CHEMWATCH

Bulletin Board

Contents

JAN. 30, 2026

(click on page numbers for links)

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CHEMICAL EFFECTS

Prioritizing steatogenic chemicals through integration ToxCast™ data, machine learning, and experimental validation

2026-01-18

As hepatic steatosis driven by environmental exposures increasingly contributes to the global burden of metabolic disease, identifying and prioritizing high-potency steatogenic chemicals is critical for enabling risk-oriented toxicological and environmental regulation. Leveraging the well-established adverse outcome pathway framework for hepatic steatosis, we integrated ToxPi scores derived from 14 molecular initiating events in the ToxCast™ database with in vivo validation in zebrafish. This integrated approach enabled the construction of a training set comprising chemicals with distinct steatogenic potency. Feature selection via Kruskal-Wallis test identified 11 key bioassays, with OT_FXR_FXRSRC1_0480 and NVS_NR_hGR contributing most to model performance. Using leave-one-out cross-validation, the SVM model achieved 91.7% accuracy in the training set. External validation on 35 compounds, although based on binary activity labels, resulted in 77.1% accuracy, indicating moderate but promising generalizability. Final predictions on 345 curated ToxCast™ chemicals (from a total of 9924) were categorized as high- (37.97%), moderate- (18.84%), and null-effect (43.19%) on steatogenic potence by Random Walk with Restart algorithm. In vivo validation of 14 predicted compounds confirmed the model's robustness, and in vitro lipid staining assays in HepG2 cells further demonstrated concordance. This study revealed that several emerging contaminants, including isodecyl diphenyl phosphate, 3,3′-dimethylbisphenol A, tetrabutyltin, tetrabromobisphenol A bis(2-hydroxyethyl) ether, trixylyl phosphate and quinoxyfen, exert high steatogenic potency. These findings underscore the utility of integrating high-throughput data with predictive modeling and experimental validation to prioritize high-potent steatogenic chemicals.

Authors: Xiaoliu Shi, Lingbing Jin, Xiaochun Ma, Qianyi Shen, Jiafan Feng, Ying Liu, Yixiang Wang, Quan Zhang, Cui Wang

Full Source: Environment international 2026 Jan 18:208:110084. doi: 10.1016/j.envint.2026.110084.

CHEMWATCH
Bulletin Board
Technical
JAN. 30, 2026

Acute Toxicity of 6PPDQ, 6PPD, and Other Transformation Products to a Freshwater Mussel (Lampsilis siliquoidea, Barnes 1823)

2026-01-25

The tire rubber antioxidant N-(1,3-dimethylbutyl)-N′-phenyl-p-phenylenediamine (6PPD) and its transformation product, 6PPD-quinone (6PPDQ), have garnered much research attention since the latter was identified as the causative agent of urban runoff mortality syndrome in Coho Salmon (Oncorhynchus kisutch) in the Pacific Northwest of the United States. Fewer than 10 species, all within the fish family Salmonidae, have been shown to be sensitive to 6PPDQ, while several fish and invertebrates are sensitive only to the parent compound. We evaluated the toxicity of 6PPD and 6PPDQ at nominal concentrations of up to 2400 and 94 µg/L, respectively, to juveniles of the freshwater mussel Lampsilis siliquoidea. This species was added to the list of those not sensitive to 6PPDQ at concentrations below solubility limits, but water spiked with 6PPD caused mortality, potentially attributed to a hydrolysis product. Additional work would help to further evaluate potential hazards of transformation products of this ubiquitous chemical.

Authors: David J Soucek, Rebecca A Dorman, Jeffery A Steevens, Viviane Yargeau, Marco Pineda, Christopher D Metcalfe, Erin R Bennett

Full Source: Bulletin of environmental contamination and toxicology 2026 Jan 25;116(2):31. doi: 10.1007/s00128-025-04109-7.

