CHEMWATCH

TECHNICAL

Week of 2 April 2026

TECHNICAL

CONTENTS

TECHNICAL

OCCUPATIONAL

Evaluating the semi-chronic effects of household air pollution exposure on cardiopulmonary health under two different ventilation conditions

Scientific reports 2026 Mar 28 · 28 Mar 2026

Household air pollution (HAP), particularly from cooking-related particulate matter (PM2.5), poses significant health risks but remains understudied compared to ambient air pollution. We evaluated the short-term cardiorespiratory effects of exposure to cooking-generated PM2.5 and examined the efficacy of automated indoor air quality interventions. Using a crossover design, seven cohorts of two participants each were exposed to two residential conditions over four weeks in a Living Lab: the Standard Control Condition (SCC), featuring basic HVAC, and the Advanced Control Condition (ACC), which included automated range hoods, portable air cleaners and exhaust systems activated by PM2.5 sensors. PM2.5 concentrations were continuously monitored in the breathing zone at the room level. The physiological markers, blood pressure (BP), heart rate (HR), heart rate variability (HRV) and fractional exhaled nitric oxide (FeNO), were measured on the occupant before and after cooking events. Cooking events caused substantial short-term increases in PM2.5 levels, median concentrations rose from < 1 μg/m³ to 263.7 μg/m³ under SCC and to 168.9 μg/m³ under ACC during HRV measurement periods, with exposure levels exceeding WHO 24-hour guidelines up to 82% of the time. Compared to SCC, the ACC significantly reduced PM2.5 exposure ($p < 0.05$). Systolic blood pressure (SBP) decreased significantly post-cooking under ACC (ΔSBP = -3.1 ± 10.0 mmHg) but not in the SCC (ΔSBP = -0.9 ± 8.0 mmHg; $p < 0.05$). HR and HRV showed no statistically significant differences between conditions, though trends in RMSSD, SDNN and LF/HF ratio suggested improved autonomic balance under ACC. HR decreased post-cooking under ACC but increased slightly under SCC (ΔHR = -4.5 ± 6.5 bpm vs. 1.0 ± 1.1 bpm; 95% CI: (-9.8 to -1.2)). FeNO decreased significantly within both conditions pre- to post-cooking, but the difference in reduction between conditions did not reach statistical significance, despite a trend toward greater decline in the ACC. These findings suggest that semi-chronic exposure to cooking-related PM2.5 can adversely affect cardiovascular function, particularly systolic BP and HR, and that automated indoor air quality interventions can meaningfully reduce pollutant exposure and associated physiological impacts. Our results support the implementation of HAP mitigation strategies in residential settings and highlight the need for further research among populations with existing cardiopulmonary conditions.

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A comprehensive review of radon concentrations and annual effective dose amongst workers in copper mines

Radiation protection dosimetry 2026 Mar 29 · 29 Mar 2026

Radon, a carcinogen for lung cancer is chemically inert, colourless, odourless, and a radioactive gas that originates from uranium and thorium radioelements. This review article presents radon concentrations and annual effective dose (Ey) amongst workers in copper mines, as well as examining factors influencing radon levels. Findings indicate varying radon concentration levels ranging from acceptable values to those exceeding the occupational exposure limit of 1000 Bq.m-3. For instance, mean radon concentrations ranged from 6.6 Bq.m-3 to 2400 Bq.m-3 under mechanical ventilation, increasing by a factor of eight under nonmechanical ventilation. Similarly, Ey values ranged from 0.80 mSv.y-1 to above the 20 mSv.y-1 threshold, reaching as high as 34 mSv.y-1. Ventilation emerged as the prominent factor influencing radon concentration, alongside temperature, seasonal changes, and mining activities. The study recommends adequate ventilation and routine radon monitoring to improve air quality and protect workers.

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CHEMICAL EFFECTS

CircRNA0052011 modulates DNA damage and repair via FEN1 ubiquitination to mitigate polystyrene nanoplastics-induced genotoxicity in the lung

