

Bulletin Board

Contents

FEB. 27, 2026

(click on page numbers for links)

OCCUPATIONAL

Exposure to low-dose ionizing radiation and dementia mortality in Canadian nuclear power plant workers	3
Exposure assessment of respirable free silica in coal mining areas	4

CHEMICAL EFFECTS

Platelet mitochondrial DNA methylation mediates the association of bisphenol, phthalate, and paraben exposures with type 2 diabetes mellitus: An exploratory nested case-control study	5
Exposure to environmentally relevant concentration of sodium p-perfluorooctanesulfonate is associated with aberrant barbering behavior in diabetic mice.....	6
Reproductive Impact of Natural, Synthetic and Emerging Chemicals on Wildlife and Domestic Animals	7

ENVIRONMENTAL RESEARCH

Integrated Human Organic Pollutant Exposome and Metabolome Analysis Reveals Biomarkers and Health Risks from Electronic Waste Exposure	8
Spatial coupling and individual-level evidence: linking rice cadmium exposure to liver cancer in a high-risk area of China.....	9
Occurrence, Transformation, and Toxicity of Tire-Derived Chemicals 6PPD and 6PPD-q in the Environment	9

PHARMACEUTICAL/TOXICOLOGY

Proteomic Mapping of Hippocampal Pathways Involved in Lead (Pb)-Induced Neurotoxicity	10
Quantitative acetylopic analysis reveals a key role of acetylation site 1168K in carbamoyl-phosphate synthase 1 (CPS1) following exposure to PFOA and PFO4DA in mouse liver	11

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Bulletin Board

Technical

FEB. 27, 2026

OCCUPATIONAL

Exposure to low-dose ionizing radiation and dementia mortality in Canadian nuclear power plant workers

2026-02-18

METHODS: Our study cohort comprised Canadian NPP workers monitored for radiation exposure between 1950 and 2018, with mortality follow-up extending through 2020. Dementia deaths were identified through record linkage to national mortality data and based on underlying or contributing causes of death. Standardized mortality ratios (SMRs) were derived to compare mortality in the workers to the Canadian population. We used Poisson regression and fit linear excess relative risk models to describe the shape of the exposure-response relationship between cumulative radiation (lagged 10 years) and dementia mortality.

RESULTS: Among 97,250 NPP workers, there were 1,458,299 person-years of follow-up. The mean whole-body lifetime accumulated exposure at the end of follow-up was 9.8 mSv. In total, there were 235 deaths with dementia listed as the underlying cause, and 450 when the definition was expanded to include contributing causes. Relative to the Canadian population, NPP workers had a reduced mortality rate of dementia (SMR = 0.69; 95% CI: 0.63, 0.76). We found some evidence that exposure to ionizing radiation was associated with an increased risk of dementia mortality, but no statistically significant linear dose-response relationship (ERR/100 mSv = 0.010; 95% CI: -0.227, 0.247). Spline analyses suggested a non-linear dose-response relationship.

CONCLUSIONS: We found modest evidence that low-dose radiation exposure is associated with increased risks of dementia mortality. These findings should be interpreted cautiously, given that they differ from those found when we modelled dementia as an incident outcome in a cohort that included many of these same workers. Taken together, our findings highlight limitations of using death data to identify etiological risk factors for dementia.

OBJECTIVE: Evidence suggests that ionizing radiation increases the risk of dementia, but data are limited for those chronically exposed to low doses (<100 millisieverts (mSv)). We addressed this gap by investigating associations between cumulative low-dose radiation exposure and

Bulletin Board

Technical

FEB. 27, 2026

dementia mortality among nuclear power plant (NPP) workers.

Authors: Brianna Frangione, Laura A Rodriguez Villamizar, Ian Colman, Franco Momoli, Estelle Davesne, Lydia Zablotska, Cheryl E Peters, Paul Demers, Minh Do, Tim Prendergast, Philippe Prince, Paul J Villeneuve

Full Source: Environmental research 2026 Feb 18:296:124067. doi: 10.1016/j.envres.2026.124067.

