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Week of 1 May 2026

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OCCUPATIONAL

Occupational exposure and respiratory health effects among electronic waste (e-waste) recycling workers in Bangladesh

International journal of environmental health research 2026 Apr 26 · 26 Apr 2026

In a cross-sectional study, we measured concentrations of heavy metals and assessed respiratory symptoms and lung function in 199 e-waste recycling and 104 non-recycling workers. Exposed workers had higher median blood Pb, similar Cd, and lower total hair Hg levels than non-exposed workers. The frequency of respiratory symptoms, such as phlegm (53% vs 34%) and wheeze (10% vs 3%) were higher in exposed than in non-exposed workers (both $p \leq 0.05$). Forced vital capacity (FVC) did not differ across the groups (74 vs 74% predicted), however forced expiratory volume in 1 s (FEV1, 75.8% vs 78% predicted), FEV1/FVC (103.7% vs 106.9%) and peak expiratory flow (PEF, 61% vs 72% predicted) were significantly lower among exposed workers. Adjusted estimates revealed that e-waste exposure significantly associated with wheeze (AOR = 6.2, 95% CI: 1.68, 22.93) and the median FEV1 and PEF were 6% and 10% unit lower, respectively, in the exposed workers than non-exposed workers. Pb concentration was significantly associated with 1.08% predicted unit decrease in PEF (95% CI: -18.85, -1.15). No significant mediation effect observed between exposure and outcomes relationships. The data implies that workers who are exposed to e-waste are at high risk of wheeze and lung function impairment, which requires preventive actions to mitigate exposure.

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Provocation of occupational asthma by non-specific irritation and its relevance for the classification of chemicals as respiratory allergens: a case study of methyl methacrylate

Regulatory toxicology and pharmacology : RTP 2026 Apr 23 · 23 Apr 2026

Allergic sensitisation of the respiratory tract by chemicals, commonly associated with asthma, is an important adverse health effect. However, the accurate identification of true chemical respiratory allergens remains problematic. In the absence of fully validated predictive test methods, or a universally agreed adverse outcome pathway, it is commonly the case that regulatory classification of chemicals as respiratory allergens relies solely upon clinical data that frequently fails to distinguish between irritant-provoked bronchial responses and true allergic sensitisation. Here this issue is explored using as a case study methyl methacrylate (MMA). In a regulatory context this chemical has been proposed for classification as a respiratory sensitiser on the basis of specific inhalation challenge data in humans. A review of the clinical data on which the proposal is based

reveals that they are unsuitable for accurate discrimination between allergic and non-specific irritant mechanisms. Furthermore, the available information supports that MMA lacks respiratory sensitising activity. This conclusion is supported by review of experimental data, derived from both animal studies, and recently published in vitro assessments, that individually and collectively indicate that MMA is not a respiratory sensitiser. It is concluded that accurate regulatory classification of chemical respiratory allergens cannot rely solely on clinical studies.

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CHEMICAL EFFECTS

PFOA Exposure Elicits Quantitative Lipidomic Changes in the Pancreas in a Mouse Model of Pancreatic Cancer

Environmental toxicology 2026 Apr 24 · 24 Apr 2026

Pancreatic cancer is the fourth leading cause of cancer deaths in the US and predicted to rise to second by 2030. Several risk factors have been identified as potential contributors to pancreatic cancer development including lifestyle factors and long-term exposure to occupational and environmental carcinogens. In mice, exposure to perfluorooctanoic acid (PFOA) leads to diabetic outcomes and pancreatic cancer, while epidemiologic studies have linked PFOA exposure to type 1, type 2, and gestational diabetes, obesity, and pancreatic cancer. While multiple studies have evaluated changes in serum lipid levels in association with PFOA exposure, none to date have evaluated PFOA-associated lipid alterations in the pancreas. Using the LSL-KRasG12D; Pdx-1-Cre (KC) mouse model of pancreatic cancer, we report upregulation of lipid species within the acylcarnitine (CAR) and ceramide (Cer) lipid groups and downregulation of lipids in the phosphatidylcholine, phosphatidylethanolamine (PE), and phosphatidylserine groups following exposure to 1 and 5 ppm PFOA for 6 months. The most upregulated lipids following PFOA exposure included individual Cer and CAR species while PE species comprised the top group of downregulated lipids. Thromboxane B2 (TXB2), a proinflammatory eicosanoid, was the most upregulated lipid in the 1 and 5 ppm KC treatment groups with a fold-change of ~12-fold and ~31-fold in the 1 and 5 ppm KC treatment groups, respectively. As these lipid alterations have been linked to mitochondrial dysfunction, obesity, and inflammation, our results suggest that PFOA-induced lipid alterations may be involved in adverse health outcomes including diabetes and pancreatic cancer.

