulation and genomic response of per- and polyfluoroalkyl substances in a placental trophoblast model, *Environ. Toxicol.*, 2020, **35**, 1395–1405.
192 B. E. Blake, B. P. Rickard and S. E. Fenton, A high-throughput toxicity screen of 42 per- and polyfluoroalkyl substances (PFAS) and functional assessment of migration and gene expression in human placental trophoblast cells, *Front. Toxicol.*, 2022, **4**, 881347.
193 A. Pham, J. Zhang and L. Feng, Exposure to perfluorobutane sulfonate and perfluorooctanesulfonic acid disrupts the production of angiogenesis factors and stress responses in human placental syncytiotrophoblast., *Reproductive toxicology (Elmsford, N.Y.)*, 2020, **98**, 269–277.
194 H.-C. Zeng, B.-Q. Zhu, Y.-Q. Wang and Q.-Z. He, ROS-Triggered Autophagy Is Involved in PFOS-Induced Apoptosis of Human Embryo Liver L-02 Cells, *BioMed Research International*, DOI:10.1155/2021/6625952.

195 N. Davidsen, A. K. Rosenmai, K. Lauschke, T. Svingen and A. M. Vinggaard, Developmental effects of PFOS, PFOA and GenX in a 3D human induced pluripotent stem cell differentiation model., *Chemosphere*, 2021, **279**, 130624.
196 S. Liu, R. Yang, N. Yin and F. Faiola, Effects of per- and poly-fluorinated alkyl substances on pancreatic and endocrine differentiation of human pluripotent stem cells., *Chemosphere*, 2020, **254**, 126709.
197 A. Di Nisio, M. Pannella, S. Vogiatzis, S. Sut, S. Dall'Acqua, M. S. Rocca, A. Antonini, A. Porzionato, R. De Caro, M. Bortolozzi, L. D. Toni and C. Foresta, Impairment of human dopaminergic neurons at different developmental stages by perfluoro-octanoic acid (PFOA) and differential human brain areas accumulation of perfluoroalkyl chemicals., *Environment international*, 2022, **158**, 106982.
198 S. Liu, R. Yang, N. Yin, Y.-L. Wang and F. Faiola, Environmental and human relevant PFOS and PFOA doses alter human mesenchymal stem cell self-renewal, adipogenesis and osteogenesis., *Ecotoxicology and environmental safety*, 2019, **169**, 564–572.
199 Y. Gao, X. Guo, S. Wang, F. Chen, X. Ren, H. Xiao and L. Wang, Perfluorooctane sulfonate enhances mRNA expression of PPARγ and ap2 in human mesenchymal stem cells monitored by long-retained intracellular nanosensor., *Environmental pollution (Barking, Essex : 1987)*, 2020, **263**, 114571.
200 S. Liu, R. Yang, N. Yin and F. Faiola, The short-chain perfluorinated compounds PFBS, PFHxS, PFBA and PFHxA, disrupt human mesenchymal stem cell self-renewal and adipogenic differentiation., *Journal of environmental sciences (China)*, 2020, **88**, 187–199.
201 R. Yang, S. Liu, X. Liang, N. Yin, T. Ruan, L. Jiang and F. Faiola, F-53B and PFOS treatments skew human embryonic stem cell in vitro cardiac differentiation towards epicardial cells by partly disrupting the WNT signaling pathway., *Environmental pollution (Barking, Essex : 1987)*, 2020, **261**, 114153.
202 S. Liang, G. Liang, Y. Zhang, X. Zhang and L. Zhong, Profiling biotoxicities of hexafluoropropylene oxide trimer acid with human embryonic stem cell-based assays., *Journal of environmental sciences (China)*, 2022, **116**, 34–42.
203 P. Pierozan, D. Cattani and O. Karlsson, Perfluorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA) induce epigenetic alterations and promote human breast cell carcinogenesis in vitro., *Archives of toxicology*, 2020, **94**, 3893–3906.
204 P. Pierozan, D. Cattani and O. Karlsson, Tumorigenic activity of alternative per- and polyfluoroalkyl substances (PFAS): Mechanistic in vitro studies., *The Science of the total environment*, 2022, **808**, 151945.
205 P. Pierozan, M. Kosnik and O. Karlsson, High-content analysis shows synergistic effects of low perfluorooctanoic acid (PFOS) and perfluorooctane sulfonic acid (PFOA) mixture concentrations on human breast epithelial cell carcinogenesis., *Environment international*, 2023, **172**, 107746.
206 Y. Xin, X.-M. Ren, B. Wan and L.-H. Guo, Comparative in Vitro and in Vivo Evaluation of the Estrogenic Effect of Hexafluoropropylene Oxide Homologues., *Environmental science & technology*, 2019, **53**, 8371–8380.
207 W. Liu and J. Irudayaraj, Perfluorooctanoic acid (PFOA) exposure inhibits DNA methyltransferase activities and alters constitutive heterochromatin organization., *Food and chemical toxicology : an international journal published for the British Industrial Biological Research Association*, 2020, **141**, 111358.

208 G. Sakai, M. Hirao-Suzuki, T. Koga, T. Kobayashi, J. Kamishikiryo, M. Tanaka, K. Fujii, M. Takiguchi, N. Sugihara, A. Toda and S. Takeda, Perfluorooctanoic acid (PFOA) as a stimulator of estrogen receptor-negative breast cancer MDA-MB-231 cell aggressiveness: Evidence for involvement of fatty acid 2-hydroxylase (FA2H) in the stimulated cell migration., *The Journal of toxicological sciences*, 2022, **47**, 159–168.