Combined cellular (Cells-on-Particles) and acellular (DTT) assessment of indoor vs outdoor PM2.5 toxicity

2026-01-23

Air pollution is a complex mixture of particles and gases, and its characterization by physical and chemical methods may not fully capture health-relevant effects. Assessing how pollutants interact with living cells provides complementary insight into their potential harmful effects. An integrated "Cells-on-Particles" platform was employed to assess the cytotoxicity of indoor and outdoor PM2.5 using human bronchial epithelial BEAS-2B cells. In this model, cells are directly cultured on collected aerosol particles, enabling an assessment of particle-induced cellular responses. The biological assay was complemented by an acellular dithiothreitol (DTT) assay, which quantifies the oxidative activity of particles and provides insight into their redox activity. Both indoor and outdoor PM2.5 induced dose-dependent cytotoxicity, with outdoor particles showing stronger effects and higher DTT activity. A strong inverse

linear association between DTT activity and cell viability was observed (for indoor R2 =0.96; for outdoor R2 = 0.99), consistent with oxidative activity contributing to the observed cellular effects within this dataset. Under the indoor sampling conditions and PM2.5 size-selective collection, indoor PM2.5 was consistent with predominantly infiltrated outdoor fine particles. The indoor-outdoor differences may reflect infiltration and indoor residence processes. These results demonstrate that integrating the "Cells-on-Particles" exposure model with the DTT assay provides an integrated, a deposition-relevant approach for evaluating the toxicological impact of complex aerosol mixtures.

Authors: Gaile Poceviciute, Violeta Kauneliene, Edvardas Bagdonas, Darius Ciuzas, Martynas Tichonovas, Jurgita Ovadnevaite, Monika Eimutyte, Dainius Martuzevicius

Full Source: Environmental pollution (Barking, Essex : 1987) 2026 Jan 23:127727. doi: 10.1016/j.envpol.2026.127727.

ENVIRONMENTAL RESEARCH

An overview of the impact of PFAS on animals, humans, and the environment using a One Health approach

2026-01-24

Per- and polyfluoroalkyl substances (PFAS) are a group of synthetic chemicals characterized by a fluorinated carbon chain that confers unique physicochemical properties. Widely used in industrial and consumer products, including textiles, food packaging, and firefighting foams, PFAS are highly persistent in the environment, earning them the designation of "forever chemicals." Their stability contributes to their widespread diffusion across different environmental compartments (water, soil, air) and multiple exposure pathways (e.g., diet). These lead to PFAS bioaccumulation and biomagnification, which poses a substantial threat to both ecosystems and human health. Exposure to PFAS has been associated with a range of adverse health effects, including liver damage, thyroid disease, immunotoxicity, reproductive issues, and various cancers in both humans and animals. While regulatory efforts have led to the phase-out of long-chain PFAS such as perfluorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA), emerging research suggest that their short-chain replacements may also raise health concerns. This review applies a One Health framework to explore the interconnected impacts of these contaminants on human, animal, and environmental health. Furthermore, it highlights knowledge gaps that hinder comprehensive risk assessment and management, emphasizing the need for a globally

CHEMWATCH
Bulletin Board
Technical
JAN. 30, 2026

coordinated, multidisciplinary approach to address the multifaceted challenges posed by PFAS.

Authors: Francesca Ferretti, Andrea Barbarossa, Anisa Bardhi

Full Source: Environmental science and pollution research international 2026 Jan 24. doi: 10.1007/s11356-026-37412-9.

