CHEMWATCH

TECHNICAL

Week of 6 March 2026

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TECHNICAL

CONTENTS

TECHNICAL

Exposure to welding fumes and health effects: A systematic evaluation based on human biomonitoring analyses

Toxicology letters 2026 Feb 25:418:111861 · 25 Feb 2026

BACKGROUND: Occupational exposure to heavy metals (HMs) through inhalation of welding emissions presents a serious health hazard to welders. This systematic review aimed to evaluate the concentrations of HMs in biological matrices (blood, urine, serum, and plasma) of welders, and to examine the associations between exposure to welding emissions, oxidative stress markers, and respiratory health outcomes.

METHODS: A comprehensive literature search was conducted using Web of Science, Scopus, PubMed, Medline, and Embase up to September 16, 2025, based on the PECO (Population, Exposure, Comparator, and Outcomes) framework. A total of 76 studies met the inclusion criteria.

RESULTS: The majority of studies reported significantly higher HM levels in welders compared to control groups. A strong association was observed between metal concentrations in welding emissions and corresponding levels in biological matrices, although the strength of this relationship was occasionally diminished due to confounding variables. More than half of the included studies demonstrated elevated oxidative stress markers such as MDA and 8-OHdG in welders. However, fewer than half of the studies reported significant differences in pulmonary function parameters, with forced vital capacity (FVC) being the most frequently affected.

CONCLUSION: These findings underscore the importance of implementing protective strategies, including routine human biomonitoring and engineering controls, to mitigate HM exposure, reduce oxidative stress, and prevent related health outcomes among welders.

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The effect of occupational exposure to petrol on biochemical and hematological parameters: a review and meta-analysis

International journal of environmental health research 2026 Feb 28 · 28 Feb 2026

Continuous occupational exposure of fuel station workers to petrol and diesel vapors, which contain volatile organic compounds such as BTEXs, may adversely affect hematological parameters. This meta-analysis evaluated the impact of such exposures on blood indices. Online databases were systematically searched using relevant MeSH keywords. After removing duplicates, articles were screened, and quality was assessed using the Newcastle - Ottawa Scale. A random-effects model was applied to estimate standardized mean differences (SMD) in blood indices between exposed and unexposed groups. Sensitivity analysis, meta-regression, and Egger's test were used to assess study influence, exposure duration, and publication bias. Thirty-seven studies, with 2244 exposed and 2106 unexposed participants, were included. Pooled results showed significant decreases in red blood cell count (SMD = -1.03, 95% CI: -1.54 to -0.52), white blood cell count (SMD = -0.54, 95% CI: -0.94 to -0.15), hemoglobin (SMD = -0.82, 95% CI: -1.34 to -0.31), and mean corpuscular hemoglobin (SMD = -0.69, 95% CI: -1.14 to -0.23), while increases were observed in mean corpuscular hemoglobin

concentration, hematocrit, platelets, lymphocytes, and neutrophils. Changes were more pronounced with longer exposure. Overall, exposure to fuel vapors was linked to reduced red blood cell indices and increased white blood cell indices, serving as potential early indicators of adverse health effects.

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CHEMICAL EFFECTS

Multi-omics biomarkers in cadmium-related lung toxicity and carcinogenesis

Clinica chimica acta; international journal of clinical chemistry 2026 Feb 26 · 26 Feb 2026

Cadmium exposure through smoking, occupational exposure, and pollution is linked to lung damage and cancer risk; however, most mechanistic studies remain limited to in vitro transformation and animal studies rather than large human cohorts. However, there are few clinically applicable biomarkers for early detection, risk assessment, and therapeutic monitoring. This review, centered on mechanisms, provides a summary and critical analysis of multi-omics biomarker evidence related to cadmium (Cd)-induced lung toxicity and carcinogenesis, with a particular focus on diagnostic laboratory medicine. We combine genomic and epigenomic signals (mutational signatures, cfDNA/ctDNA, methylation, and miRNA profiles) with proteomic and metabolomic readouts that represent inflammation, oxidative stress, extracellular matrix remodeling, and altered energy metabolism. In matrices (blood, urine, sputum, bronchoalveolar lavage, and extracellular vesicles), we emphasize key pre-analytical variables affecting measurements (collection tubes, processing delays, hemolysis, storage temperature, freeze-thaw cycles, and batch effects) rather than providing comprehensive experimental validation of the assays. Where appropriate, we comment on representative requirements for analytical performance and common pitfalls (limit of detection, precision, interference, calibration/traceability, and external quality assessment) of candidate biomarker classes; however, a comprehensive discussion of quantitative clinical performance measures (sensitivity, specificity, AUC, prospective validation) for most markers is lacking. Finally, we suggest a practical, stepwise translation pathway that emphasizes mechanistically informed multi-analyte panels, documented differences between exposure and effect biomarkers, future validation in exposed cohorts, and clinically relevant endpoints (reduction in lung function, incidence of malignancy, and response to treatment) to facilitate routine laboratory implementation. To demonstrate the clinical relevance and economic viability of controlling confounders and establishing decision limits, standard reporting of methods, rigorous control of confounders (particularly smoking and co-exposures), and explicit derivation of decision limits based on specific targeted applications (such as screening, surveillance, or monitoring of responses) should be prioritized over current evidence base achievements.