209 A. Charazac, C. Hinault, B. Dolfi, S. Hautier, C. Decondé Le Butor, F. Bost and N. Chevalier, Low Doses of PFOA Promote Prostate and Breast Cancer Cells Growth through Different Pathways., *International journal of molecular sciences*, DOI:10.3390/ijms23147900.

210 N. Evans, J. M. Conley, M. Cardon, P. Hartig, E. Medlock-Kakaley and L. E. J. Gray, In vitro activity of a panel of per- and polyfluoroalkyl substances (PFAS), fatty acids, and pharmaceuticals in peroxisome proliferator-activated receptor (PPAR) alpha, PPAR gamma, and estrogen receptor assays., *Toxicology and applied pharmacology*, 2022, **449**, 116136.

211 Y. Zhou, H. Li, C. Lin, Y. Mao, J. Rao, Y. Lou, X. Yang, X. Xu and F. Jin, Perfluorooctanoic acid (PFOA) inhibits the gap junction intercellular communication and induces apoptosis in human ovarian granulosa cells., *Reproductive toxicology (Elmsford, N.Y.)*, 2020, **98**, 125–133.

212 K. L. Clark, J. W. George, G. Hua and J. S. Davis, Perfluorooctanoic acid promotes proliferation of the human granulosa cell line HGrC1 and alters expression of cell cycle genes and Hippo pathway effector YAP1., *Reproductive toxicology (Elmsford, N.Y.)*, 2022, **110**, 49–59.

213 B. P. Rickard, X. Tan, S. E. Fenton and I. Rizvi, Select Per- and Polyfluoroalkyl Substances (PFAS) Induce Resistance to Carboplatin in Ovarian Cancer Cell Lines., *International journal of molecular sciences*, DOI:10.3390/ijms23095176.

214 Y. Qin, X. Yuan, Z. Cui, W. Chen, S. Xu, K. Chen, F. Wang, F. Zheng, H. Ni, H.-M. Shen, Y. Wu and D. Xia, Low dose PFDA induces DNA damage and DNA repair inhibition by promoting nuclear cGAS accumulation in ovarian epithelial cells., *Ecotoxicology and environmental safety*, 2023, **265**, 115503.

215 Y.-D. Yang, J.-X. Li, N. Lu and R. Tian, Serum albumin mitigated perfluorooctane sulfonate-induced cytotoxicity by affecting the cellular responses., *Biophysical chemistry*, 2023, **302**, 107110.

216 A. Di Nisio, M. S. Rocca, I. Sabovic, M. De Rocco Ponce, C. Corsini, D. Guidolin, C. Zanon, L. Acquasaliente, A. R. Carosso, L. De Toni and C. Foresta, Perfluorooctanoic acid alters progesterone activity in human endometrial cells and induces reproductive alterations in young women., *Chemosphere*, 2020, **242**, 125208.

217 W.-Y. Hu, R. Lu, D. P. Hu, O. B. Imir, Q. Zuo, D. Moline, P. Afradiasbagharani, L. Liu, S. Lowe, L. Birch, D. J. V. Griend, Z. Madak-Erdogan and G. S. Prins, Per- and polyfluoroalkyl substances target and alter human prostate stem-progenitor cells., *Biochemical pharmacology*, 2022, **197**, 114902.

218 I. Šabović, I. Cosci, L. De Toni, A. Ferramosca, M. Stornaiuolo, A. Di Nisio, S. Dall'Acqua, A. Garolla and C. Foresta, Perfluoro-octanoic acid impairs sperm motility through the alteration of plasma membrane., *Journal of endocrinological investigation*, 2020, **43**, 641–652.

219 Y. Yuan, X. Ding, Y. Cheng, H. Kang, T. Luo, X. Zhang, H. Kuang, Y. Chen, X. Zeng and D. Zhang, PFOA evokes extracellular Ca(2+) influx and compromises progesterone-induced response in human sperm., *Chemosphere*, 2020, **241**, 125074.
220 F. Coperchini, L. Croce, P. Pignatti, G. Ricci, D. Gangemi, F. Magri, M. Imbriani, M. Rotondi and L. Chiovato, The new generation PFAS C6O4 does not produce adverse effects on thyroid cells in vitro., *Journal of endocrinological investigation*, 2021, **44**, 1625–1635.
221 M. Jabeen, M. Fayyaz and J. Irudayaraj, Epigenetic Modifications, and Alterations in Cell Cycle and Apoptosis Pathway in A549 Lung Carcinoma Cell Line upon Exposure to Perfluoroalkyl Substances., *Toxics*, DOI:10.3390/toxics8040112.
222 J. B. Sørli, M. Låg, L. Ekeren, J. Perez-Gil, L. S. Haug, E. Da Silva, M. N. Matrod, K. B. Gützkow and B. Lindeman, Per- and polyfluoroalkyl substances (PFASs) modify lung surfactant function and pro-inflammatory responses in human bronchial epithelial cells., *Toxicology in vitro : an international journal published in association with BIBRA*, 2020, **62**, 104656.
223 J. H. Lucas, Q. Wang and I. Rahman, Perfluorooctane Sulfonic Acid Disrupts Protective Tight Junction Proteins via Protein Kinase D in Airway Epithelial Cells., *Toxicological sciences : an official journal of the Society of Toxicology*, 2022, **190**, 215–226.
224 J. Dragon, M. Hoaglund, A. R. Badireddy, G. Nielsen, J. Schlezinger and A. Shukla, Perfluoroalkyl Substances (PFAS) Affect Inflammation in Lung Cells and Tissues., *International journal of molecular sciences*, DOI:10.3390/ijms24108539.
225 D. R. Robarts, K. K. Venneman, S. Gunewardena and U. Apte, GenX induces fibroinflammatory gene expression in primary human hepatocytes., *Toxicology*, 2022, **477**, 153259.
226 E. Marques, M. Pfohl, W. Wei, G. Tarantola, L. Ford, O. Amaeze, J. Alesio, S. Ryu, X. Jia, H. Zhu, G. D. Bothun and A. Slitt, Replacement per- and polyfluoroalkyl substances (PFAS) are potent modulators of lipogenic and drug metabolizing gene expression signatures in primary human hepatocytes., *Toxicology and applied pharmacology*, 2022, **442**, 115991.
227 W. Liu, H. Qin, Y. Pan, F. Luo and Z. Zhang, Low concentrations of perfluorooctane sulfonate repress osteogenic and enhance adipogenic differentiation of human mesenchymal stem cells., *Toxicology and applied pharmacology*, 2019, **367**, 82–91.
228 Y. Pan, H. Qin, W. Liu, Q. Zhang, L. Zheng, C. Zhou and X. Quan, Effects of chlorinated polyfluoroalkyl ether sulfonate in comparison with perfluoroalkyl acids on gene profiles and stemness in human mesenchymal stem cells., *Chemosphere*, 2019, **237**, 124402.
229 R. Li, C. Guo, X. Lin, T. F. Chan, K. P. Lai and J. Chen, Integrative omics analyses uncover the mechanism underlying the immunotoxicity of perfluorooctanesulfonate in human lymphocytes., *Chemosphere*, 2020, **256**, 127062.
230 A. Maddalon, A. Pierzchalski, T. Kretschmer, M. Bauer, A. C. Zenclussen, M. Marinovich, E. Corsini and G. Herberth, Mixtures of per- and poly-fluoroalkyl substances (PFAS) reduce the in vitro activation of human T cells and basophils., *Chemosphere*, 2023, **336**, 139204.