The Effects of Indoor Pollutants on Health Care Workers, Patients, and Caregivers in Dental Clinics: A Systematic Review

2026-01-23

Most of the pollution inside a dental clinic comes from the external environment; therefore, the location of the building affects the air quality, as well as the work activity and the type of natural or mechanical ventilation. In the dental sector, pathologies caused by pollutants are increasing, mainly because of methyl methacrylate, 2-hydroxyethyl methacrylate, ethylene glycol dimethacrylate, and triethylene glycol dimethacrylate. However, there are still gaps in the literature regarding the potential effects of all environmental pollutants, and particularly the long-term effects on healthcare workers. A comprehensive search was conducted across PubMed, Embase, Web of Science, and Cochrane Library databases, without time limits, resulting in a total of 155 scientific articles. After the removal of the duplicates, 86 single papers remained for further analysis. The titles of these articles were manually reviewed to include relevant references related to the presence of indoor pollutants in the air of dental clinics. Following this screening process, 10 studies were identified as relevant to the topic of the systematic review. Seven scientific articles were selected to be included in this review. The seven experimental studies reported various air pollutants related to diseases affecting dental health. In particular, the levels of volatile organic compounds, carbon dioxide, and temperature were analyzed in a university dental clinic. Levels of environmental pollutants are much higher during working hours, particularly during dental procedures such as prosthetic and conservative dentistry, due to the chemical nature of the materials used. However, no study reported exceeding the limits set by national environmental regulations. Due to the heterogeneity of the studies, the variety of molecules, the variety of clinical facilities and their geographical location subject to different regulations, as well as the variety of measurement methods, including the variety of traditional and/or technological ventilation systems used in dental departments, a meta-analysis was not performed. Despite the limitations of this systematic review, it was possible to identify some key points that are useful for

further in vivo studies aimed at developing specific guidelines to protect health care workers.
Authors: Giulia Tetè, Manlio Santilli, Natasha Cinta Vinskid, Fabia Profili, Giuseppe Tafuri, Gianmaria D'Addazio, Bruna Sinjari
Full Source: European journal of dentistry 2026 Jan 23. doi: 10.1055/s-0045-1813749.

Investigation of airborne microplastics emission and characteristics in hospital laundry environments

2026-01-24
Plastic pollution has emerged as a critical global concern, with microplastics increasingly detected across various ecosystems, including the atmosphere. Among indoor sources, hospital laundry units have been identified as significant contributors to airborne microplastic emissions. This study investigates the concentration of inhalable microplastics (MPs (in the air of a hospital laundry environment. In this study, air sampling was conducted at three different time points using a personal air sampler operating at a flow rate of 0.5 L/min for 40 min per sample. Microplastics were characterized using FTIR, SEM, and EDX to ensure accurate identification. FTIR analysis identified the predominant polymer as polyamide (nylon), with characteristic peaks consistent with CH2, C = O, and N-H groups. EDX analysis indicated an elemental composition of C (59%), N (32%), O (7%), and P (0.07%). SEM images revealed pronounced diurnal and day-to-day variability, with particle concentrations ranging from 43575 to 66975 particles/m3, though statistical analysis showed these variations were not significantly influenced by environmental factors such as humidity and air velocity in this short-term study. Notably, black particles dominated the samples, representing 97% of the MPs. These results underscore the potential for direct inhalation exposure in occupational settings, raising concerns about respiratory health risks for laundry staff and patients. Therefore, further research is needed to inform the development of stricter ventilation standards, occupational safety measures, and regulatory policies to mitigate microplastic emissions in healthcare environments.
Authors: Aynaz Rangrazi, Ziaeddin Bonyadi, Maryam Sarkhosh
Full Source: Scientific reports 2026 Jan 24. doi: 10.1038/s41598-026-35421-9.