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DEHP exposure and cardiac fibrosis risk: An integrated network toxicology and molecular experimental investigation

Ecotoxicology and environmental safety 2026 Feb 28:312:119943 · 28 Feb 2026

The environmental endocrine-disrupting chemical di-(2-ethylhexyl) phthalate (DEHP) has been epidemiologically linked to cardiac fibrosis, yet the underlying molecular mechanism remains poorly defined. To dissect the mechanism, we employed an integrative strategy combining network toxicology, molecular-dynamics (MD) simulations and in vitro experimental validation. Putative DEHP targets (n = 6439) were mined from the Comparative Toxicogenomics Database (CTD) and SwissTargetPrediction, while cardiac fibrosis-related genes (n = 2288) were curated from GeneCards and OMIM. Overlap analysis identified 1054 shared candidates. These were imported into STRING to construct a high-confidence protein-protein interaction (PPI) network, which was subsequently analyzed in Cytoscape, revealing a 12-hub core enriched in SRC, STAT3, EGFR and other key regulators. Functional enrichment underscored a prominent footprint in inflammatory responses and in

the PI3K-Akt and MAPK signaling cascades. Molecular-docking analyses revealed high binding affinities between DEHP and the identified core targets, while MD simulations verified the structural stability of DEHP complexes with SRC, STAT3 and EGFR. In vitro, cardiomyocyte viability declined progressively with increasing DEHP concentrations. Exposure to 50 µg/mL DEHP elevated the BAX/BCL2 ratio, promoted cytochrome c release, and raised the number of TUNEL-positive cells, confirming that DEHP induces cardiomyocyte apoptosis. In cardiac fibroblasts, DEHP up-regulated transcription of Tgf-β1, Col1a1, and Acta2, driving their differentiation toward a myofibroblast phenotype. Western blot analyses revealed that DEHP activated SRC and STAT3 phosphorylation; pharmacologic inhibition of SRC or STAT3 phosphorylation attenuated DEHP-induced phenotypic conversion of cardiac fibroblasts. Collectively, DEHP exacerbates cardiac fibrosis by triggering cardiomyocyte apoptosis and by activating the SRC-STAT3 axis to promote the conversion of cardiac fibroblasts into myofibroblasts, providing clear evidence of DEHP-elicited cardiotoxicity and a theoretical basis for targeted intervention.

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ENVIRONMENTAL RESEARCH

Conflicting results of ecological and health risk assessment of perfluorinated compounds in major river basins in China

Journal of environmental sciences (China) 2026 Apr:162:112-120 · 1 Apr 2026

Per- and polyfluoroalkyl substances (PFASs) are persistent chemicals that exhibit potential risks to aquatic organisms and humans due to their bioaccumulation and toxic effects. As the largest producer and consumer of PFASs, China faces a critical need for ecological and health risk assessments of these substances. Unlike traditional methods that often rely on limited data, this study leverages the Interspecies correlation estimation (ICE)-species sensitivity distribution (SSD) model to expand toxicity data across species. In this study, the ICE- SSD model expanded toxicity data from three surrogate species to 33 predicted species and obtain predicted no-effect concentrations (PNECs). Based on these PNECs, ecological risk assessments were conducted for the seven major river basins in China. Subsequently, Monte Carlo simulation was used to account for uncertainties in environmental concentrations and human exposure patterns, based on aquatic product consumption patterns among the population. The results indicated that PFASs presented no or low ecological risk (risk quotient <0.1) in Chinese major river basins. However, the health risk assessment revealed that, except for Perfluorobutanoic acid (PFBA) and Perfluorobutane sulfonic acid (PFBS), all target PFASs could pose high health risks. Notably, the Hai River exhibited the highest total hazard quotient (THQ), with 33 % of adults and 47 % of children facing high health risks. And the detected Perfluorooctane Sulfonic Acid (PFOS) levels in the river basins were identified as a key factor contributing to the increased THQ. Therefore, both ecological and health risks should be considered when assessing the risks of pollutants.

