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Week of 5 June 2026

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OCCUPATIONAL

Impact of preventive measures on welding fume exposure among welders, robot operators, and bystanders: Air and biomonitoring

International journal of hygiene and environmental health 2026 May 31:276:114830 · 31 May 2026

METHODS: Welding aerosol was characterized by size distributions and number concentration, size-fractionated mass and metal composition, and morphology. Exposure to respirable aerosol and metals was measured at baseline and follow-up after company-led preventive measures. Different local exhaust ventilation solutions and the effective protection factor of powered air-purifying respirators were evaluated. Metals in blood and urine were assessed in exposed workers sampled on Monday and Friday at baseline and follow-up, and in an unexposed reference group.

RESULTS: Welding aerosol was dominated by ultrafine particles, and substantial far-field exposure highlighted potential second-hand risks. At baseline, manual welders were highly exposed, with median Mn concentrations exceeding the EU occupational exposure limit (Company 1: 68.6 µg/m³; Company 2: 146.6 µg/m³). Robot operators (Company 1: 15.3 µg/m³; Company 2: 25.2 µg/m³) and bystanders (Company 1: 10.3 µg/m³; Company 2: 16.8 µg/m³) also experienced notable exposure. Following preventive measures, Mn exposure decreased, particularly among manual welders at Company 2 (from 146.6 to 13.3 µg/m³; a 91 % reduction). Median Mn levels were below limit values for all groups at follow-up. Urinary Mn broadly reflected these trends. Powered air-purifying respirators provided low effective protection and point exhaust ventilation was more efficient than alternative solutions.

CONCLUSION: Comprehensive preventive measures reduced welding aerosol exposure across worker groups. Continued improvements are needed to ensure long-term protection for welders and bystanders.

OBJECTIVE: This study assessed occupational exposure to mild steel welding fumes among manual welders, welding-robot operators, and bystanders in two Swedish manufacturing companies, and evaluated the impact of preventive measures.

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Characterization of volatile organic compounds emissions and health risk assessment in coking industry: A case study in East China

Journal of environmental sciences (China) 2026 Jul:165:824-835 · 1 Jul 2026

Volatile organic compounds (VOCs) represent a critical category of air pollutants, and the emissions from anthropogenic sources are essential for understanding and managing VOC pollution and the

resultant ozone (O₃) pollution. Several research initiatives have evaluated VOC emissions from significant industrial sources in China; however, knowledge regarding VOC emissions in the coking industry is still insufficient. The investigation examines the characteristics of VOC emissions from the coking industry through detailed unit-based field sampling, constructs emission source profiles, identifies provincial VOC emission characteristics, and assesses potential health risks to workers. Experimental results indicate that the primary categories of VOC emissions in the coking section are alkanes and alkenes, whereas aromatics and oxygenated VOCs predominate in the gas purification and sewage treatment sections. The fingerprint VOC species in these sections are n-pentane, benzene, and i-propanol, respectively. The highest emission factor occurs in the coking oven unit. The ozone formation potential research indicated that the coking oven and the condensing and blasting units significantly influence O₃ formation, with propene being the predominant contributor. Additionally, the health risks associated with different physical work intensities for occupational workers are examined, indicating that heavy physical labor can cause serious carcinogenic risks to workers. Benzene is a predominant carcinogenic hazard, whereas 1,1,2-trichloroethane is the primary non-carcinogenic VOC species in coking industry. This study provides a comprehensive understanding of the pollution profiles and environmental risks of VOCs discharged from the coking industry, informs the implementation of VOC regulations, and promotes cleaner production.

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CHEMICAL EFFECTS

Long-term exposure to polystyrene microplastics exacerbates seizure symptoms via lipid metabolic disruption and ferroptosis: insights from multi-omics analyses

Journal of nanobiotechnology 2026 May 29 · 29 May 2026

As a consequence of global industrial growth, microplastics (plastic fragments < 5 mm) have become ubiquitous environmental contaminants, prompting serious questions about their impact on human health. Beyond the established risks of ingestion, the inhalation of these airborne particles is now a primary focus, especially regarding potential effects on the neurological system. Emerging evidence from laboratory and animal models shows that inhaled microplastics can penetrate the brain. Once there, they can trigger a cascade of harmful effects, including neuroinflammation, oxidative stress, and deficits in learning and memory. Among the general population, children are uniquely vulnerable to microplastic exposure due to their developing physiological systems, which are particularly susceptible to the chemical and physical hazards posed by these particles. Nevertheless, current scientific understanding of the health consequences of microplastic exposure remains limited, with a substantial knowledge gap concerning the long-term effects of respiratory microplastic exposure on pediatric populations, particularly those with pre-existing neurological conditions such as epilepsy. This study investigates the relationship between chronic respiratory exposure to polystyrene microplastics and seizure severity, with the aim of establishing a potential exposure-metabolite-gene regulatory network. We hypothesize that prolonged respiratory microplastic exposure induces systemic oxidative stress and inflammation, which subsequently disrupts lipid metabolism, alters gene expression profiles, triggers ferroptosis, and ultimately exacerbates seizure manifestations. This study highlights three key findings. First, chronic respiratory microplastic exposure induces systemic oxidative stress and inflammation, posing substantial health risks. Second, integrated metabolomics and Mendelian randomization analyses reveal that this exposure disrupts lipid metabolism, with metabolic perturbations strongly associated