Exposure assessment of respirable free silica in coal mining areas

2026-02-20

The International Agency for Research on Cancer (IARC) has classified free silica/quartz as a Group 1 carcinogen, indicating sufficient evidence of its carcinogenicity in humans. In the present study, suspended particulate matter (SPM), respirable dust (PM10), and free silica content in dust were assessed to determine the associated exposure risk in three mega coal mines (Bharatpur, Kaniha, and Lingaraj OCP) located in the Talcher Coalfield, Odisha, India. The respirable dust samples collected on filter paper were analyzed using Fourier Transform Infrared Spectroscopy (FTIR), Powder X-ray diffraction (PXRD), Scanning Electron Microscopy (SEM), and Energy Dispersive Spectroscopy (EDS) to characterize their composition and morphology. The highest concentrations of SPM and PM10 were observed at Bharatpur OCP, with mean values of 394 $\mu\text{g}/\text{m}^3$ and 136 $\mu\text{g}/\text{m}^3$, respectively. In contrast, Kaniha OCP exhibited slightly lower concentrations of SPM and higher concentrations of PM10, with mean values of 230 $\mu\text{g}/\text{m}^3$ and 193 $\mu\text{g}/\text{m}^3$, respectively. When compared with Bharatpur OCP, the highest concentration of free silica was observed at Kaniha OCP, with values ranging from 5.94 to 114.89 $\mu\text{g}/\text{m}^3$ and a mean concentration of 41.59 $\mu\text{g}/\text{m}^3$. The health risk assessment, conducted using USEPA methodology, indicates that Kaniha OCP poses the highest risks of exposure to respirable silica, with both non-carcinogenic and carcinogenic outcomes, followed by Bharatpur OCP. In contrast, the Lingaraj OCP exhibited comparatively lower health risk levels. The SEM/EDS analysis revealed clear evidence of respirable free silica particles at all

Bulletin Board

Technical

FEB. 27, 2026

three mining sites.

Authors: Manish Yadav, Nitin Kumar Singh, Sumit Saha

Full Source: Environmental geochemistry and health 2026 Feb 20;48(4).
doi: 10.1007/s10653-026-03071-5.

CHEMICAL EFFECTS

Platelet mitochondrial DNA methylation mediates the association of bisphenol, phthalate, and paraben exposures with type 2 diabetes mellitus: An exploratory nested case-control study

2026-02-20

Exposure to environmental pollutants has been found to be associated with epigenetic modifications of platelet mitochondria, which may influence the risk of type 2 diabetes mellitus (T2DM). However, research on the relationship between exposure to environmental endocrine disrupting chemicals (EDCs) and T2DM remains very limited at the molecular level of mitochondrial epigenetic alterations. This study aims to investigate the impact of mixed exposure to bisphenols (BPs), phthalates (PAEs), and parabens (PBs) on T2DM and platelet mitochondrial DNA (mtDNA) methylation, using a nested case-control study design. Levels of BPs, PAEs, and PBs metabolites were quantified using high-performance liquid chromatography-mass spectrometry (HPLC-MS). We used weighted quantile sum (WQS) and bayesian kernel machine regression (BKMR) models to assess the association between individual and mixed exposure to multiple EDCs and T2DM. Methylation levels of mitochondrial coding genes were measured by bisulfite pyrosequencing. In logistic regression models, MT-COX1 methylation levels were significantly negatively associated with T2DM risk, whereas MT-COX3 methylation levels were significantly positively associated. Both WQS and BKMR models indicated that mixed exposure to BPs, PAEs, and PBs was positively linked to T2DM, with DnPrP and DEHP identified as the primary contributors. Mediation analysis demonstrated that MT-COX3 methylation significantly mediated the associations of DEP, DMP, DEHP, DnPrP, DAIP, and MP with T2DM. Our findings indicate that both individual and mixed exposure to PAEs and PBs are positively associated with T2DM risk. Platelet mtDNA methylation mediates the association between EDCs exposure and T2DM risk,

Bulletin Board

Technical

FEB. 27, 2026

suggesting its potential utility as a biomarker.