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Association between prenatal exposure to per- and polyfluoroalkyl substances and pubertal development in boys and girls in the Spanish INMA cohort

Environmental research 2026 Feb 26 · 26 Feb 2026

Exposure to endocrine-disrupting chemicals (EDCs), including per- and polyfluoroalkyl substances (PFAS), has been linked to altered pubertal timing, though epidemiological findings remain inconsistent. This study examined associations between prenatal PFAS exposure and pubertal development in children. Concentrations of perfluorohexane sulfonic acid (PFHxS), perfluorooctanoic acid (PFOA), perfluorooctane sulfonic acid (PFOS) and perfluorononanoic acid (PFNA) were quantified in maternal plasma collected during the first trimester of pregnancy in the Spanish INMA (Infancia y

Medio Ambiente) cohort. Children's pubertal development was assessed longitudinally between ages 7 and 13 using the parent-reported Pubertal Development Scale (PDS), with specific scales for gonadal and adrenal development. Data were available for 492 mother-girls pairs at the 7-9-year follow up and 475 mother-boys pairs at the 11-13-year follow up based on the typical sex-specific timing of puberty. Poisson regression and Bayesian Kernel Machine Regression (BKMR) were used to estimate associations between PFAS (individually and as mixtures, respectively) and the risk of earlier puberty development (PDS stage 1 vs. 2+), adjusting for confounders. Among girls, PFHxS was associated with an increased risk of early adrenarche (Relative Risk [RR]=1.85; 95% confidence interval [CI]: 1.05-3.25), while PFOS was inversely associated with early gonadarche RR=0.61; 95% CI: 0.37-1.00). In boys, PFOS showed a marginal trend toward an increased risk of early overall pubertal onset (RR=1.47; 95% CI: 0.99-2.19). Stratified analyses mainly revealed stronger associations among overweight/obese children. The mixture analysis suggested a positive trend for early adrenal development in both sexes, with significant associations in boys. Although our findings do not provide definitive evidence of a relationship between prenatal PFAS exposure and pubertal timing, they are compatible with the endocrine-disrupting potential of PFAS. The observed patterns, including possible modification by weight status and mixture signals, warrant further research.

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Heavy metals profiling of road dust from metro stations: spatial distribution, source apportionment, and Monte Carlo simulation-based probabilistic human health assessment

Environmental geochemistry and health 2026 Feb 28;48(5) · 28 Feb 2026

Road dust in urban areas is a significant carrier of heavy metals (HMs), posing serious environmental and health threats to humans. This study investigates the concentration, sources, and associated health risks of HMs (Cr, Mn, Ni, Cu, Zn, Cd, and Pb) in road dust collected from 27 metro bus stations in Lahore, Pakistan. The mean concentrations (mg/kg) of Cr, Mn, Ni, Cu, Zn, Cd, and Pb in road dust were 51.83, 364.79, 22.36, 93.57, 421.50, 3.37, and 50.64, respectively, following the descending order: Zn > Mn > Cu > Cr > Pb > Ni > Cd. Pollution indices revealed severe contamination by Cd (CF > 6), significant contamination by Zn, and a high ecological risk index (RI = 329.90), while the pollution load index (PLI = 2.49) indicated overall high pollution. Pearson correlation analysis showed strong associations among traffic-related metals (Ni-Cr, r = 0.92; Cu-Zn, r = 0.91). PCA and HCA grouped the metals into three clusters, and PMF identified three major sources: industrial traffic mixed emissions, non-exhaust vehicular emissions, and mixed urban deposition. Health risk assessment indicated ingestion as the dominant exposure pathway. Non-carcinogenic risks (HI < 1) were within safe limits; however, carcinogenic risks for Cr (2.84 × 10-4) and Ni (4.16 × 10-4) in children exceeded the acceptable threshold. This study proposes that non-exhaust traffic emissions are the dominant contributors to HMs loading in metro-corridor dust, with children facing elevated long-term carcinogenic risks, highlighting the need for targeted mitigation strategies in urban transport environments.

