CHEMWATCH

TECHNICAL

Week of 10 April 2026

TECHNICAL

CONTENTS

TECHNICAL

OCCUPATIONAL

Occupational exposure to cancer risk factors among health and social care workers in Europe: results from the Workers' Exposure Survey

Skin Sensitisation and Dermatitis Among Epoxy-Exposed Workers in a Wind Turbine Plant: Influence of Occupational Factors

CHEMICAL EFFECTS

Longitudinal patterns of urinary biomarkers of placental and renal function in pregnancy and associations with early pregnancy exposure to phthalates and replacements

Prenatal exposure to pesticides and the risk of male genital malformations: a scoping review

ENVIRONMENTAL RESEARCH

Short-chain PFASs dominate in crops near a fluorochemical industrial park: Stratified exposure and Monte Carlo-based health risk assessment

Distribution, sources, and health risk assessment of polycyclic aromatic hydrocarbons (PAHs) in dust from petrol stations in the main urban districts of Guiyang

Metagenomic analysis of urban water systems uncovers the interplay between antibiotic resistance genes and microbial communities in response to PFAS contamination

PHARMACEUTICAL/TOXICOLOGY

The Role of sGC-cGMP-PKG Signaling Pathway-Mediated Excitotoxicity in Delayed Encephalopathy After Carbon Monoxide Poisoning

Exercise-induced irisin rescues lead-induced cognitive impairment by inhibiting prefrontal cortical senescence via SIRT3

Reprogramming of fatty acid metabolism induced by environmental pollutant exposure and its role in the toxic effects of environmental pollutants

TECHNICAL

OCCUPATIONAL

Occupational exposure to cancer risk factors among health and social care workers in Europe: results from the Workers' Exposure Survey

European journal of public health 2026 Mar 14;36(2) · 14 Mar 2026

Occupational exposure to cancer risk factors is an important avoidable cause of cancer. The European Agency for Safety and Health at Work (EU-OSHA) conducted a Workers' Exposure Survey (WES) on cancer risk factors to increase knowledge on the prevalence and circumstances of exposure to 24 known cancer risk factors and on workplace prevention strategies in Europe. This manuscript focusses on the human health and social care work activities (HeSCare) sector, one of the largest occupational sectors in Europe. WES includes 24 402 telephone interviews from 2022 to 2023 on workers in Finland, France, Germany, Hungary, Ireland, and Spain. WES uses the Occupational Integrated Database Exposure Assessment System (OccIDEAS) where probable exposure to selected cancer risk factors during the last working week was automatically estimated based on workers' answers to detailed sets of questions adapted to the EU context. There were 3041 workers affiliated with the HeSCare sector and almost two-thirds (65.3%) were female. A total of 29.5% of workers were probably exposed to one or more of the included cancer risk factors and 7.8% to two or more. The most common exposures among those considered were to ionizing radiation (7.4%), diesel engine exhaust emissions (6.2%), solar ultraviolet radiation (6.1%), formaldehyde (5.2%), and benzene (4.8%). The most frequent exposures estimated to occur at a high level in HeSCare were formaldehyde (2.3%) and ethylene oxide (2.0%). WES provides valuable sector-specific data about exposure to the most common cancer risk factors in occupational settings in Europe.

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Skin Sensitisation and Dermatitis Among Epoxy-Exposed Workers in a Wind Turbine Plant: Influence of Occupational Factors

Contact dermatitis 2026 Apr 03 · 3 Apr 2026

BACKGROUND: Epoxy polymers, widely used in industries such as wind turbine production, are common causes of allergic contact dermatitis (ACD). However, gaps remain mostly regarding the type of occupational exposure (specific tasks) and protective strategies.

METHODS: A cross-sectional study was performed in a large wind turbine blade facility. A group of volunteer workers completed a questionnaire on occupational/medical history, PPE use, and symptoms. Onsite patch testing included the European Baseline-, epoxy-, and isocyanate series. Data were analysed using SPSS and R ($p < 0.05$).

RESULTS: Among 131 workers tested (7.4% of the workforce), seven (5.3%), all from high-exposure tasks, showed positive reactions to epoxy compounds: Bisphenol F resin (5), Bisphenol A resin (4), and hardeners/additives (5). Hand dermatitis was the predominant ACD pattern, with a median time to onset of 8 months after employment. No associations were found between sensitisation and previous medical history or PPE compliance.

CONCLUSIONS: The ACD to epoxy resin is a significant concern in wind turbine blade manufacturing. Accurate diagnosis requires patch testing with epoxy-specific series, beyond the European Baseline series. Prevention should extend beyond PPE, addressing airborne exposure and cross-contamination.

OBJECTIVES: To evaluate epoxy resin sensitisation in a wind turbine blade plant and assess its associations with tasks, medical history, clinical patterns, and personal protective equipment (PPE) use.

