Bulletin de veille Perturbateurs Endocriniens N°37 – Juillet/Août 2026

Objectif : cette veille bibliographique a pour objectif la surveillance de l'actualité et de la littérature scientifique sur les perturbateurs endocriniens. Cette veille est axée sur les aspects suivants : l'exposition, la toxicité, l'évaluation, la prévention, l'épidémiologie et l'actualité.

La validation des informations fournies (exactitude, fiabilité, pertinence par rapport aux principes de prévention, etc.) est du ressort des auteurs des articles signalés dans la veille. Les informations ne sont pas le reflet de la position de l'INRS.

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Les bulletins de veille sont disponibles sur le portail documentaire de l'INRS. L'abonnement permet de recevoir une alerte mail lors de la publication d'un nouveau bulletin (bouton « M'abonner » disponible après connection à son compte).

Exposition professionnelle

Prevalence of metabolic syndrome among family farmers in Southern Brazil chronically exposed to pesticides,
Aguera, R. G., Manocchi, T. F., Da Silva, A. I., De Oliveira, N. G., De Souza, A. A., Lini, R. S., Capelari, S., Castro, J. C., Nerilo, S. B., Machinski, M., Jr., Kaneshima, A. M. D., Wutzow, K. and Mossini, S. a. G., *Environmental Toxicology and Pharmacology*, Aug 2026, Vol. 125.

Metabolic syndrome results from the interaction of cardiometabolic risk factors, including central obesity, hypertension, dyslipidemia, and impaired glucose metabolism, and is a major contributor to cardiovascular disease and type 2 diabetes. Increasing evidence suggests that chronic exposure to

pesticides, may contribute to metabolic dysregulation through inflammatory, oxidative, endocrine and metabolic pathways. This study investigated the association between chronic pesticide exposure and metabolic syndrome in 120 individuals from agricultural areas. Clinical, anthropometric, biochemical, and exposure-related data were obtained through structured interviews and laboratory analyses. Logistic regression adjusted for age, sex, alcohol consumption, and smoking showed a significant positive association between exposure duration and metabolic syndrome (adjusted OR = 1.04; 95% CI = 1.01-1.07; p = 0.020). Principal component analysis demonstrated clustering between metabolic syndrome componentes and pesticide exposure. These findings suggest that chronic occupational exposure to pesticides may contribute to metabolic disturbances and represent a potentially modifiable environmental risk factor.
https://doi.org/10.1016/j.etap.2026.105081

"Career comes at a cost:" exploring work-related reproductive health concerns of Canadian female firefighters,
Asesi, D. B., Macdermid, J. C., Osifeso, T. A., Bromhead, R. C., Jackson, K. and Jahnke, S. A., *Sexual & Reproductive Healthcare*, Sep 2026, Vol. 49.

Background: Female firefighters face unique occupational exposures that may elevate reproductive health risks. Physical exertion during firefighting has been linked to increased stress hormones and elevated blood pressure, while prior studies associate firefighting with adverse pregnancy outcomes, including preterm births and miscarriages. Mental and emotional demands further compound these risks, yet reproductive health concerns among female firefighters remain underexplored in Canada. Methods: This qualitative study employed an interpretive descriptive approach to examine reproductive health concerns and outcomes among Canadian female firefighters. Data were collected through in-depth interviews and were analyzed thematically to identify key experiences and challenges. Results: Participants reported uncertainty regarding the impact of hazardous exposures on reproductive health and highlighted policy gaps addressing female-specific needs. They described challenges with delayed motherhood, navigating the invisibility of menopause and perimenopausal symptoms, and limited access to resources or information on reproductive risks. Physical exertion and occupational stress were perceived as contributing to adverse reproductive outcomes, while mental and emotional stressors heightened vulnerability. Conclusion: Findings underscore the urgent need for targeted policies, resources, and interventions to address reproductive health risks among female firefighters. Supporting this occupational group is essential to improving well-being and promoting healthier reproductive outcomes within the fire service.
https://doi.org/10.1016/j.srhc.2026.101248

Genetic variation of CBS (844ins68) and MTHFR A1298C among aluminum-exposed workers with emphasis on the role of oxidative stress,
Aziz, N. S. A., Shahy, E. M., Taha, M. M., Mahdy-Abdallah, H., Ibrahim, K. S., Mahdy, G. M. and Ellaithy, L. S., *Journal of the Egyptian Public Health Association*, Jun 19 2026, Vol. 101, no. 1.

BackgroundAluminum (Al) is the most abundant element in the Earth's crust. Occupational exposure to aluminum can lead to serious adverse health effects for workers in the aluminum industry. Genetic variations could be considered as contributing internal factors for the susceptibility of individuals to toxicities related to Al exposure. Different experiments have investigated genetic polymorphism variations in metabolic pathways, which could significantly modulate an individual's susceptibility to aluminum-induced toxicity. This study aimed to evaluate the effects of aluminum exposure on oxidant-antioxidant status, parathyroid hormone (PTH), and free testosterone levels. Also, to show whether an association exists between MTHFR A1298C (rs1801131) and CBS (844ins68) gene polymorphisms with the aforementioned biochemical parameters among

occupationally exposed workers to Al.MethodsUrinary heavy metals (Al, Cd, Ni, Cr, Pb) were measured through ICP-MS in 95 workers in the aluminum industry and 90 control individuals. We also analyzed serum MT-1, MDA, PTH, free testosterone, and TAC using ELISA methods. CBS (844ins68) and MTHFR A1298C gene polymorphisms were performed by (PCR-RFLP). Results Revealed a significant rise in serum MT-1, MDA, and urinary heavy metals levels with reductions in TAC, Free testosterone, and PTH levels. A negative relation was found between serum TAC, duration of exposure, and between age and serum free testosterone levels. In workers, the association of the wild type of MTHFR A1298C genotype with a rise in serum MT-1 and MDA levels and a decrease in TAC was observed compared to the control, while the heterozygote genotype AC was associated with reduced serum free testosterone, TAC, and PTH levels among the workers group. Moreover, serum MT-1 showed a significant rise in NN genotype of the CBS gene with a decline in serum TAC and PTH levels, while Heterozygote NI recorded reduced TAC levels among the worker group compared to the control. Significant reduction in serum TAC was shown in the heterozygote NI among the worker group compared to the control. Conclusion Association of wild-type and heterozygote genotype of both MTHFR A1298C (rs1801131) (AA and AC) and CBS (844ins68) (NN and NI) with reduced TAC levels among the worker group. Moreover, the AC genotype of MTHFR was associated with a decrease in the PTH and free testosterone levels among Al industry workers.
https://doi.org/10.1186/s42506-026-00222-5

Chapter 4 - Environmental and health impacts of the pulp and paper industry
Bajpai, P. (2026). Nanotechnology for Cleaner Pulp and Paper Processes. Elsevier. 41-58.
The pulp and paper industry is a resource-intensive sector associated with significant environmental pollution and occupational health concerns. This chapter critically examines the sources, characteristics, and impacts of emissions generated during pulping, bleaching, and paper-making processes. Liquid effluents rich in organic matter, chlorinated compounds, and suspended solids contribute to aquatic toxicity and ecosystem degradation, while gaseous emissions and particulate matter affect ambient air quality. Solid wastes such as sludge pose additional disposal challenges. This chapter further highlights occupational exposures to chemicals, dust, and combustion by-products, linking them to respiratory disorders, dermatological effects, and long-term health risks among workers. Variability in reported impacts across studies is discussed in relation to exposure assessment, technological differences, and confounding factors. Emerging cleaner technologies and preventive strategies are also outlined to mitigate environmental and human health risks.
https://doi.org/10.1016/B978-0-443-51212-4.00014-1

Glyphosate and health in Uruguay: A 25-year review of research on agricultural worker exposure, Bühl, V., *Science of The Total Environment*, 2026/10/01/ 2026, Vol. 1050, p. 182234.

In Uruguay, glyphosate is a widely used herbicide, applied across both small- and large-scale horticultural crops. Agricultural workers in direct contact with glyphosate during its application and handling constitute a vulnerable population exposed to this herbicide. The purpose of this report is to assess the health impacts of exposure to glyphosate on workers within the Uruguayan context. A PRISMA-guided bibliographic review was conducted using databases including PubMed, Scopus, and SciELO, Google Scholar, and academic and governmental repositories in Uruguay. Studies published between 2000 and 2025 were selected, focusing on research that directly or indirectly addressed the effects of glyphosate on agricultural workers in Uruguay. Of the eight studies identified, only two centered specifically on glyphosate, while the remaining included exposure to multiple pesticides. Primary routes of exposure to glyphosate were dermal and inhalational, particularly during mixing, loading, and application activities, with reported acute symptoms involving gastrointestinal, neurological, and muscular manifestations, and chronic effects including respiratory and skin conditions. Several studies underscored the importance of biomarkers in improving correlations

between exposure levels and clinical outcomes. Additionally, one study highlighted a common misconception among workers, who, due to lack of odor of glyphosate, perceive it as less hazardous than other pesticides. This belief underscored the need to enhance education on the risks associated with glyphosate and other pesticides. This report provides key insights by reviewing studies on exposure to glyphosate among agricultural workers in Uruguay. Additionally, this work supports the international research agenda by emphasizing the importance of educational and regulatory measures to reduce occupational health risks associated with pesticide exposure in diverse agricultural contexts. https://doi.org/10.1016/j.scitotenv.2026.182234

Plasticiser exposure assessment in E-waste recycling workers linking passive samplers and urinary biomonitoring,
Callejas-Martos, S., Den Ouden, F., Cleys, P., Belova, L., Covaci, A. and Eljarrat, E., *Journal of Hazardous Materials*, 2026/08/01/ 2026, Vol. 514, p. 142907.