CHEMWATCH
Bulletin Board
Technical
JAN. 30, 2026

PHARMACEUTICAL/TOXICOLOGY

Developmental nephrotoxicity of per- and polyfluoroalkyl substances: Mechanistic insights and epidemiological evidence from prenatal and early-life exposure

2026-01-24
Per- and polyfluoroalkyl substances (PFAS) are highly persistent synthetic chemicals with widespread environmental distribution and near-universal human exposure. Increasing evidence indicates that prenatal and early-childhood periods represent critical windows of susceptibility during which PFAS can disrupt kidney development and impair renal function with potential long-term consequences. This review provides a comprehensive synthesis focusing specifically on PFAS-related nephrotoxicity during early life, integrating mechanistic, toxicological, and epidemiological data to elucidate how developmental exposure may influence lifelong renal health. PFAS readily cross the placenta, accumulate in fetal and neonatal tissues, and exhibit limited clearance in infancy due to immature renal filtration and metabolic pathways. Mechanistic studies demonstrate that PFAS induce developmental nephrotoxicity through multiple pathways, including oxidative stress, mitochondrial dysfunction, PPAR signaling disruption, dysregulated glucocorticoid metabolism, inflammatory activation, and altered nephrogenesis. In vitro and in vivo models further show histopathological changes, metabolic shifts, impaired tubular transport, and activation of apoptotic pathways following developmental exposure, suggesting direct structural and toxicological injury to the maturing kidney. Human cohort studies consistently report associations between early-life PFAS exposure and alterations in renal biomarkers, including reduced estimated glomerular filtration rate (eGFR), elevated serum creatinine and cystatin C, and increased tubular injury markers such as kidney injury molecule-1 (KIM-1) and neutrophil gelatinase-associated lipocalin (NGAL), even when clinical function appears within reference ranges. As a key interpretive consideration, these associations must be viewed in light of ongoing discussion regarding the role of renal clearance in shaping circulating PFAS concentrations, which may influence epidemiological findings. These subtle but quantifiable shifts nevertheless support a life-course hypothesis in which early molecular and functional disturbances may increase vulnerability to chronic kidney disease (CKD) later in life. Substantial gaps remain, including limited longitudinal follow-up, underutilization of mixture-based analytical approaches, and inadequate characterization of emerging PFAS

compounds. Overall, this review underscores the growing evidence that early-life PFAS exposure may impair renal development and function through combined toxicokinetic, molecular, and structural mechanisms, while highlighting the need for improved exposure mitigation, expanded biomonitoring in maternal and pediatric populations, and integrative translational research to support evidence-based public health and regulatory decision-making.
Authors: Jing Liu, Lin Lin, Hongyan Zhang, Xingqiang Li, Yuanyuan Ding, Jihang Yao
Full Source: Ecotoxicology and environmental safety 2026 Jan 24:310:119712. doi: 10.1016/j.ecoenv.2026.119712.

OCCUPATIONAL

Chronic exposure to low levels of glyphosate and metals induces kidney dysfunction

2026-01-24
Chronic kidney disease (CKD) affects 15% of U.S. adults and over 840 million people worldwide. Environmental contaminants, including pesticides and metals, are increasingly recognized as disease contributors, yet mechanisms and consequences of long-term, low-level mixture exposures remain poorly defined. Our prior work identified glyphosate and metals (cadmium, arsenic, lead, vanadium) in drinking water from agricultural regions with high CKD prevalence and showed that early-life co-exposures disrupt kidney development. Here, using adult zebrafish as a mechanistic model, we tested whether chronic, low-level exposure to glyphosate, metals, and their combination impairs kidney function and structure. We exposed zebrafish for 10 and 60 days to glyphosate (10 ppb), metals (2 ppb Cd, 4 ppb As, 5 ppb Pb, 15 V), or glyphosate + metals and evaluated low-molecular weight proteinuria, histopathology, metabolomics, mitochondrial function, mitochondrial copy number, and mitophagy in the kidney. Chronic exposure to glyphosate and metals produced distinct yet overlapping kidney toxicity signatures, including tubular injury, altered metabolism, and impaired mitochondrial function. Co-exposures generated the most severe effects, with mitochondrial beta oxidation, respiration, and mitophagy as sensitive targets. These findings demonstrate that glyphosate and metals at levels found in drinking water damage kidney function over time, with co-exposure worsening outcomes compared to individual chemicals. Our study identifies mitochondria-rich proximal tubules as critical targets of chronic glyphosate-metal exposure, providing mechanistic insight into how environmental contaminants