231 Y. Pan, H. Qin, L. Zheng, Y. Guo and W. Liu, Disturbance in transcriptomic profile, proliferation and multipotency in human mesenchymal stem cells caused by hexafluoropropylene oxides., *Environmental pollution (Barking, Essex : 1987)*, 2022, **292**, 118483.
232 A. W. F. Janssen, J. Louisse, D. Rijkers, N. E. T. Pinckaers, S. A. Hoekstra, R. L. A. P. Hoogenboom, A. A. C. M. Peijnenburg and K. Beekmann, Perfluoroalkyl substances (PFASs) decrease the expression of recombination-activating genes (RAG1 and RAG2) in human B lymphoma Namalwa cells., *Archives of toxicology*, 2022, **97**, 457–468.
233 F. Li, L. Chen, B.-X. Peng, Y. Lei, M. Li and L.-H. Guo, Perfluorooctane sulfonate promotes the migration of colorectal cancer cells by inducing epithelial-mesenchymal transition., *Journal of environmental sciences (China)*, 2024, **145**, 1–12.
234 R. Zhang, W. Lu, L. Tu, Y. Lin, J. Sun, B. Chen and T. Luan, Perfluorooctanoic acid-induced metabolic disorder via enhancing metabolism of glutamine and fatty acids in human intestinal cells, *Environmental pollution (Barking, Essex : 1987)*, 2023, **338**, 122684.
235 J. Zheng, B. Sun, D. Berardi, L. Lu, H. Yan, S. Zheng, O. Aladelokun, Y. Xie, Y. Cai, K. J. Godri Pollitt, S. A. Khan and C. H. Johnson, Perfluorooctanesulfonic Acid and Perfluorooctanoic Acid Promote Migration of Three-Dimensional Colorectal Cancer Spheroids., *Environmental science & technology*, 2023, **57**, 21016–21028.
236 S. Elumalai, U. Karunakaran, K. C. Won, S. M. Chung and J. S. Moon, Perfluorooctane sulfonate-induced oxidative stress contributes to pancreatic β-cell apoptosis by inhibiting cyclic adenosine monophosphate pathway: Prevention by pentoxifylline., *Environmental pollution (Barking, Essex : 1987)*, 2023, **320**, 120959.
237 P. Sun, L. Gu, J. Luo, Y. Qin, L. Sun and S. Jiang, ROS-mediated JNK pathway critically contributes to PFOS-triggered apoptosis in SH-SY5Y cells., *Neurotoxicology and teratology*, 2019, **75**, 106821.
238 C. L. 2nd Souders, C. L. Sanchez, W. Malphurs, J. J. Aristizabal-Henao, J. A. Bowden and C. J. Martyniuk, Metabolic profiling in human SH-SY5Y neuronal cells exposed to perfluorooctanoic acid (PFOA)., *Neurotoxicology*, 2021, **85**, 160–172.
239 H. Zhao, J. Xie, S. Wu, O. F. Sánchez, X. Zhang, J. L. Freeman and C. Yuan, Pre-differentiation exposure of PFOA induced persistent changes in DNA methylation and mitochondrial morphology in human dopaminergic-like neurons., *Environmental pollution (Barking, Essex : 1987)*, 2022, **308**, 119684.
240 O. Osemwegie, L. Butler, S. Subbiah and E. Smith, Effects of in vitro exposure of perfluorooctanoic acid and monocrotophos on astroglia SVG p12 cells., *Journal of applied toxicology : JAT*, 2021, **41**, 1380–1389.
241 R. Zhang, Y. Yao, L. Tu, T. Luan and B. Chen, Non-targeted metabolomics of multiple human cells revealing differential toxic effects of perfluorooctanoic acid., *Journal of hazardous materials*, 2021, **409**, 125017.
242 M. E. Solan, S. Senthilkumar, G. V. Aquino, E. D. Bruce and R. Lavado, Comparative cytotoxicity of seven per- and polyfluoroalkyl substances (PFAS) in six human cell lines., *Toxicology*, 2022, **477**, 153281.
243 M. E. Solan, C. P. Koperski, S. Senthilkumar and R. Lavado, Short-chain per- and polyfluoralkyl substances (PFAS) effects on oxidative stress biomarkers in human