Authors: Weixia Li, Shuhao Shi, Yuanyuan Yu, Bin Chen, Lei Zhao, Zi Lin, Jiayan Ni, Xiaoqing Li, Shanjun Song, Penghui Li, Shike Hou, Liqiong Guo

Full Source: Ecotoxicology and environmental safety 2026 Feb 20:312:119908. doi: 10.1016/j.ecoenv.2026.119908.

Exposure to environmentally relevant concentration of sodium p-perfluorooctanesulfonate is associated with aberrant barbering behavior in diabetic mice

2026-02-19

This study originated from an incidental behavioral observation. We found that exposure to environmentally relevant concentration of sodium p-perfluorooctanesulfonate (OBS; 3 µg/L), as reported in a previous field measurement study, was associated with aberrant barbering behavior in diabetic mice. To investigate the underlying mechanisms, sixteen 8-week-old male db/db mice were administered OBS at 3 µg/L for 91 days. Biochemical assays of endothelial- and barrier-related markers, hippocampal OBS quantification, stereotaxic hippocampal OBS administration, and metagenomic sequencing of the colonic contents and hippocampal tissues were performed. Based on the metagenomic results, computational biology analyses, including molecular docking, molecular dynamics simulations, and protein functional annotation, were conducted to assess potential OBS-bacterial protein interactions. The results showed that exposure to environmentally relevant concentration of OBS was associated with aberrant barbering behavior in the experimental mice (100 % prevalence). Circulating markers of endothelial activation and basal lamina injury were significantly elevated. Metagenomic analysis revealed that the abundance of *Salmonella enterica* subsp. *diarizonae* was significantly increased in both the colonic contents and hippocampal tissues, with hippocampal abundance positively correlated with colonic abundance. Molecular docking and molecular dynamics simulations indicated that OBS binds effectively to two bacterial proteins. Functional annotation suggested that these proteins are associated with central metabolic and biosynthetic processes relevant to bacterial proliferation. Together, these findings suggest that exposure to environmentally relevant concentration of OBS is associated with aberrant barbering behavior in diabetic mice and may be associated with increased

Bulletin Board

Technical

FEB. 27, 2026

colonic *S. enterica* subsp. *diarizonae* abundance and its presence in the hippocampus.

Authors: De Xin Dang, Shaoyong Xu

Full Source: *Ecotoxicology and environmental safety* 2026 Feb 19:311:119906. doi: 10.1016/j.ecoenv.2026.119906.

Reproductive Impact of Natural, Synthetic and Emerging Chemicals on Wildlife and Domestic Animals

2026-02-27

Wildlife and domesticated animals are exposed to a wide range of natural and synthetic chemicals throughout their life span. Many of these chemicals possess endocrine-disrupting properties which have the potential to disrupt reproductive and developmental process in certain animals. Organochlorine compounds, used as pesticides or in industrial and consumer products in the 1950s, were one of the earliest examples of synthetic endocrine-disrupting chemicals to be linked with adverse reproductive effects in animals. These legacy chemicals have since been restricted for use but continue to persist in the environment and threaten reproductive health of top predators, including porpoises, dolphins and whales. Newer chemicals, including some that were made to replace legacy chemicals and in use today, have been reported to also have endocrine-disrupting properties. These chemicals are manufactured for a wide range of uses, covering a broad range of chemically diverse substances, which include brominated flame retardants, plasticizers and pharmaceutically active ingredients among many other classes of chemicals. When present in the environment, they have been collectively termed emerging contaminants and have been linked with many adverse effects in animals, including impairments in reproduction and development. Whilst exposure to individual chemicals receives regular regulatory interest, there remains a particular concern about wildlife exposures to cocktails of endocrine-disrupting chemicals, which, combined with other stressors, may play a significant role in reproductive disorders that cannot be reproduced in laboratory experiments with single- or multi-chemical exposures. Regulation of chemicals, including restriction on the use of some chemicals, affords some protection to animals of the adverse effects of exposure to some EDCs, but there are presently no specific regulations on a holistic cross-government discharge limit for EDCs into the environment that would significantly reduce or

Bulletin Board

Technical

FEB. 27, 2026

eliminate animal exposure to EDCs.