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Lipid clues: Decoding the effects of endocrine-disrupting chemicals on female reproductive health

Ecotoxicology and environmental safety 2026 Apr 25:316:120173 · 25 Apr 2026

Multiple epidemiological studies across countries have linked exposure to endocrine-disrupting chemicals (EDCs) with adverse reproductive outcomes; however, the underlying mechanisms remain incompletely characterized. In this study, we profiled EDCs, lipids, and fatty acid metabolites in follicular fluid (FF) to elucidate metabolic pathways through which EDCs may affect reproductive outcomes. We further integrated network toxicology with an adverse outcome pathway (AOP) framework to delineate mechanistic routes by which EDCs may impair female reproductive health via metabolic disruption. We measured EDCs and metabolites in FF, and assessed assisted

reproductive outcomes in 173 women from the SEARCH cohort. Overall, associations between EDCs and lipid or fatty acid profiles varied by chemical and class, consistent with previous studies. We further observed evidence of oocyte lipid metabolism dysregulation in relation to EDC exposure. Specifically, the diacylglyceride (DG) and phosphatidylethanolamine (PE) → phosphatidylcholine (PC) and lysophosphatidylcholine (LPC) pathway was upregulated. Poorer oocyte developmental outcomes were characterized by significantly lower PE and LPE levels (log₂FC: -0.30 and -0.07, respectively), and PE and LPE levels were associated with oocyte development (β : 0.141 and 0.090, respectively). Notably, phthalate exposure was associated with lower LPE (β [95% CI]: -0.001 [-0.002, 0.000]). Network and adverse outcome pathway (AOP) analysis identified mitochondrial dysfunction as a central mechanism mediating the association between EDC exposure and impaired fertility. In conclusion, these findings provide novel molecular insights into environmental determinants of female reproductive success.

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ENVIRONMENTAL RESEARCH

Review of Organophosphorus Herbicide: Distribution of Residues, Toxicity Effects, Mechanisms, and Developing Trends Studies

Journal of applied toxicology : JAT 2026 Apr 26 · 26 Apr 2026

Organophosphorus herbicides play an important role in agricultural development and are widely used worldwide. Glyphosate, glufosinate, DMPA, amiprofos-methyl, butamifos, piperophos, and other organophosphorus herbicides are overused worldwide. Organophosphorus herbicides are detected in different environmental media and play a toxic role in causing ecological health risks, causing serious concern among people. This review discusses the concentration levels of organophosphorus herbicides in water, soil, plants, and other environmental media and finds that the content of organophosphorus herbicides in soil is high, the concentration range is 12-129 $\mu\text{g/g}$, and finally summarizes the concentration levels of organophosphorus herbicides in organisms and human beings and finds that the concentration of organophosphorus herbicides in pesticide industry workers is relatively high. From the point of view of detection methods, the traditional methods and advanced technologies of herbicide detection are discussed in detail, and the advantages and disadvantages of these methods are pointed out. Finally, the ecological health risks, toxic effects, and molecular mechanisms of organophosphorus herbicides are discussed. Epidemiological investigations in China, Africa, and America showed that organophosphorus herbicides were prone to induce neurotoxicity, genotoxicity, and metabolic toxicity. These findings provide a theoretical basis for environmental management and ecological health risk assessment of organophosphorus herbicides.