liver, kidney, muscle, and microglia cell lines., *Environmental research*, 2023, **223**, 115424.
244 D. Liu, N.-Y. Liu, L.-T. Chen, Y. Shao, X.-M. Shi and D.-Y. Zhu, Perfluorooctane sulfonate induced toxicity in embryonic stem cell-derived cardiomyocytes via inhibiting autophagy-lysosome pathway., *Toxicology in vitro : an international journal published in association with BIBRA*, 2020, **69**, 104988.
245 A. M. Tukker, L. M. S. Bouwman, R. G. D. M. van Kleef, H. S. Hendriks, J. Legler and R. H. S. Westerink, Perfluorooctane sulfonate (PFOS) and perfluorooctanoate (PFOA) acutely affect human α(1)β(2)γ(2L) GABA(A) receptor and spontaneous neuronal network function in vitro., *Scientific reports*, 2020, **10**, 5311.
246 S. Palazzolo, I. Caligiuri, A. A. Sfriso, M. Mauceri, R. Rotondo, D. Campagnol, V. Canzonieri and F. Rizzolio, Early Warnings by Liver Organoids on Short- and Long-Chain PFAS Toxicity., *Toxics*, DOI:10.3390/toxics10020091.
247 M. Forsthuber, R. Widhalm, S. Granitzer, A. M. Kaiser, H. Moshammer, M. Hengstschläger, H. Dolznig and C. Gundacker, Perfluorooctane sulfonic acid (PFOS) inhibits vessel formation in a human 3D co-culture angiogenesis model (NCFs/HUVECs), *Environmental Pollution*, DOI:10.1016/j.envpol.2021.118543.
248 M. E. Solan, B. Schackmuth, E. D. Bruce, S. Pradhan, C. M. Sayes and R. Lavado, Effects of short-chain per- and polyfluoroalkyl substances (PFAS) on toxicologically relevant gene expression profiles in a liver-on-a-chip model., *Environmental pollution (Barking, Essex : 1987)*, 2023, **337**, 122610.
249 G. C. Addicks, A. Rowan-Carroll, A. J. F. Reardon, K. Leingartner, A. Williams, M. J. Meier, I. Moffat, R. Carrier, L. Lorusso, B. A. Wetmore, C. L. Yauk and E. Atlas, Per- and polyfluoroalkyl substances (PFAS) in mixtures show additive effects on transcriptomic points of departure in human liver spheroids., *Toxicological sciences : an official journal of the Society of Toxicology*, 2023, **194**, 38–52.
250 Q. Liao, P. Tang, Y. Song, B. Liu, H. Huang, J. Liang, M. Lin, Y. Shao, S. Liu, D. Pan, D. Huang and X. Qiu, Association of single and multiple prefluoroalkyl substances exposure with preterm birth: Results from a Chinese birth cohort study, *Chemosphere*, 2022, **307**, 135741.
251 Y. Wang, S. Jiang, B. Wang, X. Chen and G. Lu, Comparison of developmental toxicity induced by PFOA, HFPO-DA, and HFPO-TA in zebrafish embryos., *Chemosphere*, 2023, **311**, 136999.
252 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 and G.-H. Dong, Liver function biomarkers disorder is associated with exposure to perfluoroalkyl acids in adults: Isomers of C8 Health Project in China., *Environmental research*, 2019, **172**, 81–88.
253 Y. Tian, Q. Zhou, L. Zhang, W. Li, S. Yin, F. Li and C. Xu, In utero exposure to per-/polyfluoroalkyl substances (PFASs): Preeclampsia in pregnancy and low birth weight for neonates., *Chemosphere*, 2023, **313**, 137490.
254 V. H. Amstutz, A. Cengo, F. Gehres, D. T. H. M. Sijm and M. F. Vrolijk, Investigating the cytotoxicity of per- and polyfluoroalkyl substances in HepG2 cells: A structure-activity relationship approach., *Toxicology*, 2022, **480**, 153312.
255 M. Kalyn, H. Lee, J. Curry, W. Tu, M. Ekker and J. A. Mennigen, Effects of PFOS, F-53B and OBS on locomotor behaviour, the dopaminergic system and mitochondrial