Environment international 2026 Mar 23:210:110211 · 23 Mar 2026

Polystyrene nanoplastics (PS-NPs) are one type of environmental pollutants that can penetrate biological barriers and accumulate within cells due to their small particle size. This characteristic raises concerns about their potential respiratory toxicity. However, the impact of PS-NPs on genomic stability and the underlying molecular mechanisms remained unclear. We established an in vitro model of PS-NPs-induced DNA damage based on exposure levels detected in human bronchoalveolar lavage fluid (BALF). In this study, we identified a significantly downregulated circRNA, circRNA0052011, and investigated its regulatory role in PS-NPs-induced DNA damage in the lung. We found that exposure to 20 nm PS-NPs significantly downregulated circRNA0052011 expression, leading to impaired DNA repair and increased γ-H2AX expression, a hallmark of DNA damage. Mechanistically, circRNA0052011 directly interacted with flap endonuclease 1 (FEN1), a key enzyme in the base excision repair (BER) pathway, modulating its ubiquitination and enhancing its protein stability, as revealed by high-throughput sequencing of circRNAs and proteomic analyses. Overexpression of circRNA0052011 effectively mitigated PS-NPs-induced genotoxicity by preserving FEN1 stability and promoting DNA repair. Furthermore, in vivo experiments validated the protective role of circRNA0052011 against PS-NPs-induced lung injury. These findings provide new insights into the molecular mechanisms of nanoplastics-induced DNA damage and suggest circRNA0052011 as a potential biomarker and therapeutic target for environmental pollutant-related diseases.

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Joint effects of prenatal endocrine disrupting chemicals and heavy metal mixture on birth size and maternal complication in the Korea Children's Environmental Health Cohort Study

Environmental research 2026 Mar 26 · 26 Mar 2026

BACKGROUND: Prenatal exposure to mixtures of heavy metals and endocrine-disrupting chemicals (EDCs) may adversely affect birth outcomes, but their joint effects remain understudied.

METHODS: This study analyzed 4,715 Korean mother-infant pairs from the Ko-CHENS cohort. Maternal whole blood was used to measure three heavy metals (lead, cadmium, mercury), while urine samples were collected to quantify 15 non-persistent EDCs, including phthalate metabolites, bisphenols, and parabens. Birth size was assessed using INTERGROWTH-21st z-scores. Weighted Quantile Sum (WQS) regression and Bayesian Kernel Machine Regression (BKMR) were applied to evaluate mixture effects.

RESULTS: Individual chemical analyses revealed significant negative associations between birth weight and MEOHP (β = -0.146, p = 0.034), MCOP (β = -0.057, p = 0.023), MtP (β = -0.032, p = 0.021), and Cd (β = -0.141, p = 0.023). Birth length showed similar patterns with MEOHP and MCOP. Weight-for-length was negatively associated with BPS (β = -0.100, p = 0.008) and Pb (β = -0.253, p = 0.048). WQS analysis demonstrated significant negative mixture effects on birth length (β = -0.052, p = 0.040) and weight-for-length (β = -0.108, p = 0.017). BKMR revealed threshold-dependent relationships, with adverse effects becoming significant when exposure levels exceeded the 50th percentile. Heavy metals emerged as primary drivers of mixture effects, while subgroup analyses showed no significant effects from phthalate or phenol mixtures alone.

CONCLUSIONS: Prenatal exposure to environmental chemical mixtures significantly reduces birth size through threshold-dependent mechanisms. Heavy metals are key contributors to adverse mixture effects, highlighting the need for cumulative risk assessment approaches in environmental health policy.

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Chronic, Environmentally Relevant PM(2.5) Exposure Exacerbates Metabolic Dysfunction-Associated Steatotic Liver Disease and Early-Stage Renal Dysfunction in a Rodent Model

The Kaohsiung journal of medical sciences 2026 Mar 28 · 28 Mar 2026

While epidemiological studies link fine particulate matter (PM2.5) exposure to metabolic dysfunction-associated steatotic liver disease (MASLD) and renal dysfunction, a translational gap exists, as most animal models utilize acute, high-dose exposures that poorly reflect chronic, moderate-level human scenarios. To address this, we established a long-term (seven-month) mouse model (n = 8 per group) combining a Western diet (WD) with a chronic PM2.5 exposure paradigm equivalent to a human exposure of ~50 μg/m3, assessing key biomarkers, organ histopathology, and hepatic gene expression. While the WD was the primary driver of MASLD-related steatosis and insulin resistance, PM2.5 co-exposure acted as a distinct modifier of hepatic pathogenesis. Specifically, the WD + PM group exhibited significantly exacerbated hepatic pathology compared to WD alone, characterized by a ~55% increase in serum ALT ($p < 0.05$) and a 4.7-fold increase in collagen deposition area ($p < 0.05$), alongside the marked upregulation of pro-inflammatory (TNF-α, CCL2) and pro-fibrotic (α-SMA, COL1A1) genes. In the kidney, PM2.5 exposure independently elevated serum creatinine levels (by ~64% vs. controls, $p < 0.05$) and increased the incidence of proteinuria (75% in WD + PM vs. 0% in controls). These functional alterations occurred without inducing major structural damage. This study provides crucial experimental evidence that moderate, chronic air pollution aggravates diet-induced hepatic inflammation/fibrosis and contributes to early-

stage renal dysfunction, underscoring the multi-organ health threats to metabolically vulnerable populations.