Dismantling activities in e-waste recycling facilities release significant amounts of hazardous chemicals, including phthalates, alternative plasticisers, and organophosphate esters (OPEs), which represent a relevant source of occupational exposure. This study applied an integrated approach combining passive polydimethylsiloxane (PDMS) sampling and human biomonitoring via urinary metabolites to characterise workers' exposure. Parent compounds from all three additive families (n = 31) and their respective metabolites (n = 34) were detected in the PDMS pendants and urine samples, confirming continuous exposure to complex chemical mixtures. Phthalates exhibited the highest concentrations in pendants (median = 461 ng/cm2), followed by OPEs (152 ng/cm2), and alternative plasticisers (33.5 ng/cm2). In contrast, estimated daily intakes (EDIs) derived from urinary biomarkers were dominated by alternative plasticisers (median EDI = 8719 ng/kg bw·day), with DEHA being the predominant contributor (median EDI =7751 ng/kg bw·day). Phthalates emerged as the second main contributor to urinary EDIs (median EDI = 3447 ng/kg bw·day), whereas OPEs showed substantially lower values (median = 78.3 ng/kg bw·day). Both exposure assessment strategies revealed significant qualitative and quantitative correlations, with some compounds presenting an effect estimate coefficient (β) of up to 0.586, highlighting that e-waste dismantling activities are relevant contributors to the total daily intake. Despite elevated exposure levels compared to those in the general population, no health risks were identified.
https://doi.org/10.1016/j.jhazmat.2026.142907

Phthalate and alternative plasticizer exposure and antiandrogenic risk among automobile parts manufacturing workers in South Korea,
Choi, S., Bang, S., Kho, Y., Park, N.-Y., Shin, S. and Park, J., *Journal of Hazardous Materials*, 2026/09/01/ 2026, Vol. 515, p. 142923.

Although plasticizers are widely used in automobile parts manufacturing, no large-scale biomonitoring study has simultaneously measured both legacy phthalates (PAEs) and emerging alternative plasticizers (APs). We quantified 33 urinary metabolites of 13 PAEs and 4 APs using ultra-high-performance liquid chromatography coupled with tandem mass spectrometry in paired samples (prior-to- and end-of-workweek) from 490 male workers at five South Korean automobile parts companies. K-means clustering identified three exposure profiles: PAE-dominant (n = 173), with elevated ΣDEHP and MnBP; AP-dominant (n = 143), driven by DEC (a metabolite of acetyl triethyl citrate (ATEC)); and a low-exposure cluster (n = 174), predominantly comprising office-based workers. At the population level, total plasticizer concentrations did not significantly increase across the workweek (ΣPlasticizers median: 115 vs. 121 μg/L, specific gravity-adjusted; p = 0.201). However, cluster-specific analyses demonstrated significant end-of-workweek increases in the PAE-dominant group (p < 0.001) and the AP-dominant group (p < 0.001), revealing that population-level

averaging masks occupational contributions. Painting and processing workers had significantly higher concentrations than assembly workers, and personal protective equipment use was associated with lower metabolite levels (43.3% reduction in ΣPlasticizers with combined mask and glove use; $p < 0.001$). Conventional hazard indices exceeded one in only six workers (1.2%), whereas the cumulative antiandrogenic hazard index (HIAA) exceeded one in 116 workers (23.7%). These findings demonstrate that conventional, endpoint-agnostic risk assessment substantially underestimates cumulative antiandrogenic risk, supporting endpoint-specific, mixture-based hazard evaluation for co-occurring plasticizers in occupational settings.
https://doi.org/10.1016/j.jhazmat.2026.142923

Machine Learning Prediction of Excess Relative Risk for Radiation-Induced Solid Thyroid Cancer Among Nuclear Medicine Healthcare Professionals: A Computational Modeling Study, Chouchen, M., Barki, C., Dergaa, I., Ceylan, H. I., De Giorgio, A., Bragazzi, N. L. and Rahmouni, H. B., *Bioengineering-Basel*, Jun 18 2026, Vol. 13, no. 6.

Background: Nuclear medicine healthcare professionals (NMHP) sustain chronic occupational exposure to iodine-131 (I-131), conferring an elevated risk of radiation-induced solid thyroid cancer. Established radiobiological prediction tools derive risk coefficients from atomic bomb survivor data but are not configured for rapid individualized risk assessment in occupational exposure settings. This study examined whether machine learning algorithms can serve as high-precision computational surrogates for excess relative risk estimation in NMHP. Aim: The study aimed to (i) develop and validate three machine learning algorithms for predicting the excess relative risk per unit absorbed dose for radiation-induced solid thyroid cancer (ERR/Gy.RST), (ii) characterize relationships between dosimetric and demographic features and predicted risk, and (iii) identify the optimal algorithm for deployment in occupational health surveillance. Methods: A dataset of 4657 observations was constructed from Life Span Study-derived ERR/Gy parameters, adapted to occupational low-dose conditions, using a dose-and-dose-rate effectiveness factor of 2.0, per ICRP Publication 103. Five features (gender, age at exposure, current age, distance from the I-131 source, and cumulative absorbed dose in the thyroid) were used to train a decision tree regressor (dtcr), a random forest regressor (rfr), and a multilayer perceptron (MLP) neural network algorithm. Results: Cumulative absorbed dose in the thyroid correlated positively with ERR/Gy.RST ($r = 0.63$, $p < 0.01$), while radiation source distance demonstrated a strong inverse association ($r = -0.38$, $p < 0.01$). The MLP algorithm achieved R-2 score = 0.999, MSE = 0.002, and MAE = 0.010, substantially outperforming the rfr (R-2 score = 0.700, MSE = 0.410, MAE = 0.295) and the dtcr (R-2 score = 0.510, MSE = 0.654, MAE = 0.289). Conclusions: The MLP algorithm provides a high-fidelity surrogate for established ERR/Gy.RST projection tools in the NMHP context, enabling computationally efficient, feature-integrated occupational radiation-induced thyroid cancer risk quantification. These findings suggest that machine learning-based surrogate modeling is a practical, scalable complement for occupational health practitioners and radiation protection officers to support individualized surveillance of radiation-induced thyroid cancer risk in nuclear medicine departments.
https://doi.org/10.3390/bioengineering13060696

Occupational particle exposure and diabetes: a cohort study in Swedish construction workers, Edlund, K. K., Sand, E., Andersson, M., Johannesson, S., Eliasson, B. and Stockfelt, L., *International Archives of Occupational and Environmental Health*, Jul 2026, Vol. 99, no. 5.

Purpose Diabetes is a growing global health concern. Environmental exposure to airborne particles has recently been identified as a risk factor for type 2 diabetes. Despite hundred- or thousandfold concentrations in many occupational settings, epidemiological studies in workers are very scarce. This study aims to quantify the association between occupational inorganic particle exposure and

incident type 2 diabetes among Swedish construction workers. Methods This was a retrospective cohort analysis in the Swedish Construction Workers' Cohort (Byggh & auml;lsokohorten), with data from occupational health examinations between 1971 and 1993. Exposure assessment was based on a cohort-specific job-exposure matrix. Data from the National Patient Register, the National Diabetes Register, and the National Prescribed Drug Register were combined to identify incident type 2 diabetes cases. Longitudinal, multivariable Cox proportional hazards regression models were fit to estimate adjusted hazard ratios (aHR). Results We included 275,941 male workers with a mean follow-up time of 31 years. Slightly more than half were exposed to inorganic dusts or fumes. In crude analyses, workers exposed to inorganic dust and fumes were at an increased risk of type 2 diabetes. After confounder adjustment, only workers exposed to high levels of cement, concrete, and quartz dust were associated with an increased risk of type 2 diabetes (strongest for high levels of cement dust, aHR 1.12, 95% CI 1.02-1.24). Conclusions In this large cohort of construction workers with long follow-up, workers with high exposure to cement, concrete, and quartz dust were at a moderately increased risk of type 2 diabetes. https://doi.org/10.1007/s00420-026-02224-4

Integrating Toxicological Intelligence into Global E-waste Governance: From Material Recovery to Health-protective Circularity,
Eze, C. T., Eze, O. O., Okeke, E. S., Ezeorba, T. P. C., Akatakpo, S., Ogbuene, E. B., Ugochukwu, T. E., Nwagwe, R. O., Otitoloju, A. A., Gao, P., Michelangeli, F., Stopper, H., Pohjanvirta, R., Heipieper, H. J., Weindl, G. and Vinken, M., *Journal of Hazardous Materials Advances*, 2026/08/01/ 2026, Vol. 23, p. 101352.