CHEMWATCH
Bulletin Board
Technical
JAN. 30, 2026

contribute to CKD risk. This work advances understanding of disease etiology in environmental nephropathies and highlights environmental factors as important drivers of kidney health.
Authors: Ilaria R Merutka, Kerry M Ettinger, Melissa Chernick, Ramya T Kolli, Mangala C S De Silva, Iain A Drummond, Nishad Jayasundara
Full Source: Toxicological sciences: an official journal of the Society of Toxicology 2026 Jan 24:kfag007. doi: 10.1093/toxsci/kfag007.

Migration of bisphenol A from commercially available pacifiers: HPLC-FLD analysis and exposure assessment in infants and toddlers

2026-01-24
Bisphenol A (BPA) is an endocrine-disrupting compound widely used in plastics and resins and associated with metabolic, reproductive, and neurodevelopmental disorders. Infants and toddlers are particularly vulnerable because detoxification capacity is immature and exposure occurs during sensitive developmental stages. While BPA is banned in infant feeding bottles within the European Union, its use in pacifiers remains unregulated despite frequent "BPA-free" labeling. This study quantified BPA migration from seven commercially available pacifiers and assessed potential exposure relative to the newly revised European Food Safety Authority (EFSA) tolerable daily intake (TDI; 0.2 ng kg 1 bw day 1) in a worst-case exposure scenario. Pacifiers were dissected into shield and teat components, cut into fragments, and analyzed separately using validated high-performance liquid chromatography with fluorescence detection (HPLC-FLD). Measured BPA concentrations in the eluates (c(BPA,HPLC)) ranged from below the limit of quantification (LOQ) up to 288 µg/L. Based on these measured values, the extrapolated total BPA release per pacifier was 33 to 26,536 ng, with the highest migration observed in a "BPA-free" labeled product. Even the lowest total migration exceeded the 2023 EFSA TDI, whereas exposures would have been negligible under the former 2015 t-TDI (4 µg kg 1 bw day 1). These findings demonstrate that pacifiers can constitute a relevant early-life source of BPA exposure and contribute to already critical background levels. The results underline the unreliability of voluntary "BPA-free" claims and emphasize the need for harmonized EU regulation analogous to existing restrictions for feeding bottles and toys.
Authors: Lena Herwanger, Katharina Sternecker, Jan Kühnisch, Franz-Xaver Reichl, Christof Högg
Full Source: Environmental science and pollution research international 2026 Jan 24. doi: 10.1007/s11356-026-37444-1.

Exploring novel biomarkers for endocrine disruptor exposure: insights into extra-nuclear signaling pathways of estrogen and androgen receptors

2026-01-23

Synthetic chemicals classified as endocrine disruptors (EDs) pose health risks by interfering with sex-steroid hormone signaling. This study evaluated bisphenol A (BPA) for its effects on ERα, ERβ, and AR expression and extranuclear signaling, including ERα phosphorylation, in human monocytes from healthy male and female donors, and assessed ten additional chemicals in ERα-positive breast cancer cell lines (MCF-7, T47D). BPA increased ERα phosphorylation in both male and female monocytes without altering receptor levels, while modulating downstream signaling in a sex-dependent manner and attenuating DHT- or E2-induced effects. The ten other chemicals similarly enhanced ERα phosphorylation, often independently of direct receptor binding. These findings indicate that ERα phosphorylation is a sensitive, early marker of ED activity across immune and epithelial cells and support its use as a receptor-proximal endpoint to complement conventional transcription-based assays in next-generation ED screening strategies.

Authors: Manuela Cipolletti, Ilaria Campesi, Marco Pellegrini, Marco Fiocchetti, Filippo Acconcia, Maria Marino

Full Source: Environmental toxicology and pharmacology 2026 Jan 23:104949. doi: 10.1016/j.etap.2026.104949.