with ferroptosis activation and increased seizure severity. Third, multi-omics approaches coupled with in vivo validation confirm that microplastics disrupt lipid homeostasis, dysregulate ferroptosis-related gene expression, and exacerbate seizure manifestations. Notably, our data identify melatonin as a promising therapeutic candidate for mitigating these adverse effects. Collectively, these findings substantially advance the understanding of microplastic-induced neurotoxicity and reveal actionable molecular targets for potential therapeutic interventions.

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Development of nontarget method based on GC-QTOF-HRMS for analyzing organic pollutants in human serum

Journal of environmental sciences (China) 2026 Jul:165:379-386 · 1 Jul 2026

Traditional targeted analyses often overlook unknown or emerging contaminants, highlighting the significance of nontarget and suspect screening approaches. A novel and high-sensitivity methodology for nontarget analysis of organic pollutants in human serum was newly-developed based on gas chromatography coupled with quadrupole time-of-flight high-resolution mass spectrometry. The extraction protocol employing an acetonitrile-ethyl acetate (9:1, V:V) mixture significantly improved the extraction efficiency while minimizing matrix effect. A hybridized analytical strategy integrating nontarget and suspect screening was developed to achieve comprehensive identification and classification of pollutants, employing the National Institute of Standards and Technology (NIST) 20 library and Agilent Technologies Personal Compound Database and Library (PCDL). This approach successfully characterized 273 organic contaminants spanning 12 categories, including polycyclic aromatic hydrocarbons (PAHs) and their derivatives, esters, and phenolic compounds in human serum, with a significant increase in detection specificity compared to conventional workflows. The methodology used serum samples of the workers from coking industry, revealing widespread contamination dominated by PAHs and PAH derivatives. Among the target analytes, three were identified solely by NIST and six solely by PCDL, indicating the complementary benefits of combining these different databases. Notably, this work reported the first confirmed detection of 2-naphthalenamine in human serum. This optimized approach demonstrates enhanced sensitivity and reliability in serum analysis, advancing biomonitoring capabilities and providing a deep understanding of human exposure to environmental pollutants.

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Aristolochic acid and the risk of female lung cancer: Population-based case-control study in Taiwan

Cancer epidemiology 2026 May 30:103:103131 · 30 May 2026

BACKGROUND: Aristolochic acid (AA) is a known human carcinogen that induces DNA adducts and lung tumors in rodents exposed to it. SBS22a, linked to aristolochic acid, appeared almost exclusively in Taiwanese never-smoking lung cancer patients. As there are relatively few female smokers in Taiwan, the objective of this study is to examine the potential risk of lung cancer in women who consume herbs containing AA.

METHODS: A case-control study based on the population was conducted, where cases were individuals newly diagnosed with lung cancer (ICD-9 162) between January 1, 1999, and December

31, 2013. Logistic regression was utilized to evaluate the risk of lung cancer in relation to the total dose of AA-containing herbs and the estimated cumulative dose of AA.

RESULTS: Women who used Chinese herbal products containing aristolochic acid (AA) had a slightly increased risk of lung cancer compared with non-users (adjusted OR: 1.08; 95% CI: 1.04-1.12). Increased risks were observed across several duration-of-use categories, with a significant trend for duration of estimated AA exposure. However, no clear dose-response relationship was identified for individual AA-containing herbs or estimated cumulative AA dose. Several comorbidities and medications, including chronic obstructive pulmonary disease, tuberculosis, pneumococcal pneumonia, human papillomavirus infection, alcohol-related disease, hyperlipidemia, hormone replacement therapy, and non-steroidal anti-inflammatory drug use, were also associated with increased lung cancer risk.

CONCLUSIONS: The study found that consumption of herbs containing aristolochic acid was associated with a modestly increased risk of lung cancer in women. However, the observed association was small, and no clear dose-response relationship was identified. Therefore, the findings should be interpreted cautiously given the potential for residual confounding and limitations in exposure assessment.