The rapid growth of electrical and electronic equipment production and disposal have positioned e-waste at the intersection of environmental contamination, public health risk, and circular economy policy. This review synthesizes global evidence on environmental contamination and biomonitoring data to examine how chemical exposures from e-waste-impacted ecosystems translate into human and ecological health risks. We identify dominant contaminant profiles across environmental compartments, highlighting spatial disparities in exposure burdens, and evaluate documented associations with adverse biological outcomes. While circular economy strategies are increasingly promoted to address material recovery and resource efficiency, there are persistent toxicological blind spots in current e-waste management paradigms. Recycling technologies are rarely assessed for their potential to generate secondary emissions, transformation products, or regrettable substitutions. Therefore, a transformation toward truly sustainable circularity requires systematic integration of hazard identification, exposure science, and lifecycle-based risk assessment into innovation and policy frameworks. To this end, the applicability and limitations of safe-and-sustainable-by-design approaches and new approach methodologies for anticipatory chemical safety evaluation within circular e-waste systems are critically examined. There is a clear need for harmonized toxicological reference values to prevent the recirculation of hazardous substances in secondary material streams. Future advancement toward toxicologically informed circularity depends on reframing circular economy policy from a predominantly resource-efficiency paradigm to a health-centered systems approach. Embedding structured, hazard-informed assessment across the electrical and electronic equipment value chain and e-waste life cycle can help mitigate long-term human and ecological risks while supporting innovation aligned with environmental integrity and global justice. https://doi.org/10.1016/j.hazadv.2026.101352

Polycyclic aromatic hydrocarbons and perinatal outcomes: a scoping review,
Gopal, S. H., Suter, M. A., Moorthy, B. and Pammi, M., *Pediatric Research*, 2026.

Polycyclic aromatic hydrocarbons (PAHs) are generated during the incomplete combustion of carbon-based materials and have been identified as mutagenic, teratogenic, carcinogenic, and

immunotoxic agents. Maternal exposure to PAHs via inhalation, occupational exposures, dietary intake, or dermal absorption has been linked to an elevated risk of infertility, spontaneous abortion and preterm birth. PAHs can cross the placenta, potentially leading to adverse fetal outcomes including growth restriction and congenital anomalies. Additionally, both prenatal and postnatal exposure to PAHs have been associated with a range of childhood disorders, including asthma, attention deficit hyperactivity disorder, autism spectrum disorders, pediatric cancers, and allergic conditions. The objective of this scoping review is to provide a comprehensive overview of current evidence regarding the effects of PAH exposure on maternal, fetal, neonatal, and childhood health outcomes. https://doi.org/10.1038/s41390-026-05264-1

PCB exposure during remediation and demolition of contaminated buildings: Full congener analysis and effects of hygiene measures,
Hammel, S. C., Jensen, S. P., Kines, P., Ribalta, C., Kofoed-Sørensen, V., Jensen, K. A., Fonseca, A. S., Schlünssen, V., Meyer, H. W., Hougaard, K. S. and Frederiksen, M., *Environment International*, 2026/08/01/ 2026, Vol. 214, p. 110384.

Several residential buildings in Denmark were highly contaminated with polychlorinated biphenyls (PCBs), exceeding indoor air guidance values, and were set for demolition. We conducted a comprehensive evaluation of occupational exposure to PCBs during the demolition process using personal and stationary air sampling, hand and surface wipes, t-shirts, and glove wipes, and compared them to previously analyzed silicone wristbands and the original PCB sealant source. We further evaluated the utility of hygiene measures like handwashing for reducing exposure. Samples were analyzed for all 209 PCBs via microwave-assisted extraction and gas chromatography-tandem mass spectrometry (GC–MS/MS), and > 140 PCB features (individual congeners and co-elutions) were detected in at least one sample. Overall, tetra-CBs were the dominant homolog group across all samples, but the most abundant individual congeners depended on the sample matrix, with lower chlorinated PCBs being more prominent in air compared to wipes and wristbands. Levels of personal exposures were highly task-specific, with workers who cut the PCB-containing sealants having the highest exposure across sample types. Handwashing was shown to reduce PCB levels on workers' hands, while PCBs were also observed to permeate typical textile work gloves and worn t-shirts showed varying degrees of contamination. Across all sample types, total congener analysis demonstrated that traditional indicator-based calculations underestimated total PCB content by 25% or more. Overall, our results indicated that personal protective equipment and simple hygiene can reduce exposure, and demolition workers have high potential for PCB exposure when handling contaminated materials, necessitating careful exposure mitigation and task-specific controls. https://doi.org/10.1016/j.envint.2026.110384

Dual-Enzyme Capillary Flow-Driven Microfluidic Sensor for Multiplexed Screening of Occupational and Take-Home Pesticide Exposure,
Hefner, C. E., Aryal, P., Elam, T., Brack, E. and Henry, C. S., *Acs Sensors*, 2026.

Over 3.7 million tons of pesticides are applied each year in agriculture to maintain crop production. However, mixing, loading, and spraying expose agricultural workers to pesticide residues. Beyond occupational exposure, take-home pathways, where residues on gloves, clothing, and tools are transported into vehicles and homes, pose additional risks to families, especially vulnerable children. Pesticide exposure has been linked to nausea, respiratory distress, neurological impairment, and endocrine disruption, underscoring the need for effective monitoring in both occupational and domestic settings. Conventional analytical methods are costly, require trained personnel, and rely on bulky instruments, limiting their suitability for rapid, on-site screening. Paper-based microfluidic devices have recently emerged as low-cost, portable tools for environmental monitoring, capable of

integrating colorimetric enzyme assays on disposable platforms. Yet, the most current systems detect only a single pesticide class, limiting their applicability to complex real-world mixtures. To address this, we developed a dual-enzyme microfluidic device for simultaneous colorimetric screening of representative organophosphate and dithiocarbamate pesticides dichlorvos and ziram, respectively. The system is simple (two steps), low-cost (<$1/test), and user-friendly, enabling rapid field deployment and citizen-based monitoring. Detection limits of 0.15 ppm for dichlorvos and 0.02 ppm for ziram enable environmentally relevant quantification. Testing spiked pesticide residues on leather, denim, and cotton substrates yielded recovery rates of 51-148% for dichlorvos and 29-84% for ziram, demonstrating suitability for monitoring residues on common clothing materials. This dual-enzyme platform provides a practical advance toward accessible, multi-target pesticide exposure monitoring across occupational and household settings.
https://doi.org/10.1021/acssensors.6c01184

Associations between serum per- and polyfluoroalkyl substances and serum metabolomic profiles in elite athletes: a cross-sectional multi-sport study,
Hwang, J., Seo, Y., Lee, H.-J., Lee, M., Cho, J., Lee, H. J., Lee, H. and Min, H., *Environment International*, 2026/09/01/ 2026, Vol. 215, p. 110440.

Per- and polyfluoroalkyl substances (PFAS) are persistent synthetic chemicals linked to adverse health outcomes in the general population and occupationally exposed groups. However, PFAS exposure in elite athletes remains poorly characterized despite potential sport-related exposure media. We investigated serum PFAS concentrations and associated metabolomic profiles in 242 elite athletes across 19 sport disciplines classified into six environment-based groups. Serum concentrations of 45 endocrine-disrupting chemicals and 304 metabolites were measured using targeted LC-MS/MS. Serum PFOA and PFHxS concentrations were 1.7- and 2.1-fold higher than KoNEHS cycle 5 adult reference values, and the sum of seven PFAS exceeded the NASEM clinical follow-up threshold of 20 ng/mL in 90.1% of athletes. PFAS levels differed significantly among sport environment groups ($p < 0.001$), with the highest levels in the Synthetic/Outdoor group. After adjustment for sex and correction for multiple comparisons, 27 metabolites were significantly associated with PFOS, including an inverse association with serotonin and positive associations with branched-chain amino acids, acylcarnitines, and glycerophosphocholine. Pathway analysis indicated enrichment of branched-chain amino acid metabolism. Consistent associations were observed for PFOS, PFOA, and PFHxS, and post hoc weighted quantile sum (WQS) analysis showed mixture associations with serotonin and acetylcholine. This study provides an initial characterization of PFAS exposure and associated serum metabolomic profiles in elite athletes. These cross-sectional findings support further longitudinal studies with detailed exposure assessment in athlete populations.
https://doi.org/10.1016/j.envint.2026.110440

Investigation of Exposure to Plasticizers Among Electronic Waste Recycling Workers and Their Health: Protocol for a Cross-Sectional Study,
Ketema, R. M., Le, T. H., Nguyen, S. V., Ngo, H. T. T., Zeng, Y., Yasuda, A., Marsela, M., Minatoya, M., Le, T. H., Ta, B. T., Pham, V. T., Nguyen, H. T., Pham, T. V., Tran, H. S., Ha, P. L., Phan, C. T. T., Ait Bamai, Y., Kishi, R. and Ikeda, A., *JMIR Research Protocols*, 2026/01/01/ 2026, Vol. 15.