function in developing zebrafish (Danio rerio)., *Environmental pollution (Barking, Essex : 1987)*, 2023, **326**, 121479.
256 C. Donat-Vargas, I. A. Bergdahl, A. Tornevi, M. Wennberg, J. Sommar, H. Kiviranta, J. Koponen, O. Rolandsson and A. Åkesson, Perfluoroalkyl substances and risk of type II diabetes: A prospective nested case-control study., *Environment international*, 2019, **123**, 390–398.
257 P. Girardi and E. Merler, A mortality study on male subjects exposed to polyfluoroalkyl acids with high internal dose of perfluorooctanoic acid, *Environ. Res.*, 2019, **179**, 108743.
258 R. B. Jain, Concentration of selected liver enzymes across the stages of glomerular function: the associations with PFOA and PFOS, *Heliyon*, 2019, **5**, e02168.
259 R. B. Jain and A. Ducatman, Selective Associations of Recent Low Concentrations of Perfluoroalkyl Substances With Liver Function Biomarkers: NHANES 2011 to 2014 Data on US Adults Aged ≥20 Years., *Journal of occupational and environmental medicine*, 2019, **61**, 293–302.
260 W. Wang, W. Zhou, S. Wu, F. Liang, Y. Li, J. Zhang, L. Cui, Y. Feng and Y. Wang, Perfluoroalkyl substances exposure and risk of polycystic ovarian syndrome related infertility in Chinese women, *Environmental Pollution*, 2019, **247**, 824–831.
261 J. Wang, X.-W. Zeng, M. S. Bloom, Z. Qian, L. J. Hinyard, R. Belue, S. Lin, S.-Q. Wang, Y.-P. Tian, M. Yang, C. Chu, N. Gurram, L.-W. Hu, K.-K. Liu, B.-Y. Yang, D. Feng, R.-Q. Liu and G.-H. Dong, Renal function and isomers of perfluorooctanoate (PFOA) and perfluorooctanesulfonate (PFOS): Isomers of C8 Health Project in China, *Chemosphere*, 2019, **218**, 1042–1049.
262 H.-J. Wen, S.-L. Wang, Y.-C. Chuang, P.-C. Chen and Y. L. Guo, Prenatal perfluorooctanoic acid exposure is associated with early onset atopic dermatitis in 5-year-old children, *Chemosphere*, 2019, **231**, 25–31.
263 Y. Ait Bamai, H. Goudarzi, A. Araki, E. Okada, I. Kashino, C. Miyashita and R. Kishi, Effect of prenatal exposure to per- and polyfluoroalkyl substances on childhood allergies and common infectious diseases in children up to age 7 years: The Hokkaido study on environment and children's health., *Environment international*, 2020, **143**, 105979.
264 C. Canova, G. Barbieri, M. Zare Jeddi, M. Gion, A. Fabricio, F. Daprà, F. Russo, T. Fletcher and G. Pitter, Associations between perfluoroalkyl substances and lipid profile in a highly exposed young adult population in the Veneto Region., *Environment international*, 2020, **145**, 106117.
265 B. A. Cohn, M. A. La Merrill, N. Y. Krigbaum, M. Wang, J.-S. Park, M. Petreas, G. Yeh, R. C. Hovey, L. Zimmermann and P. M. Cirillo, In utero exposure to poly- and perfluoroalkyl substances (PFASs) and subsequent breast cancer., *Reproductive toxicology (Elmsford, N.Y.)*, 2020, **92**, 112–119.
266 L. De Toni, C. M. Radu, I. Sabovic, A. Di Nisio, S. Dall'Acqua, D. Guidolin, S. Spampinato, E. Campello, P. Simioni and C. Foresta, Increased Cardiovascular Risk Associated with Chemical Sensitivity to Perfluoro–Octanoic Acid: Role of Impaired Platelet Aggregation, *IJMS*, 2020, **21**, 399.
267 A. F. Dreyer, R. C. Jensen, D. Glintborg, A. V. Schmedes, I. Brandslund, F. Nielsen, H. B. Kyhl, T. K. Jensen and M. S. Andersen, Perfluoroalkyl Substance Exposure Early In Pregnancy Was Negatively Associated With Late Pregnancy

Cortisone Levels, *The Journal of Clinical Endocrinology & Metabolism*, 2020, **105**, e2834–e2844.
268 I. Kashino, S. Sasaki, E. Okada, H. Matsuura, H. Goudarzi, C. Miyashita, E. Okada, Y. M. Ito, A. Araki and R. Kishi, Prenatal exposure to 11 perfluoroalkyl substances and fetal growth: A large-scale, prospective birth cohort study, *Environment International*, 2020, **136**, 105355.
269 X. Liu, D. Chen, B. Wang, F. Xu, Y. Pang, L. Zhang, Y. Zhang, L. Jin, Z. Li and A. Ren, Does Low Maternal Exposure to Per- and Polyfluoroalkyl Substances Elevate the Risk of Spontaneous Preterm Birth? A Nested Case-Control Study in China., *Environmental science & technology*, 2020, **54**, 8259–8268.
270 M. Martinsson, C. Nielsen, J. Björk, L. Rylander, E. Malmqvist, C. Lindh and A. Rignell-Hydbom, Intrauterine exposure to perfluorinated compounds and overweight at age 4: A case-control study, *PLoS ONE*, 2020, **15**, e0230137.
271 S. Salihovic, L. Lind, A. Larsson and P. M. Lind, Plasma perfluoroalkyls are associated with decreased levels of proteomic inflammatory markers in a cross-sectional study of an elderly population, *Environment International*, 2020, **145**, 106099.
272 H.-M. Shin, D. H. Bennett, A. M. Calafat, D. Tancredi and I. Hertz-Picciotto, Modeled prenatal exposure to per- and polyfluoroalkyl substances in association with child autism spectrum disorder: A case-control study., *Environmental research*, 2020, **186**, 109514.
273 L. Sinisalu, P. Sen, S. Salihović, S. M. Virtanen, H. Hyöty, J. Ilonen, J. Toppari, R. Veijola, M. Orešič, M. Knip and T. Hyötyläinen, Early-life exposure to perfluorinated alkyl substances modulates lipid metabolism in progression to celiac disease, *Environmental Research*, 2020, **188**, 109864.
274 M. C. O. Souza, M. C. P. Saraiva, M. Honda, M. A. Barbieri, H. Bettiol, F. Barbosa and K. Kannan, Exposure to per- and polyfluorinated alkyl substances in pregnant Brazilian women and its association with fetal growth., *Environmental research*, 2020, **187**, 109585.
275 C. A. G. Timmermann, K. J. Jensen, F. Nielsen, E. Budtz-Jørgensen, F. van der Klis, C. S. Benn, P. Grandjean and A. B. Fisker, Serum Perfluoroalkyl Substances, Vaccine Responses, and Morbidity in a Cohort of Guinea-Bissau Children, *Environmental Health Perspectives*, 2020, **128**, 87002.
276 M.-S. Tsai, S.-H. Chang, W.-H. Kuo, C.-H. Kuo, S.-Y. Li, M.-Y. Wang, D.-Y. Chang, Y.-S. Lu, C.-S. Huang, A.-L. Cheng, C.-H. Lin and P.-C. Chen, A case-control study of perfluoroalkyl substances and the risk of breast cancer in Taiwanese women, *Environ. Int.*, 2020, **142**, 105850.
277 H. Xu, Q. Zhou, J. Zhang, X. Chen, H. Zhao, H. Lu, B. Ma, Z. Wang, C. Wu, C. Ying, Y. Xiong, Z. Zhou and X. Li, Exposure to elevated per- and polyfluoroalkyl substances in early pregnancy is related to increased risk of gestational diabetes mellitus: A nested case-control study in Shanghai, China, *Environment International*, 2020, **143**, 105952.
278 C. M. Bulka, V. Avula and R. C. Fry, Associations of exposure to perfluoroalkyl substances individually and in mixtures with persistent infections: Recent findings from NHANES 1999-2016., *Environmental pollution (Barking, Essex : 1987)*, 2021, **275**, 116619.