Authors: Rakesh Kanda

Full Source: *Advances in experimental medicine and biology* 2026:16:167-208. doi: 10.1007/978-3-031-87707-0_6.

ENVIRONMENTAL RESEARCH

Integrated Human Organic Pollutant Exposome and Metabolome Analysis Reveals Biomarkers and Health Risks from Electronic Waste Exposure

2026-02-20

Escalating global electronic waste (e-waste) generation contrasts with <20% formal recycling rates. Policy gaps and inadequate enforcement exacerbate pollution transfer to under-regulated regions, causing substantial environmental and health problems. To address this, we investigated chronic exposure hazards and developed rapid pollution identification technologies. We recruited 2028 participants from e-waste recycling sites and other industrial parks, profiling their urinary organic pollutant exposome (>200 chemicals), oxidative damage, and metabolome by integrating nontargeted and targeted screening methods. Results showed that exposure to pollutant mixtures was significantly associated with increased oxidative damage to nucleic acids and cholesterol. Moreover, these pollutant mixtures collectively explained 46.2% of the variance in urinary metabolome alterations among e-waste workers. The affected metabolites were primarily associated with inflammatory diseases, metabolic disorders, neurological conditions, and cancers. By identifying e-waste exposure characteristic pollutants, we further developed accurate e-waste exposure prediction models (AUC > 0.986; ACC > 0.938) and derived simplified prediction functions and diagnostic indexes with comparable efficacy, which performed well across populations and industrial settings. Overall, this study underscores the significant health risks of e-waste exposure in occupational workers and offers rapid screening tools for e-waste pollution in informal settings, advancing the repurposing of large-scale national exposure monitoring

Bulletin Board

Technical

FEB. 27, 2026

databases for pollution tracking.

Authors: Hong-Xuan Kuang, Ye Liu, Meng-Yang Li, Tong Zheng, Guo-Cheng Hu, Ming-Deng Xiang, Ming-Zhong Ren, Yun-Jiang Yu

Full Source: Environmental science & technology 2026 Feb 20. doi: 10.1021/acs.est.5c13657.

Spatial coupling and individual-level evidence: linking rice cadmium exposure to liver cancer in a high-risk area of China

2026-02-20

Liver cancer ranks as the fourth most common malignant tumor and the second leading cause of cancer deaths in China. Guangxi is a high-risk region, with a crude incidence rate of 41.65/105, significantly exceeding the national average. At a regional scale, liver cancer incidence in Guangxi exhibits a distinct zonal distribution, which shows notable spatial coupling with cadmium (Cd) concentrations in rice grains, though the underlying mechanism remains unclear. This study systematically investigated this relationship by collecting hepatocellular carcinoma incidence and rice grain Cd data from 44 counties/cities, analyzing blood Cd levels in 105 patients and 105 healthy controls, and comparing paired blood (n=316) and rice samples (n=216) from five typical areas. Results demonstrated a significant spatial coupling and correlation between rice Cd distribution and liver cancer incidence. Liver cancer patients had elevated blood Cd levels. High consistency between blood Cd and rice grain Cd levels identified rice consumption as the primary exposure pathway. This study suggests a link between rice cadmium levels and liver cancer in Guangxi, potentially contributing to its geographical distribution. This provincial-scale study provides fundamental data for understanding the chronic hepatocarcinogenic effects of Cd exposure via rice.

Authors: Jianxun Qin, Jin Li, Sibiao Su, Guoli Yuan, Xindong Ouyang, Guodong Zheng, Menglong Xue, Zhe Liu, Xiaoxia Huang, Meixue Liang Changxi Wei

Full Source: Environmental geochemistry and health 2026 Feb 20;48(4). doi: 10.1007/s10653-026-03081-3.