Background The global consumption of electronic and electrical products has increased the generation of electronic waste (e-waste), which poses environmental and public health concerns. In Vietnam, informal e-waste processing, characterized by unregulated collection and dismantling, is widespread and exposes workers to hazardous substances. However, data on the health status of e-waste workers remain limited, and exposure to phthalates, which are endocrine-disrupting chemicals widely used in electronics, has not yet been evaluated. Objective This study aims to assess

the health status of e-waste workers in Vietnam, measure their phthalate exposure levels, and examine associations with health outcomes. Methods We conducted a cross-sectional study of adult e-waste workers in Bui village, Vietnam. Trained staff collected demographic, occupational, respiratory, and dietary habit information using a structured questionnaire in Vietnamese. Medical examinations were performed and body composition measurements, blood samples, and urine samples were obtained. Blood samples were analyzed for hematological parameters and several biochemical markers; urine samples were collected for routine urinalysis parameters and phthalate quantification. Results Field survey data were collected in March 2025, and 253 e-waste workers were recruited. Preliminary descriptive analysis indicated that most participants were female, had low income, and relied on e-waste processing as their primary income. Personal protective equipment, particularly dust masks and gloves, was commonly used. Laboratory measurements of urinary phthalate metabolites were completed by November 2025. Conclusions This protocol provides a framework for evaluating the health status, metabolic indicators, and phthalate exposure among e-waste workers in Vietnam and supports occupational health evaluation. The preliminary findings highlight the feasibility of this study, which supports the evaluation of occupational exposure and health outcomes. https://doi.org/10.2196/96702

Occupational exposure to pesticides increases the risk of amyotrophic lateral sclerosis: a systematic review and meta-analysis,
Labreche, F., Prud'homme, P., Gagnon, M., Dupre, N. and Gravel, S., *Occup Environ Med*, Aug 23 2026, Vol. 83, no. 5, p. 291-300.

OBJECTIVE: To systematically review the evidence on the association between occupational exposure to pesticides and the risk of amyotrophic lateral sclerosis (ALS). METHODS: A systematic search, conducted in eight bibliographic databases for publications between 1990 and 2025, identified observational studies estimating the risk of ALS after occupational pesticide exposure. Study quality was assessed using the WHO Risk of Bias (RoB) assessment instrument for systematic reviews, with the ROBINS-E (RoB in non-randomised studies of exposure) tool domains of bias. Pooled risk estimates were produced using random-effects models with restricted maximum likelihood, heterogeneity was assessed with I(2) statistics, and meta-regressions and publication bias explored with funnel plots and Egger's test. RESULTS: Eight case-control studies (1734 cases) were retained for meta-analysis from 767 initially screened articles. 'Ever' occupational exposure to pesticides was associated with an increased risk of ALS (n=6 studies, pooled OR (pOR)=1.6; 95% CI 1.1, 2.2; I(2)=57%), for combined sexes. The risk for exposure to herbicides was slightly greater (pOR=1.7, I(2)=0.0%) than for exposure to insecticides or fungicides (pORs=1.6, I(2)=0.0%). Based on three studies, ever exposure to high levels of pesticides was associated with a higher risk (pOR=2.7; 95% CI=1.4, 5.0) than exposure to low levels (pOR=1.9; 95% CI=1.0, 3.7). Self-reported exposure assessment methods and older publication dates (<2015) were statistically significant predictors of the effect size. CONCLUSION: Despite the small number of studies and some heterogeneity, our results add to the evidence suggesting that occupational exposure to pesticides may increase the risk of ALS. https://doi.org/10.1136/oemed-2025-110662

Additive effects of heat and volatile organic compounds co-exposure on metabolic syndrome: evidence from petrochemical workers in China,
Li, M. H., Xu, R., Zhang, Y. L., Du, S. S., Xu, R., Zu, Q., Lv, Z. H., Zheng, W., Ye, W. M. and Xiang, J. J., *International Archives of Occupational and Environmental Health*, Jul 11 2026, Vol. 99, no. 6.

Objective To investigate the independent and joint associations of heat and volatile organic compounds (VOCs) exposure with metabolic syndrome (MetS) among petrochemical workers. Methods A cross-sectional study was conducted among 1140 petrochemical workers in the

Quangang Petrochemical Industrial Park, China. Occupational heat and VOCs exposures were assessed following national occupational standards, and MetS was defined according to the 2020 Chinese Type 2 Diabetes Prevention and Treatment Guidelines. Multivariable logistic regression and interaction analysis using the relative excess risk due to interaction (RERI) were performed to evaluate additive associations. Machine learning models were further applied to explore the relative contributions of exposures. Results The prevalence of MetS among workers was 27.8%. After adjustment for demographic, behavioral, and occupational factors, heat exposure (OR = 1.53, 95% CI: 1.12-1.94) and VOCs exposure (OR = 1.38, 95% CI: 1.08-1.79) were both associated with higher MetS risk. Workers exposed to both heat and VOCs showed a stronger association with MetS (OR = 2.08, 95% CI: 1.32-2.96), with evidence of a positive additive interaction (RERI = 0.405, Pinteraction = 0.012). Exploratory machine-learning analyses further suggested that combined heat and VOC exposure contributed more to MetS than either exposure alone. Conclusions Combined exposure to heat and VOCs showed evidence of a positive additive interaction in relation to MetS among petrochemical workers. These findings underscore the need for integrated occupational health strategies in heat-sensitive industries under a warming climate. https://doi.org/10.1007/s00420-026-02227-1

Metabolic alterations bridge exposure to urinary halogenated phenols and bisphenols to dyslipidemia: Evidence from an e-waste recycling area,
Li, M.-Y., Kuang, H.-X., Chen, T.-H., Wang, X.-F., Song, Y.-F., Xiang, M.-D., Yu, Y.-X. and Yu, Y.-J., *Environmental Pollution*, 2026/09/01/ 2026, p. 129065.

Halogenated phenols and bisphenols are metabolic-disrupting chemicals related to electronic waste (e-waste) dismantling activities. However, their adverse effects on lipid metabolism and the underlying mechanisms remain insufficiently understood. Based on a cross-sectional study of 227 e-waste workers and residents from an e-waste recycling area in China, we measured 11 urinary halogenated phenols and bisphenols, quantified four serum lipid parameters, and performed untargeted serum metabolomics. Dyslipidemia and its subtypes (hypercholesterolemia, hypertriglyceridemia, and hyper low-density lipoprotein cholesterol) were defined according to clinical criteria. After covariate adjustment, six phenolic compounds (PCs) and their mixture were associated with increased odds of dyslipidemia and its subtypes, with 3,5-dichlorophenol contributing most to dyslipidemia, hyper low-density lipoprotein cholesterol, and hypertriglyceridemia, whereas 3,4-dichlorophenol contributed most to hypercholesterolemia. Stronger associations were observed in males, normal-weight individuals, and those aged <60 years. Mediation analysis identified nine metabolites that significantly mediated these associations (proportions mediated: 14.8%-63.7%), annotated to amino acid, energy, and retinol metabolism pathways. Hierarchical partitioning revealed that PC exposure alone explained 4.28% of the variance in lipid metabolism, indicating a meaningful contribution. These findings provide potential mechanistic insights into the associations of halogenated phenol and bisphenol exposure with lipid homeostasis and highlight the need to reduce exposure to these chemicals among e-waste workers and nearby residents. https://doi.org/10.1016/j.envpol.2026.129065

Environmental Exposures and Pregnancy Loss: A Critical Gap Analysis for Unexplained Recurrent Pregnancy Loss,
Liu, C., Huang, J. and Wang, F., *Reproductive Toxicology*, 2026/09/01/ 2026, p. 109341.

Recurrent pregnancy loss (RPL), defined as two or more pregnancy failures, affects approximately 1%–3% of reproductive-age couples worldwide. Despite advances in clinical evaluation, 50%–60% of RPL cases remain unexplained, referred to as unexplained recurrent pregnancy loss (URPL), leaving a substantial proportion of patients without an identifiable etiology or evidence-based preventive

strategies. Emerging evidence suggests that environmental exposures may represent an underrecognized yet modifiable contributor to URPL; however, the existing literature remains fragmented and methodologically flawed by the homogenization of pregnancy loss. This narrative review and critical gap analysis evaluates the epidemiological and mechanistic evidence linking a broad spectrum of environmental exposures to pregnancy loss. We systematically map the evidence base across ambient air pollutants, heavy metals, endocrine-disrupting chemicals, pesticides, and lifestyle/occupational hazards, with a strict focus on isolating URPL-specific data from general miscarriage cohorts. We further examine the principal biological mechanisms through which these exposures precipitate pregnancy loss, including oxidative stress, maternal immune dysregulation, epigenetic reprogramming, endocrine disruption, and placental vascular impairment. This critical gap analysis reveals that while environmental pollutants consistently increase the risk of sporadic pregnancy loss, URPL-specific evidence remains profoundly scarce. We highlight the urgent need to address the pervasive conflation of sporadic and recurrent loss in existing cohorts, call for future prospective studies prioritizing mixture-based exposures, and propose a forward-looking, non-judgmental framework for preconception wellness that mitigates the psychological burden on URPL patients. https://doi.org/10.1016/j.reprotox.2026.109341

Association between Microplastic Exposure and Hepatic Dysfunction in Humans: A Cross-Sectional Study of Occupationally Exposed Individuals,
Lu, Y., Tu, N., Zhang, H., Li, B., Qi, G., Chen, S., Pang, Y. and Chen, L., *Environ Health*, Aug 21 2026, Vol. 4, no. 8, p. 1664-1673.