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CHEMICAL EFFECTS

Longitudinal patterns of urinary biomarkers of placental and renal function in pregnancy and associations with early pregnancy exposure to phthalates and replacements

Reproductive toxicology (Elmsford, N.Y.) 2026 Mar 31:142:109234 · 31 Mar 2026

Phthalates and replacements are endocrine-disrupting chemicals that may interfere with early pregnancy processes such as placentation and renal adaptation. Placental and renal biomarkers may help examine these toxicologic mechanisms. This study aims to characterize longitudinal changes in urinary protein biomarkers of placental angiogenesis (sFlt-1, PlGF, sEng) and renal function (KIM-1, NGAL, nephrin), and investigate associations with early pregnancy exposure to phthalates and replacements. Among 291 participants from the Human Placenta and Phthalates prospective cohort, urinary metabolites of eight phthalates and two replacements were quantified at up to 2 time points from 12 to 15 weeks gestation. Repeated measures were averaged as early pregnancy exposure biomarkers. Placental and renal biomarkers were quantified longitudinally in urine samples from early (median 13 weeks), middle (21 weeks), and late (33 weeks) pregnancy. Linear mixed effects models with participant-specific random effects estimated the percent change in placental or renal biomarker concentrations throughout pregnancy in association with a one interquartile range increase in early pregnancy phthalate and replacement biomarkers. Certain protein biomarkers showed trends across gestation (sFlt-1, PlGF, NGAL), while others did not (sEng, KIM-1, nephrin). Early pregnancy mono-3-carboxypropyl phthalate was associated with decreased urinary concentrations of NGAL (-6.66% [95% confidence interval: -12.23%, -0.74%]), but this association was nonsignificant after multiple comparisons correction. Associations between other phthalate and replacement biomarkers with placental and renal proteins were imprecise. Urinary biomarkers of placental angiogenesis and renal function change dynamically in pregnancy; further research is needed to validate associations between urinary proteins and phthalate metabolites, and to explore additional toxicity mechanisms.

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Prenatal exposure to pesticides and the risk of male genital malformations: a scoping review

Reproductive toxicology (Elmsford, N.Y.) 2026 Apr 03:143:109238 · 3 Apr 2026

INTRODUCTION: Hypospadias and undescended testis (UDT) are common genital malformations with multifactorial origins. Endocrine-disrupting chemicals, including pesticides, are suspected contributors, but prior reviews are outdated, and none has mapped evidence for both conditions together.

METHOD: Following PRISMA-ScR guidance, we searched PubMed, Web of Science, Cochrane, and Embase from 2003 to 2024. Eligible items were original human studies and reviews. Study selection and data extraction were performed by two reviewers.

RESULTS: We included 117 articles (62 case-control studies, 7 population-based/cohort/epidemiological studies, 4 meta-analyses, and 44 reviews). Evidence from primary studies was heterogeneous across designs, but several signals recurred: increased risks associated with maternal occupational or residential exposure in agricultural settings; positive associations when exposure was assessed using bioaccumulative matrices (placenta, breast milk, meconium); and limited or null findings with single time-point blood/urine measures. Data on household insecticides use remain scarce, particularly for pyrethroid insecticides. Few studies examined gene-environment interactions.

CONCLUSION: This scoping review highlights suggestive but inconsistent evidence that prenatal pesticide exposure contributes to hypospadias/UDT, with organochlorines (e.g., DDT/DDE, atrazine) most frequently implicated. Key gaps include pyrethroid exposure, prospective designs with improved exposure assessment, and stronger links between human epidemiological findings and existing mechanistic evidence. While causal inference is limited in a scoping review framework, the overall body of evidence supports adopting a precautionary approach to minimizing pesticide exposure during pregnancy.

OBJECTIVE: To map evidence on the association between prenatal pesticide exposure and hypospadias/UDT, with particular attention to underexplored exposures such as pyrethroids and household pesticides, and to identify research gaps.

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ENVIRONMENTAL RESEARCH

Short-chain PFASs dominate in crops near a fluorochemical industrial park: Stratified exposure and Monte Carlo-based health risk assessment

Journal of hazardous materials 2026 Apr 04:508:141987 · 4 Apr 2026

Fluorochemical Industrial Parks (FIPs) are critical point sources of per- and polyfluoroalkyl substances (PFASs), raising serious concerns about their impacts on surrounding agricultural environment and food safety. This study conducted a targeted investigation of priority PFASs in the agricultural environment surrounding a major domestic FIP, to evaluate the subsequent health risks for local residents through the consumption of homegrown vegetables and rice. The results indicated that PFASs were widespread in the soil, with total concentrations ranging from 40.7 to 72.0 ng/g. The average concentration of PFASs followed the order: traditional PFASs > emerging