279 C. Clarity, J. Trowbridge, R. Gerona, K. Ona, M. McMaster, V. Bessonneau, R. Rudel, H. Buren and R. Morello-Frosch, Associations between polyfluoroalkyl substance and organophosphate flame retardant exposures and telomere length in a cohort of women firefighters and office workers in San Francisco., *Environmental health : a global access science source*, 2021, **20**, 97.
280 Y. Duan, H. Sun, Y. Yao, Y. Li, Y. Meng, Y. Lu, L. Han and L. Chen, Serum concentrations of per-/polyfluoroalkyl substances and risk of type 2 diabetes: A case-control study, *Science of The Total Environment*, 2021, **787**, 147476.
281 F. Fart, S. Salihović, A. McGlinchey, M. G. Gareau, M. Orešič, J. Halfvarson, T. Hyötyläinen and I. Schoultz, Perfluoroalkyl substances are increased in patients with late-onset ulcerative colitis and induce intestinal barrier defects *ex vivo* in murine intestinal tissue, *Scandinavian Journal of Gastroenterology*, 2021, **56**, 1286–1295.
282 X. Han, L. Meng, G. Zhang, Y. Li, Y. Shi, Q. Zhang and G. Jiang, Exposure to novel and legacy per- and polyfluoroalkyl substances (PFASs) and associations with type 2 diabetes: A case-control study in East China., *Environment international*, 2021, **156**, 106637.
283 K. Luo, X. Liu, M. Nian, Y. Wang, J. Qiu, H. Yu, X. Chen and J. Zhang, Environmental exposure to per- and polyfluoroalkyl substances mixture and male reproductive hormones., *Environment international*, 2021, **152**, 106496.
284 J. Oh, D. H. Bennett, A. M. Calafat, D. Tancredi, D. L. Roa, R. J. Schmidt, I. Hertz-Picciotto and H.-M. Shin, Prenatal exposure to per- and polyfluoroalkyl substances in association with autism spectrum disorder in the MARBLES study, *Environment International*, 2021, **147**, 106328.
285 O. E. Omoike, R. P. Pack, H. M. Mamudu, Y. Liu and L. Wang, A cross-sectional study of the association between perfluorinated chemical exposure and cancers related to deregulation of estrogen receptors, *Environ. Res.*, 2021, **196**, 110329.
286 Y. Ou, X. Zeng, S. Lin, M. S. Bloom, F. Han, X. Xiao, H. Wang, R. Matala, X. Li, Y. Qu, Z. Nie, G. Dong and X. Liu, Gestational exposure to perfluoroalkyl substances and congenital heart defects: A nested case-control pilot study, *Environment International*, 2021, **154**, 106567.
287 E. Papadopoulou, N. Stratakis, X. Basagaña, A. L. Brantsæter, M. Casas, S. Fossati, R. Gražulevičienė, L. Småstuen Haug, B. Heude, L. Maitre, R. R. C. McEachan, O. Robinson, T. Roumeliotaki, E. Sabidó, E. Borràs, J. Urquiza, M. Vafeiadi, Y. Zhao, R. Slama, J. Wright, D. V. Conti, M. Vrijheid and L. Chatzi, Prenatal and postnatal exposure to PFAS and cardiometabolic factors and inflammation status in children from six European cohorts., *Environment international*, 2021, **157**, 106853.
288 T. Schillemans, L. Shi, C. Donat-Vargas, K. Hanhineva, A. Tornevi, I. Johansson, J. Koponen, H. Kiviranta, O. Rolandsson, I. A. Bergdahl, R. Landberg, A. Åkesson and C. Brunius, Plasma metabolites associated with exposure to perfluoroalkyl substances and risk of type 2 diabetes - A nested case-control study., *Environment international*, 2021, **146**, 106180.
289 T. S. Skogheim, K. V. F. Weyde, H. Aase, S. M. Engel, P. Surén, M. G. Øie, G. Biele, T. Reichborn-Kjennerud, A. L. Brantsæter, L. S. Haug, A. Sabaredzovic, B. Auyeung and G. D. Villanger, Prenatal exposure to per- and polyfluoroalkyl substances