Microplastics (MPs) are ubiquitous environmental pollutants that accumulate in human liver tissue, yet their hepatotoxicity in humans remains incompletely defined. To investigate the association between occupational MPs exposure and hepatic dysfunction in humans, we recruited 141 participants from the Health Examination Centre of Guangxi Workers' Hospital, China, including 43 plastic-factory workers with >/=6 months of occupational exposure and 98 occupationally unexposed controls. An eight-item questionnaire-derived exposure risk score (0-8) was used to quantify MPs exposure levels. Linear and logistic regression models, adjusted for smoking, alcohol consumption, dust exposure, body mass index (BMI), and physical activity, were employed to analyze relationships between exposure (occupational status/risk score) and liver-function markers. Exposed workers exhibited higher risk scores (1.9 +/- 1.1 vs 1.4 +/- 0.9, P = 0.013) and twice the prevalence of abnormal liver function (48.8% vs 26.5%, P = 0.013). Each one-point increase in the risk score was associated with a significant elevation in aspartate aminotransferase (AST) by 2.82 U/L (beta = 2.82, 95% CI: 0.27-5.38,P = 0.031) and total bilirubin (T-Bil) by 1.05 mumol/L (beta = 1.05, 95% CI: 0.34-1.76,P = 0.004). Occupational exposure independently predicted abnormal liver function (OR = 3.97, 95% CI: 1.64-10.3, P = 0.041), with longer exposure duration linked to higher AST elevation rates. These results demonstrate that occupational MPs exposure impairs liver function, providing evidence for health-risk assessment of microplastics.
https://doi.org/10.1021/envhealth.5c00716

Dietary Microplastic Exposure in Athletes: Implications for Metabolism, Gut Health, and Performance,
Meccariello, R., Tafuri, M. G. and D'angelo, S., *Nutrients*, Jul 22 2026, Vol. 18, no. 14.

Microplastics (MPs) are emerging environmental contaminants increasingly detected in foods, beverages, and food-contact materials, making dietary intake a relevant route of human exposure. In sports nutrition, this issue may be particularly important because athletes often have high food and fluid consumption, frequent use of packaged sports nutrition products, dietary supplements, bottled beverages, and sport-specific hydration strategies. This narrative review, supported by a

structured literature search, examines dietary MP exposure and its potential relevance to gastrointestinal function, gut microbiota, oxidative stress, inflammation, mitochondrial activity, endocrine regulation, metabolism, recovery, adaptation, and performance-related outcomes in athletes. Current evidence suggests that MPs and nanoplastics may interact with biological systems through mechanisms involving intestinal barrier disruption, microbiota alterations, inflammatory activation, oxidative damage, mitochondrial perturbation, endocrine-disrupting chemicals, and metabolic dysregulation. However, most available data derive from in vitro studies, animal models, food contamination analyses, exposure-estimation studies, and indirect human biomonitoring evidence. Direct studies in athletic populations are currently lacking. Therefore, the possible implications of MP exposure on recovery, adaptation, and exercise performance should be interpreted as biologically plausible but unproven. From a practical perspective, evidence-informed strategies may include reducing avoidable plastic-related exposure while maintaining adequate hydration, energy availability, nutrient timing, supplement quality, and dietary patterns that support antioxidant defenses, inflammatory balance, gut health, and physiological resilience. Future research should prioritize standardized exposure assessment, validated biomarkers, human biomonitoring, and sport-specific studies evaluating MP exposure in relation to physiological and performance-related outcomes. https://doi.org/10.3390/nu18142398

Additive fingerprinting of airborne plastic-related particulate matter in occupational environments: Insights from targeted and untargeted analysis,
Moneta, B. G., Balducci, C., Santoro, S., Perilli, M., Pietrodangelo, A., Sargolini, T., Giusto, M., Rossi, T., Pomata, D. and Cerasa, M., *J Hazard Mater*, Sep 1 2026, Vol. 515, no. 1873-3336 (Electronic), p. 143100.

This study aimed to assess the potential of polymer additives as chemical markers of plastic-related emissions in airborne particulate matter in occupational environments, within the broader context of airborne micro- and nanoplastics (MNPs). Particulate matter collected in three facilities- a textile production (FTX, Facility - Textile), a mineral water bottling plant (FBW, Facility-Bottling Water) and a tyre service and repair facility (FTR, Facility - Tyre Repair)-was investigated to explore the relationship between polymer additives and particulate composition. Samples from multiple sites and size fractions were analysed using a combined targeted and high-resolution untargeted GC-MS approach to characterise plastic additives (e.g. phthalates, alternative plasticisers, antioxidants, UV filters) and related organic compounds. Distinct chemical fingerprints were observed for each facility: FTX was characterised by high-molecular-weight phthalates and terephthalates, with mean concentrations of 5.8 ng/m(3) for DEHP and 4.3 ng/m(3) for diethyl terephthalate. FBW was dominated by citrate- and adipate-based plasticisers, where acetyl tributyl citrate and di(2-ethylhexyl) adipate showed a mean concentration of 9.4 ng/m(3) and 1.7 ng/m(3), respectively. FTR showed higher levels of tyre-derived antioxidants and rubber markers, such as tris(2,4-di-tert-butylphenyl) phosphate (mean: 20 ng/m(3)), 6PPD (3.3 ng/m(3)), and benzothiazole (1.5 ng/m(3)). Size-resolved analysis showed enrichment of additives in fine fractions at FTX and in coarse fractions at FTR, reflecting process-specific emission mechanisms. Untargeted screening revealed a complex mixture including auxiliary materials, lubricants, surface treatments and other process-related compounds, providing a comprehensive characterisation of indoor particulate composition. Overall, the results demonstrate that the combined targeted and untargeted chemical profiles reflect site-specific processes and materials, allowing the discrimination of different emission scenarios across the investigated facilities. https://doi.org/10.1016/j.jhazmat.2026.143100

Occupation and serum concentrations of per- and polyfluoroalkyl substances: data from the 2013 to 2014 National Health and Nutrition Examination Survey.,
Ojo, A. a.-O., Heavner, K. a.-O., Bello, D. a.-O., Li, W. a.-O. and Bello, A. a.-O. X., *Ann Work Expo Health*, no. 2398-7316 (Electronic).

BACKGROUND: Occupational exposure to per- and polyfluoroalkyl substances (PFASs) is a growing concern, as workers may experience higher exposures compared to the general population. However, the contribution of occupational exposure to PFAS on overall body burden remains understudied. OBJECTIVE: This study aims to identify occupational groups with high PFAS body burden based on the data from 2013 to 2014 National Health and Nutrition Examination Survey (NHANES) and assess potential health risks using the National Academy of Sciences, Engineering, and Medicine (NASEM) guidelines for clinicians. METHODS: Serum concentration of 12 PFAS compounds among U.S. residents aged ≥16 yr was obtained from the 2013 to 2014 NHANES (N = 2099), which provides the most recent cycle with detailed information on current and longest jobs. Occupational history was obtained from 2010 U.S. Bureau of the Census Industrial & Occupational Classification coding system reported in the NHANES dataset. Occupations and industries associated with the highest PFAS body burden were determined and categorized into 3 risk groups per NASEM thresholds (<2, 2 to 20, and >20 ng/mL) for the sum of 7 PFAS. Survey-weighted linear regressions were conducted to compare PFAS levels across 23 occupational/industry groups, stratified by gender and age. RESULTS: Higher total PFAS concentrations (sum of all detectable PFAS) were observed for both current and longest jobs (GMs: 12.4 to 14.4 ng/mL) in construction/extraction, installation/maintenance/repair, and arts/design/entertainment/sports/media occupations. PFOA, PFOS, PFHxS, and PFNA accounted for >70% of total PFAS body burden. Over 20% of participants exceeded the NASEM high-risk threshold (≥20 ng/mL) in installation/maintenance/repair, construction, manufacturing, durable goods, and transportation/warehousing, with GMs ranging from 28.6 to 37.6 ng/mL. Elevated PFAS levels were observed in construction and installation/maintenance/repair groups in adjusted models, relative to their respective reference groups. Significantly elevated associations were most pronounced among adults aged 30 to 64 and among females in installation/maintenance/repair compared with sales. CONCLUSIONS: The observed differences in PFAS serum levels, including elevated body burdens among workers in construction/extraction, arts/design/entertainment/sports/media, and installation/maintenance/repair, underscore the need for targeted biomonitoring and exposure intervention among these occupational groups. https://doi.org/10.1093/annweh/wxag052

Occupational heavy metal exposure and endocrine-metabolic biomarkers: a retrospective analysis of 34,595 screened individuals in Türkiye. ,
Or Koca, A., Cesur, M., Çakal, E., Akkale, T. and Yetkin, İ., *BMC Endocr Disord*, no. 1472-6823 (Electronic).