Bulletin Board

Technical

FEB. 27, 2026

Occurrence, Transformation, and Toxicity of Tire-Derived Chemicals 6PPD and 6PPD-q in the Environment

2026-02-21

6PPD (N1-(4-methylpentan-2-yl)-N4-phenylbenzene-1,4-diamine), a widely used rubber antioxidant in tire manufacturing, has garnered increasing global attention, following the discovery that its ozone-oxidation product, 6PPD-q, is the primary toxicant responsible for urban runoff mortality syndrome (URMS) in salmon. This work provides a comprehensive review of 6PPD and 6PPD-q in the environment, focusing on four key aspects: source, occurrence, transformation, and toxicity. Key findings include the following: (1) Tire wear particles are the main source of 6PPD and 6PPD-q in the environment, with their release directly linked to traffic density. (2) Ozone levels, temperature, climate conditions (e.g., snowmelt and rainy seasons), and environmental settings (e.g., roadways, tunnels, and parking lots) can greatly affect exposure levels. (3) In addition to atmospheric ozonation, oxidation in the atmosphere, free radical oxidation, and photocatalytic oxidation also play key roles in transforming 6PPD to 6PPD-q within aquatic systems and soils. (4) URMS is caused by 6PPD-q attacking the organs of fish, leading to blood-brain barrier and vascular dysfunction, with observed interspecies differences in sensitivity; these differences may be linked to variations in metabolic capacity. (5) Both 6PPD and 6PPD-q can enter the human body through the food chain, but their metabolic mechanisms and pathological changes are still unclear. Given the significant research gaps, this review concludes with proposed future research directions to deepen understanding of 6PPD's and 6PPD-q's environmental impacts.

Authors: Shuang Wang, Shuhan Xu, Junhe Lu, Yongshan Wan, Zhenyu Kang, Hao Chen, Shahidul Islam, Bin Gao

Full Source: Environmental science & technology 2026 Feb 21. doi: 10.1021/acs.est.5c12923.

Bulletin Board

Technical

FEB. 27, 2026

PHARMACEUTICAL/TOXICOLOGY

Proteomic Mapping of Hippocampal Pathways Involved in Lead (Pb)-Induced Neurotoxicity

2026-02-22

Lead (Pb) is a well-known xenobiotic and neurotoxin. Chronic Pb exposure remains a major public health concern, particularly in developing countries, and is associated with cognitive impairment, memory deficits, and peripheral and central nervous system toxicity. Pb readily crosses the blood-brain barrier by competing with iron for transport via divalent metal transporter 1 and mimicking calcium to enter through Ca-permeable ion channels, thereby disrupting Fe homeostasis and blood-brain barrier integrity. Pb accumulation promotes excessive generation of reactive oxygen species, mitochondrial dysfunction, lipid peroxidation, and neuroinflammatory responses in the brain. These events alter apoptotic signaling pathways, impair Ca-dependent neuronal communication, and disrupt cholinergic neurotransmission, leading to synaptic dysfunction and neuronal loss in the hippocampus. Proteomic studies have provided insights into the molecular mechanisms underlying Pb-induced neurotoxicity by identifying various Pb-interacting proteins involved in metal transport, oxidative stress regulation, apoptosis, synaptic plasticity, and neurotransmitter signaling. In the absence of effective pharmaceutical treatments for Pb poisoning, these proteomic insights highlight potential diagnostic biomarkers and therapeutic targets that play a central role in antioxidant defense and inflammation control. Emerging enzymatic biosensors also offer promising tools for the rapid and sensitive detection of Pb exposure. Collectively, this review integrates mechanistic, proteomic, and translational perspectives to understand Pb-induced neurotoxicity and support the development of improved diagnostic and mitigation strategies for Pb exposure.

Authors: Kartikey Matte, Shubhangi Pingle, Manjula Kannasandra Ramaiah, Rajani Tumane, Prasad Sherekar

Full Source: Journal of applied toxicology : JAT 2026 Feb 22. doi: 10.1002/jat.70123.