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ENVIRONMENTAL RESEARCH

Critical review of manganese sources, pollution, ecotoxicological risks, and mine remediation: Synthesis of a site-specific restoration framework

Journal of environmental management 2026 Mar 27:404:129500 · 27 Mar 2026

Manganese (Mn) enters and is mobilized within the environment (soil, air and water) through anthropogenic (battery manufacturing/recycling unit, mining, and agriculture) and geogenic sources (volcanic emissions and weathering of Mn-bearing rocks), via emission, transport, and redistribution pathways. Higher Mn concentrations in soil and water that exceed prescribed regulatory limits (irrigation water quality standards (FAO, 2023), soil screening levels (USEPA, 2007), and occupational exposure thresholds (Dahlstrom and Bloomhuff, 2014)) are toxic to ecosystems and human health via contamination in various trophic levels. Many previous individual studies attempted to reduce Mn bioavailability and toxicity via various physical, chemical, and biological methods. While prior reviews cover Mn biogeochemistry or toxicity, comprehensive synthesis linking Mn biogeochemistry, ecotoxicology, integrated restoration strategies and risk management is limited. Therefore, the present review summarizes (1) a critical and comprehensive investigation into anthropogenic/geogenic sources of Mn, pollution distribution dynamics across terrestrial/aquatic ecosystems, and human health hazards, (2) reviews physical, chemical, and biological remediation methods and their feasibility, (3) Furthermore, the present review proposes a Site-Specific Ecological Restoration Framework (SSERF), a phased, decision-based, site-adaptive strategy integrating physical, chemical and biological methods for ecological restoration of Mn mines. The review covers specific knowledge gaps and future research directions. Thereby, the present review presents recent knowledge about the fate of Mn in the environment and recent risk management strategies. The findings of the present review provide a valuable foundation for developing innovative and appropriately integrated restoration measures for Mn bioavailability and toxicity in Mn mine settings.

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Seasonal dependence of oxidative toxicity of atmospheric fine particulate matter: A case study of Xi'an, a megacity in Northwest China

Journal of hazardous materials 2026 Mar 26:508:141878 · 26 Mar 2026

The health risks of atmospheric particulate matter (PM) are intrinsically linked to its oxidative potential (OP), while OP exhibits significant seasonal variations, the underlying mechanisms remain unclear. This study investigates the interactions between transition metals and organic components in water-soluble PM using Xi'an, a representative megacity in Northwest China, as a case study to decipher the mechanisms behind its toxicity. We found that while summer PM2.5 concentrations were one-third to one-half of winter levels, its oxidative toxicity exceeded winter levels by more than twofold. This indicates that health risks fundamentally depend on chemical composition rather than mass concentration alone. In summer, total OP from water-soluble matter (OPWSM) primarily

consists of highly polar components, mainly soluble metals. Secondary formation of non-oxidizing organic compounds (e.g., organic sulfates and carboxylic acids) enhances aerosol acidity, further promoting metal complexation and generating an antagonistic effect that suppresses OPWSM. In contrast, winter PM exhibits a synergistic mechanism. When atmospheric oxidation capacity weakens, primary combustion derived humic-like substances (HULIS), especially nitrogen-containing aromatic compounds, significantly contribute to OPWSM (30%) and synergistically enhance OPWSM with soluble metals. This study reveals that metal-organic coupling is a key mechanism driving the seasonal dependence of PM2.5 oxidative toxicity. This finding provides crucial scientific evidence for formulating air quality management policies, promoting a shift from traditional "concentration reduction" to a more targeted "toxicity-oriented" approach. It also establishes a theoretical foundation for implementing seasonally differentiated precision control strategies to more effectively mitigate the health impacts of PM.