BACKGROUND: Heavy metals and trace elements can act as endocrine-disrupting chemicals and have been linked to thyroid, glucose, lipid, and hepatic abnormalities. We aimed to explore associations between routinely measured metal concentrations and biochemical/endocrinological parameters in a large occupational screening dataset from Türkiye. METHODS: Records from 37,763 individuals evaluated at a national occupational and environmental diseases hospital between November 2021 and October 2023 were retrospectively reviewed; after prespecified exclusions, 34,595 unique participants were included. Metal measurements were performed in whole blood, serum, and urine samples as requested in routine surveillance; for each analyte, results were categorized as within or above the interpretive limit by the laboratory information system. Primary comparisons focused on arsenic, cadmium, lead, manganese, and zinc because of occupational relevance and sample size. Routine biochemistry, lipid profile, and thyroid function tests were

extracted when available, and estimated glomerular filtration rate (eGFR, CKD-EPI) was calculated. RESULTS: Among participants with available thyroid function tests (n = 9,956), thyroid dysfunction was observed in 14.3% (subclinical hypothyroidism 11.9%). Among participants with available fasting glucose and/or HbA1c data (n = 20,738), diabetes mellitus and prediabetes criteria were met in 12.8% and 10.0%, respectively. In univariable comparisons, several metals above the interpretive limit were associated with differences in metabolic biomarkers, including lower HDL with elevated cadmium, lower HDL/LDL with elevated manganese, and higher ALT/AST and triglycerides with elevated zinc. Elevated metal strata differed in age and sex distribution. After within-metal false discovery rate correction, only a limited subset of biomarker differences remained significant. CONCLUSION: Among tested participants, thyroid dysfunction and dysglycemia were observed, and selected metal measurements above interpretive limits were associated with lipid and liver enzyme patterns. Given the clinically driven testing and limited covariate data, these findings should be interpreted as exploratory surveillance signals rather than workforce-level prevalence estimates. Heavy metal exposure may exert endocrine-disrupting effects on workers in relevant industries, particularly on thyroid function and glycemic regulation, suggesting that periodic surveillance of these parameters is warranted in exposure-prone workforces. https://doi.org/10.1186/s12902-026-02388-7

Urinary biomarkers of oxidative stress and their association with isocyanate and epoxy exposure in industrial painters,
Patel, P., Bello, A., Palacios, N. and Bello, D., *International Journal of Hygiene and Environmental Health*, 2026/07/01/ 2026, Vol. 276, p. 114854.

Background Industrial painters (IPs) are routinely exposed to complex mixtures of reactive chemicals, including isocyanates and epoxy resins used in metal coating systems and spray polyurethane foam (SPF) applicators. Oxidative stress (OS), resulting from an imbalance between reactive oxygen species and antioxidant defenses, is a key mechanism of toxic injury linked to numerous diseases like cancer, asthma, dermatitis, and neurotoxicity. However, data on OS markers among IPs are limited. Objectives To characterize urinary OS biomarker concentrations in IPs and SPF applicators and evaluate their associations with urinary biomarkers of isocyanate and epoxy exposure. Methods Pre- and post-shift urine samples were collected from 50 workers applying either isocyanate-based top-coats or epoxy-based mid-coats and SPF. Nine urinary OS biomarkers were analyzed to reflect DNA/RNA damage (8-hydroxy-2'-deoxyguanosine [8OHdG], 8-hydroxyguanosine [8OHG], 5-hydroxymethyluracil [5OHMeU]), protein oxidation (o-tyrosine, 3-chlorotyrosine, 3-nitrotyrosine), and lipid peroxidation (8-isoprostane, 4-hydroxynonenal [4-HNE], malondialdehyde [MnDAI]). Associations between OS and exposure biomarkers were evaluated using multivariable linear regression adjusted for creatinine and age. Results Top-coat painters exhibited significant post-shift increases in 8OHdG, 8OHG, and 3-nitrotyrosine, whereas o-tyrosine and 4-HNE decreased. After adjustment, 8OHdG was positively associated with isocyanate (top-coat) and epoxy (mid-coat) exposure, while 4-HNE was inversely associated with isocyanate exposure. Workers with severe dehydration (specific gravity >1.030) had significantly higher composite OS scores. Conclusions Exposure to reactive coating formulations in IPs and SPF induces measurable oxidative stress, likely via multiple mechanisms, including depletion of the antioxidant pools, impaired antioxidant defenses and direct attack on sensitive biomolecules. Elevated urinary OS biomarkers and their association with exposure metrics highlight the need for improved exposure controls and increased risk of chronic disease development in this workforce. https://doi.org/10.1016/j.ijheh.2026.114854

Parental occupational exposure to pesticides at birth and risk of adult testicular germ cell tumours in offspring: a French nationwide case-control study,
Paul, A., Danjou, A., Coste, A., Kromhout, H., Lefevre, M., Spinosi, J., Dananche, B., Olsson, A., Lamouroux, C., Guth, M., Beranger, R., Perol, O., Boyle, H., Hersant, C., Loup-Cabaniols, V., Veau, S., Perrin, J., Schuz, J., Charbotel, B., Fervers, B. and Group, T. S., *Occup Environ Med*, Jul 22 2026, Vol. 83, no. 4, p. 195-203.

OBJECTIVE: To assess the association between parental occupational pesticide exposures at birth and adult testicular germ cell tumour (TGCT) by histological subtype and the agreement between two pesticide job-exposure matrices (JEM). METHODS: TGCT cases (n=454) and matched controls (n=670) aged 18-45 years were recruited from 20 French university hospitals into the TESTIS national case-control study. Paternal and maternal jobs at birth were obtained from interviews of participants and their mothers/relatives and coded into official nomenclatures (International Standard Classification of Occupations, French nomenclature of activity-1999). Two complementary JEMs, ALOHA+ and FRIJEM, assessed occupational exposure to all pesticides, herbicides, insecticides, fungicides with ALOHA+ for each parent by linking job titles to JEM-derived probability and intensity estimates (no/low/high). Cohen's Kappa coefficient (kappa) assessed their agreement. ORs and 95% CIs comparing exposure and levels of exposure with no exposure were estimated using conditional logistic regression adjusted for literature-based covariates statistically associated with TGCT and stratified by histological subtypes. RESULTS: Agreement between ALOHA+and FRIJEM was moderate to substantial (kappa 0.52-0.80). A statistically non-significant higher TGCT risk was observed for high paternal occupational pesticide exposure with ALOHA+ (OR 1.95, CI 0.98 to 3.84) and FRIJEM (OR 1.70, CI 0.88 to 3.28). With ALOHA+, statistically significant positive associations were seen for high paternal occupational exposure to herbicides (overall: OR 4.23, CI 1.75 to 10.22; seminomas: OR 4.78, CI 1.79 to 12.77; non-seminomas: OR 3.53, CI 1.21 to 10.3) and fungicides (overall: OR 2.09, CI 1.05 to 4.18; seminomas: OR 2.31, CI 1.02 to 5.23). No statistically significant associations were observed for other levels of paternal exposure or pesticide groups nor for maternal exposure. CONCLUSION: Only high paternal occupational exposure to pesticides at birth, especially herbicides and fungicides, was positively associated with TGCT. https://doi.org/10.1136/oemed-2024-109893

Industrial Hydrocarbons Exposure Deteriorates Sperm Integrity and Reproductive Function,
Pichandi, M. K., Ranganathanan, B. and Abilash, D., *Journal of Biochemical and Molecular Toxicology*, Aug 3 2026, Vol. 40, no. 8.

Occupational exposure to polycyclic aromatic hydrocarbons (PAHs) or metal fumes in the workplace may be detrimental to male fertility. The present study compared sperm concentration, total motility, strict morphology and oxidative stress and SDF levels between 81 men, controls (n = 17) and workers from plastic industry workers (n = 18), steel EAF process (n = 15), waste incineration plants (n = 16) or aluminium factories smelting in the same plant for all workplaces involved in this cross-sectional research. In all treated groups, the sperm concentration, motility, normal morphology, acrosome integrity and membrane function dropped significantly compared with those in controls ($p < 0.001$). Elevated ROS, MDA and low SOD and CAT activities confirmed the presence of severe oxidoreductive imbalance, particularly in Aluminium and incineration workers. DNA breaks assayed by SCD and comet showed a clear positive exposure-response relationship with the highest OTM in aluminium workers. Network pharmacologic work showed that 67 shared targets were regulated by PAH exposure and male infertility. The hub gene analysis pointed at EGFR, ESR1, RELA, PTGS2, MMP2 and MMP9 as the core hub genes while KEGG enrichment suggested that the HIF-1 and TNF-related inflammatory pathway, along with the endocrine disruption side of stress activating signalling, was involved. Inflammatory signal activation and endocrine disturbance together seemed to end up with really severe damage to sperm DNA, and it also turned on a bunch

of mechanisms through which these chemical carcinogens can exert effect. ROS and MDA were negatively correlated with motility as well as sperm concentration significantly ($p < 0.05$), whereas antioxidant enzymes showed a positive correlation. These findings show how PAH-related industrial poisoning disrupts important reproductive output through ROS-mediated genetic instability and suggest the relevance of protection measures in high-risk industries.
https://doi.org/10.1002/jbt.71052

A simple and sensitive QuEChERS-based LC-MS/MS method for quantifying 30 per-and polyfluoroalkyl substances (PFAS) in human serum: application to firefighter biomonitoring, Sweeney, C. L., Suwanratanabus, R., Seyedsadjadi, N., Nguyen, D., Rennert, J. and Masoud, F., *Talanta*, Feb 1 2027, Vol. 312.