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Interplay of Chemical Toxicants and Urban Microenvironments in the Oxidative Potential and Comprehensive Risk of Road Dust

Environmental research 2026 Apr 22 · 22 Apr 2026

The oxidative potential (OP) of urban road dust PM_{2.5} poses major health implications, primarily driven by key toxicants including heavy metals and polycyclic aromatic compounds (PACs). This

study systematically measured toxics components in road dust across commercial, traffic, and residential urban functional areas in the Guanzhong Plain. Interpretable machine learning (XGBoost-SHAP), land-use regression (LUR), and a sources-pathways-receptors (S-P-R) framework were applied together to quantify drivers of OP, linking contaminant patterns to urban functional area. All areas exhibited high ecological risks from PACs and metals. Spatially, heavy metal was universally driven by non-exhaust traffic emissions, while dense e-waste and electronic repair activities created extreme Co and Ni hotspots in commercial areas. For PACs, primary tailpipe exhaust dominated traffic areas, whereas commercial areas were more influenced by congested idling traffic and high-temperature catering. Machine learning revealed that specific PACs strongly drive OP, which synergistically coupled with co-occurring transition metals to amplify oxidative toxicity. LUR models further identified that high OP in traffic areas was primarily driven by estimated vehicle number and transportation facilities, and elevated OP in residential areas were closely associated with catering and residential activities. Using the S-P-R framework, this study found that sources (47.1%), pathways (21.2%) and receptors (31.8%) jointly determined road dust health risk. Commercial areas showed the highest mean comprehensive risk (CR = 0.257), but traffic areas exhibited more widespread high risk hotspots driven chiefly by OP and traffic-related exposure pathways, while residential areas were distinguished by concentrated sensitive receptors, e.g., kindergartens, schools, green spaces. This study provides a novel assessment system and highlights the interactions between urban functional areas and road dust toxicity, underscoring the need for targeted strategies to mitigate urban dust pollution.

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Geospatial assessment of groundwater toxicity from hazardous environmental contaminants and background concentration in quaternary aquifers of a semi-arid region

Environmental geochemistry and health 2026 Apr 25;48(7) · 25 Apr 2026

Heavy metal contamination in groundwater has significantly increased over the past decade due to increased anthropogenic activities in the environment. In particular, the contamination of groundwater resources due to industry waste disposal with archaic treatment techniques has resulted in severe risks to the well-being of flora and fauna in the industrial regions. In this study, groundwater samples were collected from 98 separate sites in the Mathura region of India during pre-monsoon and post-monsoon seasons and were analyzed for heavy metal exposure to different age and gender groups using Monte-Carlo simulation. Statistical tests and pollution indices were also adopted to understand the distribution of heavy metals in the study region. The descriptive analysis of results revealed that the concentration of heavy metals is in the order Pb > Fe > Cr > Ni > Zn > Mn > Cd > Cu for the pre-monsoon season and Pb > Ni > Cr > Zn > Fe > Mn > Cd > Cu for the post-monsoon season. Adopted pollution indices also indicate increased heavy metal pollution after seasonal rainfall. The study discusses the complex nature of heavy metal contamination in the study area according to the desirable and permissible limits of BIS and emphasizes the preparation of countermeasures against cancer risks across specific sites of the study region through geospatial analysis, and recommends the necessity of enhanced treatment of groundwater prior to ingestion and disposal.

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Comprehensive overview of triclosan neurotoxicity and construction of adverse outcome pathways using a systems toxicology approach: Triclosan-induced attention-deficit hyperactivity disorder as an example

Ecotoxicology and environmental safety 2026 Apr 24:316:120169 · 24 Apr 2026

Triclosan (TCS) is widely applied to daily necessities as a chemical bacteriostatic agent. Environmental TCS levels have increased significantly following the coronavirus disease 2019 pandemic, posing a potential threat to humans and ecosystems. Epidemiological investigations and toxicological studies have shown that long-term TCS exposure can damage human tissues and organs and induce neurodevelopmental disorders in offspring. However, studies on the mechanisms underlying its neurotoxicity and toxicity risk assessments are limited. This review summarizes the status of environmental and human TCS exposure and systematically outlines its neurotoxic effects. To further elucidate the mechanisms underlying TCS neurotoxicity, using TCS-induced attention-deficit hyperactivity disorder (ADHD) as an example, we constructed an adverse outcome pathway (AOP) framework based on a systematic toxicology approach. We found that TCS increased the expression of cannabinoid receptor 1, which activates the "neuroactive ligand-receptor interaction" pathway, leading to ADHD-like behaviors, including cognitive, learning, and memory deficits, by modulating chemical synaptic transmission and neurotransmitter levels. The AOP framework was further used to assess the associated neurodevelopmental toxicity risks of TCS, contributing to a better understanding of its characteristics and safety. Thus, future research on the mechanisms underlying TCS toxicity and issues related to its detection and regulation should be emphasized.