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PHARMACEUTICAL/TOXICOLOGY

Advances in aluminum oxide nanoparticles-induced neurotoxicity: CNS translocation, phenotypic outcomes, and mechanistic insights

Toxicology letters 2026 Feb 27 · 27 Feb 2026

Aluminum oxide nanoparticles (Al_2O_3 NPs) exhibit remarkable physicochemical properties, making them essential for many applications such as high-temperature refractory materials, catalysis, biomedical uses, and environmental remediation. The extensive use of Al_2O_3 NPs increases the risk of

human exposure through various routes. Owing to their small size, Al2O3 NPs can cross biological barriers and accumulate within the central nervous system (CNS), where they could impair cognitive function and promote neuropathological changes. This review provides a critical, mechanism-oriented synthesis of current evidence on Al2O3 NPs-induced neurotoxicity. We first summarize the principal CNS entry routes, emphasizing size-dependent translocation and the influence of crystalline phase. We then evaluate experimental data on neurobehavioral, neurodevelopmental, and electrophysiological outcomes. Particular attention is given to oxidative stress, mitochondrial dysfunction, neuroinflammation, abnormal protein expression, autophagy, and programmed cell death as key mechanistic pathways. Collectively, data support Al2O3 NPs as plausible neurotoxic hazards, particularly under conditions favoring CNS exposure (small particle size and prolonged or developmental exposure windows). However, interpretation is constrained by incomplete nanoparticle characterization, limited use of realistic exposure scenarios, and insufficient distinction between nanoparticle-specific and dissolved Al^{3+}-mediated effects. On this basis, we propose three priority research directions: phase- and size-dependent neurotoxicity, speciation-informed toxicokinetics, and cell-type-resolved mechanisms linking microglial and astrocytic responses to neuronal injury and neurodegenerative risk. Addressing these gaps will advance mechanistically grounded risk assessment of Al2O3 NPs-related CNS dysfunction.

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Genome-wide screen identifies genes conferring sensitivity to the pollutant bisphenol A in Saccharomyces cerevisiae

Ecotoxicology and environmental safety 2026 Feb 28:312:119883 · 28 Feb 2026

Bisphenol A (BPA), a widely used industrial and agricultural chemical, is a pervasive environmental pollutant with documented harmful effects on ecosystems. Though BPA is known to adversely affect plants, animals, and microorganisms, the precise molecular mechanisms underlying its toxicity remain poorly understood. To address this gap, we investigated the cellular response to BPA using the unicellular eukaryotic model organism Saccharomyces cerevisiae. A genome-wide screen of the diploid single-gene deletion library (comprising 4741 non-essential gene deletion mutants) identified 108 strains exhibiting hypersensitivity to BPA. Functional categorization revealed that the corresponding genes are primarily involved in metabolism, cell cycle regulation, transcription, cellular transport, protein synthesis, and modification. Further analysis via high-performance liquid chromatography demonstrated that 34 of the sensitive mutants accumulated significantly higher intracellular levels of BPA compared to the wild-type strain. Our findings may provide systematic insights into the genetic determinants of BPA sensitivity in yeast and highlight various cellular pathways implicated in its toxic mechanism.

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Structural Analogs TOCP and TPHP Induce Immunotoxicity in Zebrafish through Divergent Disruption of Energy Metabolism

Environmental pollution (Barking, Essex : 1987) 2026 Feb 27 · 27 Feb 2026

Aryl organophosphate flame retardants (aryl-OPFRs), such as triphenyl phosphate (TPHP) and tris(2-methylphenyl) phosphate (TOCP), have been identified as ubiquitous environmental contaminants, but their immunotoxicity and underlying mechanisms in aquatic organisms remain largely unclear. This study investigated the immunomodulatory effects of two structurally analogous aryl-OPFRs using zebrafish larvae during the window of exclusively innate immunity, prior to the maturation of the adaptive immune system. Strikingly, they induced opposite immune responses: TOCP increased macrophage numbers and provoked inflammation, whereas TPHP decreased neutrophil and macrophage counts and suppressed inflammation. This divergence challenges the conventional structure-activity relationship paradigm. Both compounds ultimately compromised antibacterial capacity. To elucidate the mechanism, integrated transcriptomic and metabolomic analyses were

performed, revealing that the opposing phenotypes were linked to differential regulation of energy-related pathways, including glycolysis and purine metabolism. Molecular docking predicted AMPK as a potential common target, while subsequent molecular dynamics simulations provided a critical dynamic perspective: TPHP rapidly dissociated from AMPK, whereas TOCP maintained a more persistent interaction. This differential binding stability offers a plausible mechanistic basis for their distinct immunometabolic reprogramming. Overall, these findings demonstrate that the structural analogs TOCP and TPHP induce divergent immunotoxicity in zebrafish, potentially mediated through a differential disruption of host energy metabolism during the early innate immune phase.

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