Occupational exposure to per-and polyfluoroalkyl substances (PFAS) is under scrutiny as a potential cancer risk for firefighters. Existing methods for PFAS biomonitoring involve solid-phase extraction (SPE), either conventional or online, followed by liquid chromatography-tandem mass spectrometry (LC-MS/MS). SPE requires a significant investment of time and technical skill for optimizing many variables to achieve maximum accuracy and precision. This increases analytical costs, making routine PFAS biomonitoring cost-prohibitive for a majority of fire service members. The motivation for this study was to develop an analytical method for measuring 30 PFAS in firefighters' serum (N = 84) using a QuEChERS (quick, easy, cheap, effective, rugged, and safe) protocol. For all target analytes, process efficiency (PE) and recovery efficiency (RE) were between 89 and 126% at 1 & micro;g L-1 and between 85 and 133% at 25 & micro;g L-1 (%RSD <= 13) (N = 5). At 1 and 25 & micro;g L-1, matrix effects (ME) for all target PFAS ranged from 78 to 110% and 87 to 117%, respectively (%RSD <= 11). Precision criteria were met for all target analytes in both intra-day and inter-day precision assessments (%RSD <= 19). Limits of detection (LOD) and quantitation (LOQ) ranged from 0.006 to 0.3 & micro;g L-1 and 0.02 to 0.9 & micro;g L-1, respectively. This highperformance, high-throughput, and cost-effective alternative to the current gold standard for PFAS measurement in serum advances current firefighter biomonitoring capabilities by decreasing the time and cost of analysis, reducing constraints imposed by specialized laboratory equipment and expertise, and optimizing analytical batch processing and workflow.
https://doi.org/10.1016/j.talanta.2026.130400

Widespread bisphenol S analogues in E-waste recycling dust and air: Gas–particle partitioning behavior and human exposure implications, Xie, R., Du, B., Zeng, L. and Pan, Y., *Journal of Hazardous Materials*, 2026/08/01/ 2026, Vol. 514, p. 142909.

Bisphenol S (BPS) analogues are increasingly used as alternatives to bisphenol A (BPA), but their occurrence, phase behavior, and exposure implications in e-waste recycling environments remain poorly characterized. Here, we conducted an integrated investigation of BPS analogues (BPSs) in indoor dust and paired gas- and particle-phase air samples from e-waste dismantling workshops in South China, to characterize their occurrence profiles, gas–particle partitioning, endocrine-disruption screening, and occupational exposure risks. BPS and 11 analogues were detected in dust, while BPS and 10 analogues were detected in indoor air. ∑12BPSs ranged from 48.1 to 2120 ng/g in dust and 135–1790 pg/m³ in air. DBSP and BPS dominated dust, whereas DBSP dominated the particulate phase and DPS dominated the gaseous phase. Notably, DPS was the only compound detected in all gas-phase samples, indicating distinct phase preference. Gas–particle partitioning coefficients correlated significantly with predicted subcooled liquid vapor pressures and octanol–air partition coefficients ($p < 0.01$). EDC-Predictor screening indicated endocrine-related interaction potential for several analogues, particularly DBSP and DPS. Estimated daily intakes via dust

ingestion and inhalation reached 3.32 ng/kg bw/day under the high-end scenario, higher than the recently revised BPA TDI in a screening-level comparison. These findings reveal that overlooked BPS substitutes, particularly DBSP and DPS, act as phase-specific contributors to occupational exposure in e-waste workshops. https://doi.org/10.1016/j.jhazmat.2026.142909

Leaching kinetics and release mechanisms of microplastics/nanoparticles from medical personal protective equipment into physiological fluids: A hidden occupational hazard,
Xie, Y., Wang, Y., Kong, Z., Han, R. and Ding, T., *J Hazard Mater*, Aug 1 2026, Vol. 514, no. 1873-3336 (Electronic), p. 142741.

Currently, research on microplastics (MPs) primarily focuses on natural environmental media, while medical waste and personal protective equipment are emerging as overlooked sources of MPs and nanoparticles release. Research on personal protective equipment as a source of MPs remains limited. This study quantified MPs release from ten types of personal protective equipment into body fluid and sweat. Our work provides the first systematic analysis of dynamic MPs release from personal protective equipment under surgical procedures. Total release in sweat substantially exceeded that in body fluid. MPs release was governed by material structure, liquid medium, and exposed area. Although high-porosity materials exhibited elevated unit release, actual exposure depended on usable surface area. The released MPs contained multiple polymer types, primarily in the form of fragments and fibers. Mean particle sizes were 57.17 +/- 16.75 mum in body fluid and 45.00 +/- 12.34 mum in sweat, indicating smaller particles prevalence. Average nanoparticle concentrations reached 5.2 x 10(7) particles/mL in body fluid and 7.2 x 10(7) particles/mL in sweat. Leaching kinetics demonstrated first-order diffusion in nonwovens (R(2)>0.97), whereas coated fabrics exhibited zero-order kinetics at 1.2 x 10(7) particles(/)mL.h(-1). Health-risk assessment indicated non-carcinogenic hazard indices and carcinogenic risk below safety thresholds. However, both models estimated high annual exposure levels for healthcare professionals, suggesting potential long-term health risks that warrant further investigation. Analysis of the release mechanism reveals that non-woven materials are governed by internal diffusion, whereas surface-coated materials follow a surface-controlled release pattern. These findings provide critical data on MPs release and associated occupational risks. https://doi.org/10.1016/j.jhazmat.2026.142741

Epidémiologie

Mapping Steroidogenic Perturbations Under Endocrine Disruptor Mixtures Across Demographic Subgroups: Structural and Metabolomic Insights,
Chen, Y. L., Huang, L., Jiang, Y. T., Zhao, G. H., Zhu, M. Y., Lu, X. L., Lang, L., Esther, A. O., Fan, L. H., Jiang, Y. Q., Sun, C., Zhang, X. L., Zhou, K., Ji, X. M. and Chen, M. J., *Advanced Science*, Aug 2026, Vol. 13, no. 46.

Endocrine-disrupting chemicals (EDCs) are pervasive environmental chemicals affecting hormone homeostasis, yet mapping mixture exposures to mechanisms at the population scale remains challenging. Here, analyses of 4255 U.S. participants are integrated with computational and experimental validation, linking population signals to molecular targets. Profiling 37 EDCs reveals consistent associations between phthalate metabolites and lower testosterone, strongest in adult males. Mono-(3-carboxypropyl) phthalate (MCPP) is identified as a key disruptor and is shown to inhibit CYP17A1, consistent with engagement of the enzyme's iron-protoporphyrin IX (heme) center. Docking/molecular dynamics prioritizes this interaction, which is supported by surface plasmon resonance, multi-matrix metabolomics in vivo and in vitro, and functional assays in primary Leydig cells. An interactive web resource (https://chembio.njmu.edu.cn/edc-hormone-explorer.html)

provides open access to the association landscape and supports risk prioritization. https://doi.org/10.1002/advs.76381

Prenatal occupational exposure to quaternary ammonium compounds and birthweight: findings from a pilot study using meconium as a biological matrix,
Fisher, S., Pavilonis, B., Harari, H., Lahage, N., Gupta, A., Aguilar, D. and Mcdermott, S., *Journal of Perinatology*, 2026/08/24 2026.

To evaluate prenatal exposure to quaternary ammonium compounds (QACs) using meconium analysis and assess associations with newborn birthweight. https://doi.org/10.1038/s41372-026-02884-7

Association between bisphenol exposure and polycystic ovary syndrome risk: an integrated systematic review and meta-analysis,
Gui, Z. Y., Lin, H. Z., Wang, C. H. and Yao, Y. S., *Frontiers in Endocrinology*, Jun 11 2026, Vol. 17.