IMPACT: In a low-smoking population where the aristolochic acid-associated mutational signature (SBS22a) has been observed predominantly in Taiwanese never-smokers, this nationwide case-control study suggests a possible association between AA-containing herbal products and lung cancer risk in women. Further studies with improved exposure characterization and molecular validation are needed to clarify the role of AA exposure in lung carcinogenesis.

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ENVIRONMENTAL RESEARCH

Reemissions of polycyclic aromatic compounds from soil to air in chemical industrial parks

Environmental research 2026 May 30 · 30 May 2026

Air-soil exchange is a critical process controlling the fate of toxic organics such as polycyclic aromatic compounds (PACs), yet it remains poorly characterized in chemical industrial parks. This study investigated the air-soil exchange of PACs in a coking park and a petrochemical park, two major industrial sources of PAC emissions, using paired passive air and soil samples collected in summer and winter. Significantly different levels, source profiles, and spatial distributions of PACs in air and soil were observed between the two parks. Volatilization tendency, assessed by fugacity fractions, was stronger in the coking park than in the petrochemical park, and stronger inside the parks than at the control sites. This spatial heterogeneity was driven by both temperature and the air-to-soil concentration ratio (C_a/C_s), with temperature as the primary driver; notably, the effect of C_a/C_s became non-negligible in winter, revealing a seasonal shift in the relative importance of the two drivers. High-molecular-weight PACs showed greater sensitivity of the volatilization tendency to temperature changes. Flux calculations showed that soils in both parks acted as secondary sources of atmospheric PACs in winter and summer, which indicates that chemical industrial park soils may act as secondary sources of atmospheric PACs, and their management should therefore take soil volatilization into account.

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Residential radon exposure and lung cancer histology and stage: a population-based ecological study in Central Germany

Journal of cancer research and clinical oncology 2026 May 31;152(6) · 31 May 2026

PURPOSE: Residential radon exposure is recognised as one of the leading environmental risk factors for lung cancer and acts synergistically with tobacco smoking. Thuringia, Germany, exhibits high geogenic radon potential; however, population-level associations with lung cancer histology and stage at diagnosis remain insufficiently characterised.

METHODS: We conducted a retrospective ecological analysis of 893 lung cancer cases (2018-2022) from the Thuringian State Cancer Registry. Cases from 96 communities were classified as high- or low-radon exposure based on geogenic radon potential and soil radon activity. Demographics, histology and stage were compared between groups.

RESULTS: Population-level lung cancer incidence did not differ significantly between low- and high-radon communities, consistent with age- and sex-adjusted Poisson regression showing no association with residential radon exposure (adjusted IRR 1.05, $p = 0.473$). However, all cases in women younger than 50 years occurred in high-radon communities. Adenocarcinoma was nominally more frequent (56% vs. 48%, $p = 0.04$), and among patients with small cell lung cancer (SCLC), extensive-stage disease was more common (64% vs. 49%, $p = 0.04$), with combined SCLC occurring exclusively; however, these findings were not significant after correction for multiple testing.

CONCLUSION: In this ecological analysis, residential radon exposure was not associated with annual lung cancer incidence but was linked to tumour histology and stage at diagnosis. Given the ecological design and lack of smoking and occupational exposure data, these findings are hypothesis-generating and warrant further investigation to clarify radon's role in lung cancer biology and prevention.

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Machine learning identifies traffic-related pollutant mixtures as potential factors suggestive of association with osteoporosis in a cross-sectional analysis of NHANES

Medicine 2026 May 29;105(22):e48884 · 29 May 2026

The combined effect of traffic-related pollutant mixtures on osteoporosis (OP) remains unclear. This study aimed to evaluate such associations using machine learning and mixture modeling approaches. A cross-sectional analysis was conducted among 3053 participants from the National Health and Nutrition Examination Survey 2015 to 2018. Eighteen exposures were assessed, including heavy metals, polycyclic aromatic hydrocarbons, phthalates, per- and polyfluoroalkyl substances, volatile organic compounds, diesel exhaust, and behavioral factors. OP was defined by femoral neck bone mineral density T-score ≤ -2.5 . Weighted quantile sum regression and 5 machine learning models were applied, accounting for complex survey design. OP prevalence was 12.97%. Weighted quantile sum regression showed a positive but nonsignificant mixture odds ratio (odds ratio = 1.21; 95% confidence interval: 0.77-2.63; $P = .412$). The largest positive weights were daily outdoor work

duration (0.240) and occupational diesel exhaust (0.136). Random forest identified female sex, age, vitamin D, blood lead, and body mass index as top predictors. Least Absolute Shrinkage and Selection Operator achieved the highest test area under the receiver operating characteristic curve (0.81) with the lowest cross-validation standard deviation (0.0167). Its coefficients highlighted occupational diesel exhaust ($\beta = 0.40$) as a notable environmental factor alongside female sex ($\beta = 0.95$) and age ($\beta = 0.70$). Traffic-related pollutant mixtures showed a cross-sectional association with OP in a nationally representative US population, with occupational diesel exhaust and prolonged outdoor work emerging as key contributors in mixture models. However, the nonsignificant overall mixture effect and potential for reverse causation warrant caution in interpretation. These findings support considering environmental and occupational factors in OP prevention, although the overall mixture effect did not reach statistical significance.