(PFAS) and associations with attention-deficit/hyperactivity disorder and autism spectrum disorder in children., *Environmental research*, 2021, **202**, 111692.
290 G. Yu, M. Jin, Y. Huang, R. Aimuzi, T. Zheng, M. Nian, Y. Tian, W. Wang, Z. Luo, L. Shen, X. Wang, Q. Du, W. Xu and J. Zhang, Environmental exposure to perfluoroalkyl substances in early pregnancy, maternal glucose homeostasis and the risk of gestational diabetes: A prospective cohort study, *Environment International*, 2021, **156**, 106621.
291 S. Yu, W.-R. Feng, Z.-M. Liang, X.-Y. Zeng, M. S. Bloom, G.-C. Hu, Y. Zhou, Y.-Q. Ou, C. Chu, Q.-Q. Li, Y. Yu, X.-W. Zeng and G.-H. Dong, Perfluorooctane sulfonate alternatives and metabolic syndrome in adults: New evidence from the Isomers of C8 Health Project in China., *Environmental pollution (Barking, Essex : 1987)*, 2021, **283**, 117078.
292 E. Batzella, P. Girardi, F. Russo, G. Pitter, F. Da Re, T. Fletcher and C. Canova, Perfluoroalkyl substance mixtures and cardio-metabolic outcomes in highly exposed male workers in the Veneto Region: A mixture-based approach., *Environmental Research*, DOI:10.1016/j.envres.2022.113225.
293 Y. Gao, J. Luo, Y. Zhang, C. Pan, Y. Ren, J. Zhang and Y. Tian, Prenatal Exposure to Per- and Polyfluoroalkyl Substances and Child Growth Trajectories in the First Two Years., *Environmental health perspectives*, 2022, **130**, 37006.
294 M.-S. Ku, W.-C. Pan, Y.-T. Huang, W.-S. Hsieh, Y.-H. Hsu, P.-C. Chen and C.-Y. Liu, Associations between prenatal exposure to perfluoroalkyl substances, hypomethylation of MEST imprinted gene and birth outcomes., *Environmental pollution (Barking, Essex : 1987)*, 2022, **304**, 119183.
295 H. Li, S. Hammarstrand, B. Midberg, Y. Xu, Y. Li, D. S. Olsson, T. Fletcher, K. Jakobsson and E. M. Andersson, Cancer incidence in a Swedish cohort with high exposure to perfluoroalkyl substances in drinking water, *Environmental Research*, 2022, **204**, 112217.
296 Q.-Q. Li, J.-J. Liu, F. Su, Y.-T. Zhang, L.-Y. Wu, C. Chu, Y. Zhou, X. Shen, S. Xiong, S. D. Geiger, Z. M. Qian, S. E. McMillin, G.-H. Dong and X.-W. Zeng, Chlorinated Polyfluorinated Ether Sulfonates and Thyroid Hormone Levels in Adults: Isomers of C8 Health Project in China, *Environmental Science & Technology*, 2022, **56**, 6152–6161.
297 M. Lin, Q. Liao, P. Tang, Y. Song, J. Liang, J. Li, C. Mu, S. Liu, X. Qiu, R. Yi, Q. Pang, D. Pan, X. Zeng and D. Huang, Association of maternal perfluoroalkyl substance exposure with postpartum haemorrhage in Guangxi, China, *Ecotoxicol. Environ. Saf.*, 2022, **245**, 114078.
298 B. Liu, B. Wei, M. Mo, Y. Song, C. Tang, P. Tang, X. Guo, C. Tan, S. Liu, D. Huang and X. Qiu, Exposure to perfluoroalkyl substances in early pregnancy and the risk of hypertensive disorders of pregnancy: A nested case-control study in Guangxi, China, *Chemosphere*, 2022, **288**, 132468.
299 P. Lochhead, H. Khalili, A. N. Ananthakrishnan, K. E. Burke, J. M. Richter, Q. Sun, P. Grandjean and A. T. Chan, Plasma concentrations of perfluoroalkyl substances and risk of inflammatory bowel diseases in women: A nested case control analysis in the Nurses' Health Study cohorts., *Environmental research*, 2022, **207**, 112222.
300 M. Nian, X. Huo, J. Zhang, Y. Mao, F. Jin, Y. Shi and J. Zhang, Association of emerging and legacy per- and polyfluoroalkyl substances with unexplained recurrent spontaneous abortion, *Ecotoxicol. Environ. Saf.*, 2022, **239**, 113691.