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Surface-water PFOA/PFOS in Northern Vietnam: spatiotemporal patterns and a control-prioritization framework

Environmental monitoring and assessment 2026 Mar 30;198(4) · 30 Mar 2026

Perfluorooctanoic acid (PFOA) and perfluorooctanesulfonate (PFOS) are persistent PFAS of growing regulatory concern, yet robust spatiotemporal evidence in Vietnam remains limited. Here, we developed a 12-month surface-water monitoring dataset with triplicate sampling across an ≈45-site lake-river-estuary-coastal network in Northern Vietnam, quantified PFOA/PFOS by LC-MS/MS, and translated the observations into a control-prioritization framework. Across pooled station-month observations (n = 552), PFOA showed a median of 7.56 ng/L (p95 = 30.95), while PFOS was higher with a median of 16.55 ng/L (p95 = 107.77). Concentrations consistently increased along the basin transport-retention continuum, with stable hotspots in downstream and estuarine nodes (e.g., Ba Lat, Cua Day, Do Son, Sam Son, Tra Co) and the water-body hierarchy estuaries > river (downstream) > coastal > lakes. Clear seasonality was observed, with PFOS peaking in January/December and PFOA peaking in February/December. Mechanistic interpretation combined field covariates with water-particle-DOC partitioning concepts, adsorption-desorption tests using reference sediment, and multivariable modeling, consistently identifying suspended solids and dissolved organic carbon as dominant drivers, especially for PFOS. Ecological screening using risk quotients (RQ) indicated PFOS-driven concern at priority downstream/estuarine nodes. In parallel, integrated exposure scenarios (drinking water and fish consumption) suggested seafood intake can dominate PFOS exposure, yielding tail risks for children and high-seafood consumers even when mixture-level indices remain generally acceptable under baseline scenarios.

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PHARMACEUTICAL/TOXICOLOGY

PFOS-mediated suppression of SOX30 impairs DNA double-strand break repair leading to male reproductive dysfunction

Ecotoxicology and environmental safety 2026 Mar 28:314:120081 · 28 Mar 2026

Perfluorooctanesulfonic acid (PFOS), a persistent organic pollutant and representative per- and polyfluoroalkyl substance (PFAS), is ubiquitously detected in the environment and human tissues, and mounting evidence has implicated it in male reproductive dysfunction. However, the direct impact of PFOS on meiosis in spermatocytes and the underlying molecular mechanisms remain poorly understood. In this study, by combining chromosome spreading with high-resolution microscopy analysis of spermatocyte subtypes and morphology, we revealed that PFOS exposure decreased the proportion of pachytene spermatocytes and induced abnormal DNA double-strand break (DSB) repair, ultimately leading to a reduction in sperm density. Mechanistically, PFOS exposure suppressed the expression of SOX30, a germ-specific transcription factor, leading to downregulation of its downstream targets RPA2 and RAD51-key proteins of homologous recombination repair (HRR). This suppression resulted in unrepaired DSBs, triggering the pachytene checkpoint and leading to spermatocyte apoptosis. Critically, overexpression of SOX30 in PFOS-exposed GC2 spermatocyte cells restored RPA2/RAD51 expression and rescued DSB repair capacity. These findings identify SOX30 as a critical mediator of PFOS-induced meiotic DSB repair defects and establish it as a potential therapeutic target for mitigating PFAS-associated male infertility. This study provides novel insights into the molecular link between environmental pollutant exposure and impaired meiosis, offering a foundation for developing preventive and therapeutic strategies against PFOS-induced reproductive toxicity.

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Fibrous Zeolites and Pulmonary Fibroblasts: Toxicological Impact and EPR-Based Insights into Cellular Alterations

Environmental pollution (Barking, Essex : 1987) 2026 Mar 26 · 26 Mar 2026

This study investigates the potential cytotoxicity of the carcinogenic erionite and other fibrous zeolites, including mordenite, ferrierite and scolecite. Their interactions with human lung fibroblast cells (MRC-5) were evaluated using biological assays combined with Electron Paramagnetic Resonance (EPR) spectroscopy. All fibrous zeolites were found to interact with lung fibroblasts, leading to increased intracellular ROS levels and alterations in the lysosomal compartment after 24 hours of exposure. EPR measurements after CAT16 incubation provided further insight into cellular membrane interaction dynamics, revealing notable differences in spectral intensity and hyperfine coupling constants, which suggest distinct interaction mechanisms among the fibers. Erionite showed the highest radical solubilization, implying potential cytotoxic effects. Prolonged exposure to mordenite and ferrierite also significantly reduced cell viability, at levels comparable to erionite. These findings highlight key factors involved in early inflammatory responses and their potential contribution to the development of long-term chronic diseases and cancer. The biological effects of fibrous zeolites are controlled by a multifactorial interplay between morphology, surface area, chemical composition, and biopersistence, which together determine the extent of membrane interaction, oxidative stress generation, and intracellular processing, ultimately governing cytotoxicity and potential carcinogenicity. This study therefore provides an initial step toward understanding the biological mechanisms underlying the toxicity and carcinogenic potential of fibrous zeolites, improving awareness of their potential human health risks.

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