PFASs, with short-chain PFCAs (C≤8) being predominant. Significant accumulation of PFASs was observed in the edible portions of vegetables, particularly in leafy greens, which exhibited the highest bioaccumulation capacity. In rice, PFASs concentrations ranged from 0.329 to 11.3 ng/g dw, and short-chain PFCAs were also the dominant compounds. Residents were stratified by age and gender to estimate their daily exposure doses (EDI), which ranged from 70.5 to 136 ng/kg bw/d. Notably, Monte Carlo simulations revealed that a substantial proportion of local residents exceeded the tolerable daily intake levels for PFOA and PFOS, suggesting potential health risks associated with long-term exposure. This study demonstrated that the FIP served as a direct and significant source of PFASs contamination in the local agricultural ecosystem, resulting in non-occupational human exposure. The findings underscored the urgent need for regulatory oversight and the development of effective mitigation strategies to safeguard the health of residents living in proximity to fluorine chemical production facilities.

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Distribution, sources, and health risk assessment of polycyclic aromatic hydrocarbons (PAHs) in dust from petrol stations in the main urban districts of Guiyang

Environmental geochemistry and health 2026 Apr 04;48(6) · 4 Apr 2026

As typical urban service facilities, petrol stations are vulnerable to pollution caused by motor vehicle exhaust and petrol spills. Dust samples collected from petrol stations in the main urban districts of Guiyang were analyzed in this study to determine the concentrations, sources, and health risks of 16 polycyclic aromatic hydrocarbons (PAHs). All 16 PAHs were detected in over 90% of the dust samples, with total concentrations (∑16PAHs) ranging from 0.190 to 14.968 mg/kg (mean: 1.550 mg/kg), a mean level considered moderate when compared to other cities in China and globally. The predominant PAHs in the dust were medium molecular weight (MMW) and high molecular weight (HMW) compounds, which together accounted for 80.6% of the mean concentration of the ∑16PAHs. The frequent occurrence of traffic congestion was the primary factor contributing to the higher mean concentrations of MMW PAHs, HMW PAHs, carcinogenic PAHs (∑carPAHs) and ∑16PAHs in dust from Nanming districts (NMD) and Yunyan districts (YYD). Diagnostic ratio results revealed that the PAHs were predominantly derived from vehicle exhaust, with a lesser contribution from fuel spillage and biomass combustion. The results of the incremental lifetime cancer risk (ILCR) showed that ingestion, dermal contact, and inhalation posed no carcinogenic risk to non-occupationally exposed adults (NOEA). For petrol station workers (PSW), dermal contact presented a high carcinogenic risk, while the ingestion pathway posed a potential carcinogenic risk. Personal protective measures, such as wearing protective suits and gloves, should be implemented to reduce the ILCR from dermal contact, while the use of masks and increasing frequency of personal cleaning could reduce the ILCR from ingestion. Furthermore, promoting the adoption of green energy sources, such as electric vehicles, can effectively reduce fuel consumption and shorten exposure time at petrol stations, thereby lowering the concentration of pollution sources.

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Metagenomic analysis of urban water systems uncovers the interplay between antibiotic resistance genes and microbial communities in response to PFAS contamination

Journal of hazardous materials 2026 Mar 27:508:141890 · 27 Mar 2026

Urban water systems (UWS) are facing the severe challenge of coexisting emerging contaminants per- and polyfluoroalkyl substances (PFAS) and antibiotic resistance genes (ARGs). Herein, we analyze 15 PFAS at all key nodes within the UWS and the manufacturing plant park (MPP) in industrial clusters. Meanwhile, 16S rRNA and metagenomic approach were employed to annotate microbial community and ARGs, investigating their response to PFAS contamination. Fifteen PFAS were detected in MPP wastewater with total concentrations ranging from 30.28 to 3738.51 (557.68 ± 1072.03) ng/L, with short-chain accounting for 63.5%. Wastewater treatment plant (WWTP) serves as both sink and source of PFAS, with a negative average removal efficiency (mean = -158.6%) ultimately contributing to the prevalence of PFAS in the drinking water treatment plants (DWTPs) and tap water (17.64 -84.72, 36.06 ± 18.52 ng/L). 1141 ARGs subtypes were identified by metagenomic with significant differences in relative abundance between different nodes samples ($p = 0.00$). Additionally, the co-occurrence network revealed 14 genera may as potential hosts for 25 ARGs subtypes. However, significant differences in microbial diversity and abundance were observed at different nodes samples ($R = 0.408$, $p = 0.00$), with PFAS reducing microbial community diversity, particularly in river system ($R = 0.723$, $p = 0.00$). Finally, the structural equation modeling (SEM) revealed that PFAS exerted the greatest negative contribution to ARGs profiles (total effect = -1.39) through synergistic effects involving direct negative impacts on microbial diversity (-0.679) and mobile genetic elements (MGEs) (-0.121). This suggests that PFAS may influence the ARGs profiles by synergistically inhibiting gene-level transfer mediated by MGEs within potential host microbial. Additionally, physicochemical parameters (0.42), nutrient levels (-0.29), and ion concentrations (0.06) were also minor drivers of ARGs profiles.