301 J. Oh, H.-M. Shin, K. Kannan, S. A. Busgang, R. J. Schmidt, J. B. Schweitzer, I. Hertz-Picciotto and D. H. Bennett, Childhood exposure to per- and polyfluoroalkyl substances and neurodevelopment in the CHARGE case-control study, *Environmental Research*, 2022, **215**, 114322.
302 J. Qu, Y. Zhao, L. Zhang, S. Hu, K. Liao, M. Zhao, P. Wu and H. Jin, Evaluated serum perfluoroalkyl acids and their relationships with the incidence of rheumatoid arthritis in the general population in Hangzhou, China, *Environmental Pollution*, DOI:10.1016/j.envpol.2022.119505.
303 Y. Xu, C. H. Lindh, T. Fletcher, K. Jakobsson and K. Engström, Perfluoroalkyl substances influence DNA methylation in school-age children highly exposed through drinking water contaminated from firefighting foam: a cohort study in Ronneby, Sweden, *Environmental Epigenetics*, 2022, **8**, dvac004.
304 Q. Yao, A. Vinturache, X. Lei, Z. Wang, C. Pan, R. Shi, T. Yuan, Y. Gao and Y. Tian, Prenatal exposure to per- and polyfluoroalkyl substances, fetal thyroid hormones, and infant neurodevelopment., *Environmental research*, 2022, **206**, 112561.
305 Y. Yu, X.-D. Qin, M. S. Bloom, C. Chu, X. Dai, Q. Li, Z.-X. Chen, M.-L. Kong, Y.-Q. Xie, W.-J. Meng, B.-Y. Yang, L.-W. Hu, X.-W. Zeng, X.-M. Zhao, Y. Zhou and G.-H. Dong, Associations of prenatal exposure to perfluoroalkyl substances with preterm birth: A family-based birth cohort study, *Environmental Research*, 2022, **214**, 113803.
306 Y. Zhang, V. Mustieles, Y. Sun, Y. Oulhote, Y.-X. Wang and C. Messerlian, Association between serum per- and polyfluoroalkyl substances concentrations and common cold among children and adolescents in the United States, *Environ. Int.*, 2022, **164**, 107239.
307 Y. Zhao, H. Jin, J. Qu, S. Zhang, S. Hu, J. Xue and M. Zhao, The influences of perfluoroalkyl substances on the rheumatoid arthritis clinic, *BMC Immunol*, 2022, **23**, 10.
308 J. M. Bailey, L. Wang, J. M. McDonald, J. S. Gray, J. G. Petrie, E. T. Martin, D. A. Savitz, T. A. Karrer, K. A. Fisher, M. J. Geiger and E. A. Wasilevich, Immune response to COVID-19 vaccination in a population with a history of elevated exposure to per- and polyfluoroalkyl substances (PFAS) through drinking water., *Journal of exposure science & environmental epidemiology*, 2023, **33**, 725–736.
309 Y. Dai, J. Zhang, Z. Wang, J. Ding, S. Xu, B. Zhang, J. Guo, X. Qi, X. Chang, C. Wu and Z. Zhou, Per- and polyfluoroalkyl substances in umbilical cord serum and body mass index trajectories from birth to age 10 years: Findings from a longitudinal birth cohort (SMBCS), *Environment International*, 2023, **180**, 108238.
310 A. He, J. Li, Z. Li, Y. Lu, Y. Liang, Z. Zhou, Z. Man, J. Lv, Y. Wang and G. Jiang, Novel Insights into the Adverse Health Effects of per- and Polyfluoroalkyl Substances on the Kidney via Human Urine Metabolomics, *Environ. Sci. Technol.*, 2023, **57**, 16244–16254.
311 H. Li, J. Chen, Jingchao Lu, J. Yang, Z. Tan, L. Li, F. Xiao, Z. An, C. Ma, Y. Liu, L. Wang, X. Zhang and H. Guo, Association of exposure to perfluoroalkyl substances and risk of the acute coronary syndrome: A case-control study in Shijiazhuang Hebei Province, *Chemosphere*, 2023, **313**, 137464.
312 H. Li, M. Yang, J. Yang, S. Seery, C. Ma, Y. Liu, X. Zhang, A. Li and H. Guo, Per- and polyfluoroalkyl substances and the associated thyroid cancer risk: A case-control study in China, *Chemosphere*, 2023, **337**, 139411.

313 L.-X. Liang, P. Dong, Y. Zhou, L. Zhang, Z. Qian, S. D. Geiger, E. Bingheim, X. Tang, Y. Wu, J. Lv, L.-Z. Lin, M. Zeeshan, X.-W. Zeng, W. Feng and G.-H. Dong, Joint effects of per- and polyfluoroalkyl substance alternatives and heavy metals on renal health: A community-based population study in China, *Environmental Research*, 2023, **219**, 115057.
314 J. Rhee, V. C. Chang, I. Cheng, A. M. Calafat, J. C. Botelho, J. J. Shearer, J. N. Sampson, V. W. Setiawan, L. R. Wilkens, D. T. Silverman, M. P. Purdue and J. N. Hofmann, Serum concentrations of per- and polyfluoroalkyl substances and risk of renal cell carcinoma in the Multiethnic Cohort Study, *Environment International*, 2023, **180**, 108197.
315 R. C. Siwakoti, A. Cathey, K. K. Ferguson, W. Hao, D. E. Cantonwine, B. Mukherjee, T. F. McElrath and J. D. Meeker, Prenatal per- and polyfluoroalkyl substances (PFAS) exposure in relation to preterm birth subtypes and size-for-gestational age in the LIFECODES cohort 2006-2008., *Environmental research*, 2023, **237**, 116967.
316 Y. Xu, K. Jakobsson, F. Harari, E. M. Andersson and Y. Li, Exposure to high levels of PFAS through drinking water is associated with increased risk of type 2 diabetes—findings from a register-based study in Ronneby, Sweden, *Environmental Research*, 2023, **225**, 115525.
317 L. Zang, X. Liu, X. Xie, X. Zhou, Y. Pan and J. Dai, Exposure to per- and polyfluoroalkyl substances in early pregnancy, risk of gestational diabetes mellitus, potential pathways, and influencing factors in pregnant women: A nested case-control study, *Environmental Pollution*, 2023, **326**, 121504.
318 B. Zhang, Z. Wang, J. Zhang, Y. Dai, C. Feng, Y. Lin, L. Zhang, J. Guo, X. Qi, X. Chang, D. Lu, C. Wu and Z. Zhou, Prenatal perfluoroalkyl substances exposure and neurodevelopment in toddlers: Findings from SMBCS., *Chemosphere*, 2023, **313**, 137587.
319 M. van Gerwen, E. Colicino, H. Guan, G. Dolios, G. N. Nadkarni, R. C. H. Vermeulen, M. S. Wolff, M. Arora, E. M. Genden and L. M. Petrick, Per- and polyfluoroalkyl substances (PFAS) exposure and thyroid cancer risk, *EBioMedicine*, 2023, **97**, 104831.
320 J. Ao, R. Zhang, X. Huo, W. Zhu and J. Zhang, Environmental exposure to legacy and emerging per- and polyfluoroalkyl substances and endometriosis in women of childbearing age, *Science of The Total Environment*, 2024, **907**, 167838.
321 Y. Chen, K. C. Paul, D. I. Walker, D. P. Jones, X. Wang, B. R. Ritz and J. E. Heck, Neonatal per- and polyfluoroalkyl substance exposure in relation to retinoblastoma., *Environmental research*, 2024, **240**, 117435.