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ZIP14-VDAC2 mediates the transport of extracellular iron into mitochondria in hepatocytes under PFOS exposure

Chemico-biological interactions 2026 Apr 24:434:112108 · 24 Apr 2026

Perfluorooctane sulfonate (PFOS), classified as a persistent organic pollutant, promotes mitochondrial iron overload through voltage-dependent anion channel 2 (VDAC2). However, the source of iron transported through VDAC2 remains unclear. Here, we reported that PFOS enhanced extracellular iron influx into human hepatocytes HepG2, which peaked at 6 h. Under PFOS exposure, total VDAC2 protein levels remained unchanged, whereas its expression increased in mitochondrial lysates but decreased in cytoplasmic lysates, which were observed both in HepG2 cells and mice liver. Meanwhile, the total protein expression of the extracellular iron importer ZRT/IRT-like protein 14 (ZIP14) was upregulated, whereas absent from the mitochondrial lysates. Knockdown of either VDAC2 or ZIP14 inhibited PFOS-induced extracellular iron influx and mitochondrial iron overload, but did not affect cytoplasmic iron levels. Notably, immunofluorescence colocalization analysis revealed that the timeline of mitochondrial redistribution toward the plasma membrane closely paralleled the course of increased extracellular iron influx. Furthermore, the result of molecular docking suggested the potential interaction between VDAC2 and ZIP14. Together, these works pointed out and further advanced our understanding of the extracellular iron transport into mitochondria in hepatocytes under PFOS exposure and revealed a potential therapeutic target for mitigating the disease mediated by iron-related mechanism.

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Alizarin Dye: Toxicity, Genotoxicity, and Histopathological Alterations in Model Organisms

Environmental and molecular mutagenesis 2026 Mar;67(3):e70046 · 1 Mar 2026

Alizarin is an anthraquinone red dye from natural or synthetic sources, widely used in textiles. Effluents of this activity can contain residual dyes, which may contaminate the aquatic environment. Studies report alizarin's aquatic toxicity, mutagenic, and carcinogenic effects. This study aimed to complement the aquatic toxicity evaluation and confirm its ability to cause genotoxicity in alternative models. Acute toxicity was performed with crustaceans, mussels, and fish embryos, while chronic toxicity was assessed in algae. Light effects on toxicity were evaluated using *Daphnia similis*. Histopathological effects on the gonads of *Mytilus galloprovincialis* and somatic mutations and sperm genotoxicity in *Parhyale hawaiiensis* were investigated. Mutagenicity was confirmed using a miniaturized Ames test. The effect concentration 50% (EC50) for *D. similis* was 90.3, 105, and 68.6 $\mu\text{g L}^{-1}$ for photoperiod (16 h light:8 h dark), light and dark, respectively. For *Danio rerio* embryos, the lethal concentration 50% (LC50) was 45.8 $\mu\text{g L}^{-1}$, and an EC10 of 20.8 $\mu\text{g L}^{-1}$ was calculated for sublethal effects. In vivo exposures caused alterations in the digestive gland and gonads of *M. galloprovincialis*, even in a short-term exposure, and increased the frequency of micronuclei and DNA damage in hemocytes and spermatozooids, respectively, of *P. hawaiiensis*. It was mutagenic in the miniaturized Ames test using strain TA1537 (10% and 30% S9). Alizarin can be classified as a Category 1 acute aquatic toxicity according to the globally harmonized system (GHS). Due to adverse histopathological and DNA effects on reproductive systems in model organisms, it is considered a potential germ cell mutagen.

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