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PHARMACEUTICAL/TOXICOLOGY

The Role of sGC-cGMP-PKG Signaling Pathway-Mediated Excitotoxicity in Delayed Encephalopathy After Carbon Monoxide Poisoning

Neurotoxicology 2026 Apr 01 · 1 Apr 2026

This study investigated the role of soluble guanylate cyclase-cyclic guanosine monophosphate-protein kinase G (sGC-cGMP-PKG) signaling pathway in the regulation of carbon monoxide (CO) on glutamate neurotransmitter system and neuronal excitotoxicity in vitro and in vivo, and revealed the possible mechanism of delayed encephalopathy after acute carbon monoxide poisoning (DEACMP). The results showed that compared with the control group, a large number of apoptotic cells appeared in the hippocampus of mice 7 and 14 days after CO exposure, and the learning and spatial memory functions were decreased. The protein expression levels of heme oxygenase-1 (HO-1), sGC, PKG, N-methyl-D-aspartate receptor 2A (NR2A) and N-methyl-D-aspartate receptor 2B (NR2B) and the concentrations of glutamate, cGMP and calcium ion (Ca^{2+}) were significantly increased at different time points after CO exposure. At the same time, in the in vitro model of acute CO poisoning, the expression of HO-1, sGC, PKG, NR2A and NR2B in PC12 cells exposed to different

concentrations of CO increased, glutamate release increased, Ca^{2+} overload, excitotoxicity, and then led to cell apoptosis. After treatment with sGC specific inhibitor, the expression level of N-methyl-D-aspartate (NMDA) receptor protein and Ca^{2+} concentration were significantly decreased, the number of apoptotic cells was significantly decreased, and the animal behavior related indicators were improved. The results of the present study suggest that excitotoxicity mediated by sGC-cGMP-PKG signaling plays an important role in DEACMP induced by CO poisoning.

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Exercise-induced irisin rescues lead-induced cognitive impairment by inhibiting prefrontal cortical senescence via SIRT3

Neuroscience letters 2026 Apr 02:879:138597 · 2 Apr 2026

Lead (Pb) is a pervasive environmental hazard that impairs cognitive function. However, the underlying mechanisms and non-pharmacological interventions need to be further investigated. Here, Pb-induced senescence in the prefrontal cortex was associated with cognitive impairment. We found that exercise and exercise serum ameliorated Pb-induced prefrontal cortex senescence and cognitive impairment in mice. Further analyses revealed that exercise-derived irisin alleviated cellular senescence by regulating the SIRT3 signaling pathway. Meanwhile, irisin also mediated the association between blood Pb levels and MoCA scores in humans. Together, these findings suggest that exercise alleviates Pb-induced cognitive impairment via the irisin-SIRT3 pathway and identify irisin as a potential therapeutic target.

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Reprogramming of fatty acid metabolism induced by environmental pollutant exposure and its role in the toxic effects of environmental pollutants

Environmental geochemistry and health 2026 Apr 04;48(6) · 4 Apr 2026

Fatty acids (FAs) are vital biomolecules that serve not only as energy substrates but also as bioactive molecules, moieties of other lipids such as phospholipids and triglycerides, structural components of membranes, and precursors to other bioactive molecules. Environmental pollutants, are ubiquitous across environmental, wildlife, and human systems and disrupt FA homeostasis by targeting FA receptors and their associated metabolic networks. Although pollutant-induced disruption of FA metabolism is increasingly recognized, existing studies and reviews have often focused on individual pollutant classes, single receptors, or isolated metabolic processes, which has limited an integrated understanding of how chemically diverse pollutants converge on shared FA metabolic programs. This review provides a cross-pollutant synthesis of mechanistic insights into pollutant-induced disturbances in FA metabolism, focusing on receptor-mediated FA synthesis, oxidation, transport, storage, and utilization for the synthesis of other lipids. We review how pollutants interact with FA receptors and modulate downstream metabolic pathways, thereby altering the levels of FAs and other metabolites and contributing to toxic effects in multiple tissues. This review discusses several receptor-mediated signaling cascades and downstream metabolic pathways that drive pollutant-induced FA metabolic reprogramming and subsequent lipid accumulation. By delineating the pollutant-receptor-metabolite axis, this review provides a unifying framework for understanding how environmental pollutants across major classes drive metabolic reprogramming and for identifying potential intervention targets to mitigate their associated toxicity.

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