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PHARMACEUTICAL/TOXICOLOGY

Molecular Mechanisms of Manganese Oxide Nanoparticles Toxicity in Brain and Other Tissues: An Overview

Frontiers in bioscience (Landmark edition) 2026 May 12;31(5):47103 · 12 May 2026

The use of manganese oxide nanoparticles (MnOxNPs) in biomedicine increases the risk of their accumulation in the body, potentially leading to toxicity in various organs and tissues. In addition, occupational exposure to MnOxNPs-containing aerosols may also occur. MnOxNPs have been shown to accumulate in the brain and induce neurobehavioral alterations. However, the specific mechanisms of MnOxNPs toxicity in the brain and other tissues remain incompletely understood. Therefore, the objective of this review is to summarize existing data on the toxicity of MnOxNPs in the brain and other tissues, and to discuss the molecular mechanisms underlying their neurotoxic effects. It has been shown that MnOxNPs induce neuronal death through induction of mitochondrial dysfunction and subsequent apoptosis, and overaccumulation of tau protein and amyloid- β . Neurotoxic effects of MnOxNPs may also be mediated by blood-brain barrier disruption, and dysregulation of dopaminergic and glutaminergic signaling. Exposure to MnOxNPs induces neuroinflammation through activation of nuclear factor kappa B (NF- κ B) and p38 mitogen-activated protein kinase (p38 MAPK) pathways in a reactive oxygen species-dependent manner. In vitro studies further demonstrate that MnOxNPs exhibit a dose-dependent cytotoxic effects in alveolar macrophages, as well as in respiratory, colonic, and other epithelial cells, through the promotion of oxidative stress and an inflammatory response. Overexposure to MnOxNPs has significant nephrotoxic, hepatotoxic, and immunotoxic effects, as well as affecting the reproductive system. Smaller particles exhibit more pronounced toxic effects in the brain and other tissues than larger nanoparticles or microparticles. However, the mechanisms underlying the different toxicities of MnOxNPs of different sizes, shapes, and surface modifications remain unclear. These observations highlight the potential of MnOxNP exposure to contribute to neurological disorders and dysfunction of other systems, underscoring the need for further mechanistic studies to ensure their safe application in biomedicine.

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Cell Painting phenomics reveals size-dependent phenotypic responses to titanium dioxide nanoparticles in HepG2 cells

Toxicology 2026 May 30:526:154512 · 30 May 2026

Titanium dioxide nanoparticles (TiO₂NPs) are widely produced engineered nanomaterials with ongoing human exposure through consumer and occupational uses. Conventional in vitro assays often focus on cytotoxicity and may therefore overlook early or sublethal cellular perturbations. Here, we applied Cell Painting-based phenomics to resolve size-dependent sub-lethal phenotypic signatures of TiO₂NP exposure in human HepG2 hepatocytes. Two TiO₂NPs (<25 nm and <100 nm) were characterized by field emission scanning electron microscopy and evaluated following 24-hour exposure at five concentrations: 6.25, 12.5, 25, 50, and 100 µg/mL. Cell viability was assessed using the alamarBlue assay, and high-dimensional phenotypic profiles were generated using Cell Painting-based phenomics, including automated high-content imaging and CellProfiler-based feature extraction. TiO₂NP exposure induced modest reductions in viability at the highest concentration, indicating limited acute cytotoxicity. In contrast, phenomic profiling revealed clear, concentration-dependent phenotypic perturbations for both size fractions, with markedly stronger and more consistent effects for the < 100 nm TiO₂NPs. At 100 µg/mL, the < 100 nm TiO₂NPs altered 50.9% of the measured phenotypic features, compared with 28.9% for the < 25 nm particles, with prominent contributions from endoplasmic reticulum-, actin/Golgi/plasma membrane-, mitochondria-, and RNA-associated features. Dimensionality reduction and correlation analyses confirmed reproducible, concentration-dependent phenotypic trajectories. Importantly, the TiO₂NP-induced phenotypes were distinct from those induced by the reference chemical CA-074Me, which produced broad perturbations and served as a reference chemical to verify assay sensitivity and dynamic range. Overall, Cell Painting phenomics sensitively captures size-dependent, sublethal cellular phenotypes induced by TiO₂NPs, supporting its value as a New Approach Methodology for nanosafety assessment beyond conventional viability endpoints.

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