Background: Polycystic ovary syndrome (PCOS) is a prevalent endocrine disorder associated with environmental endocrine-disrupting chemicals, particularly bisphenols (BPs) such as bisphenol A (BPA) and bisphenol S (BPS). BPs may disrupt reproductive and metabolic pathways through estrogenic, anti-androgenic, and obesogenic effects, contributing to the pathogenesis of PCOS. However, the epidemiological evidence remains inconsistent. Methods: This PRISMA-guided systematic review and meta-analysis evaluated observational studies (cross-sectional, case-control, longitudinal) from PubMed and Web of Science up to December 2024. Seventeen studies involving 3, 010 women were included after screening. The quality of the studies was assessed using the Newcastle-Ottawa Scale. Standardized mean differences (SMDs) with 95% confidence intervals (CIs) were calculated using random-effects models due to high heterogeneity (I & sup2; > 50%). Subgroup analyses were conducted to explore the sources of heterogeneity. Result: The meta-analysis revealed significant positive associations between PCOS risk and serum BPA (SMD = 1.32, 95% CI: 0.83-1.82), urinary BPA (SMD = 2.69, 95% CI: 1.41-3.97), and serum BPS (SMD = 0.26, 95% CI: 0.07-0.46). Heterogeneity was high for BPA (I & sup2; = 95-99%) but absent for BPS (I & sup2; = 0%). Sensitivity analyses excluding lower-quality studies confirmed the robustness of the findings. Subgroup analyses utilizing the Rotterdam criteria slightly attenuated the associations for BPA. Funnel plots indicated no significant publication bias. Conclusion: Exposure to BPs, represented by BPA and BPS, was significantly associated with an increased risk of PCOS, likely mediated by endocrine disruption, metabolic dysregulation, and epigenetic mechanisms. These findings underscore the necessity for regulatory policies aimed at reducing population-level exposure to BPs, the clinical integration of BPs biomonitoring in the management of PCOS, and further research on gene-environment interactions and non-monotonic dose effects. Addressing exposure to BPs may offer novel preventive and therapeutic strategies for PCOS.
https://doi.org/10.3389/fendo.2026.1859611

Paternal Exposure to Endocrine Disrupting Chemicals and Couples' Pregnancy Outcomes: A Qualitative Review of the Literature,
Han, H., Hauser, R. and Mínguez-Alarcón, L., *Current Environmental Health Reports*, Aug 14 2026, Vol. 13, no. 1.

Purpose of Review Endocrine-disrupting chemicals (EDCs) are ubiquitous, and exposure to them has been associated with adverse reproductive health in both animals and humans. While the adverse effects of maternal EDC exposures on pregnancy outcomes have been extensively investigated, an increasing body of literature is now examining the role of paternal exposure, given its potential

impact on sperm epigenetics. This review summarizes human studies examining associations between paternal exposure to several EDCs and couples' pregnancy outcomes.Recent Findings Biomarker-based studies suggest potential negative associations between paternal preconception exposure to certain phthalates and couples' pregnancy outcomes, whereas studies for bisphenol A and parabens have shown little to no associations. The evidence regarding benzophenones, per- and polyfluoroalkyl substances, and flame-retardant chemicals remains limited and inconclusive.Summary Given the critical knowledge gaps in this field, additional couple-based studies with repeated preconception exposure measurements are needed to clarify the effects of paternal exposure to individual EDCs and their mixtures on couples' pregnancy outcomes. Additional studies on potential biological mechanisms that may explain these associations (e.g., sperm epigenome) are also warranted. https://doi.org/10.1007/s40572-026-00550-w

Exploring the association between environmental exposure to phthalates and bisphenol A and changes in reproductive hormones in women: a cross-sectional study,
Hashemi, F., Hoepner, L., Hamidinejad, F. S., Omeragić, E., Haluza, D., Hashemi, H., Esmaeili, A., Guo, C., Stanciu, G., Kabiri, B. and Seidhani-Nahal, A., *Scientific Reports*, 2026/08/23 2026.

Endocrine-disrupting chemicals (EDCs) can harm the female reproductive system by disrupting hormonal balance. This study investigates the association between exposure to EDCs)- specifically, phthalates and bisphenol A (BPA), and serum concentrations of estrogen (ES) and progesterone (P) in women. A comparative analysis was conducted on a group of 90 women, comprising an exposed group (n = 50) and a control group (n = 40). The relationships between 11 urinary phthalate metabolites, BPA, and reproductive hormone levels were analyzed using Spearman correlation, linear regression, and Bayesian Kernel Machine Regression (BKMR). The application of BKMR enabled the modeling of the overall mixture effect of the EDCs. This approach further identified non-linear dose–response relationships and delineated specific lifestyle and behavioral factors that modify the association between EDCs exposure and hormone concentrations. The findings showed that there was a positive association between BPA (β = 0.045; p < 0.05), MBP (β = 0.042; p < 0.05), ∑DEHP (β = 0.041; p < 0.05) with estrogen. Every unit increase in ln-transformed BPA, MBP, and ∑DEHP was associated with 0.045, 0.041, and 0.04 ln-unit increases in estrogen, respectively. Also, there was a positive and significant association between MBP, MEP, and BPA (p < 0.05) with progesterone. The BKMR analysis indicated a significantly positive overall effect on ES and P concentrations with high phthalate metabolites and BPA concentrations. The exposure variables showed that there was a significant positive relationship between smoking, consuming bottled water, drinking bottled beverages, consumption of ready-to-eat foods, and using cosmetics with ∑DEHP and BPA (p < 0.05). The findings indicate associations between exposure to phthalates and BPA and hormone imbalances that may compromise women's health as one of the vulnerable groups. https://doi.org/10.1038/s41598-026-67569-9

Prenatal exposure to a mixture of non-persistent endocrine-disrupting chemicals and attention and hyperactivity symptoms in children in the ECHO cohort,
Herrera, T., Hyman, S., Newman, R., Dickerson, A. S., Quirós-Alcalá, L., Bennett, D. H., Oh, J., Huerta-Montañez, G., Titus, A. R., Díaz, I., Watkins, D. J., Trasande, L., Baker, B. H., Ferrara, A., Cassidy-Bushrow, A. E., Hazlehurst, M. and Ghassabian, A., *Environment International*, 2026/08/29/ 2026, p. 110488.

Objective To examine the association between prenatal exposure to a mixture of EDCs and attention-deficit/hyperactivity disorder (ADHD) symptoms in children Methods Participating were mother–child pairs from the Environmental influences on Child Health Outcomes (ECHO) Cohort (n = 3,962, 18 Sites, 2001–2021). Maternal spot urine samples collected during pregnancy were sent

to the Wadsworth Center Human Health Exposure Analysis Resource laboratory and analyzed using a single assay workflow. From those samples, 24 urinary metabolites of EDCs with detection frequency > 70%, were grouped as molar sum of high- and low-molecular-weight (HMW, LMW) phthalates, polycyclic aromatic hydrocarbons, bisphenol S, organophosphate esters, and organophosphate and pyrethroid pesticides. Child ADHD symptoms were assessed using raw scores from the Child Behavior Checklist for preschool-aged (CBCL/1½-5) and school-aged (CBCL/6–18) Attention-Deficit/Hyperactivity Problems subscale. Scores ≥ the 85th and 95th percentiles were respectively classified as borderline and clinical thresholds. Quantile g-computation estimated the expected change in ADHD outcome associated with a one-quartile increase in all prenatal EDC mixture concentrations, using negative binomial models for raw scores and binomial models for percentile thresholds. Results Prenatal exposure to the EDC mixture was not associated with child ADHD symptoms in preschool-aged or school-aged children in adjusted models for either continuous raw scores or percentile thresholds. Conclusion Overall our findings on prenatal EDC mixture exposure and ADHD symptoms were not robust to full adjustment. Future research that can delineate factors such as windows of vulnerability and trajectories of ADHD-related symptoms may help clarify mixed findings in the literature. https://doi.org/10.1016/j.envint.2026.110488

Associations of bisphenol mixture exposure with glucose metabolism during mid-pregnancy: A focus on emerging substitutes in a Chinese birth cohort,
Huang, Z. H., Zhang, A. H., Zhu, M., Zhai, Q., Dou, L. J. and Mei, J. Y., *Food and Chemical Toxicology*, Sep 2026, Vol. 215.

With the widespread use of bisphenol A (BPA) substitutes such as BPS and BPF, pregnant women are commonly exposed to complex mixtures of these endocrine-disrupting chemicals. However, epidemiological evidence on the combined effects of bisphenol mixtures on gestational glucose metabolism remains limited. This study included 1258 pregnant women from the Wuhu Birth Cohort in China. Serum concentrations of BPA, BPS, BPF, and BPAF were measured using UHPLC-MS/MS. Glucose metabolism was assessed by 75-g OGTT measuring fasting, 1-h, and 2-h plasma glucose. We employed multivariable linear regression for single-chemical analysis and three advanced mixture models-Weighted Quantile Sum (WQS) regression, Quantile-based g-computation (Qgcomp), and Bayesian Kernel Machine Regression (BKMR)-to evaluate the joint effects. All four bisphenols were frequently detected, with BPS showing 100% detection rate. Single-chemical models showed significant associations of BPS and BPF with elevated glucose at all time points (e.g., for 1-h glucose and BPS: (3 = 0.371 mmol/L per SD increase in ln-BPS, 95% CI: 0.218-0.524). The mixture analysis consistently demonstrated significant joint effects: WQS showed a positive association between the mixture index and all glucose measures (e.g., (3 = 0.129, 95% CI: 0.058-0.200 for 1-h glucose); Qgcomp indicated that a simultaneous quartile increase in all bisphenols was associated with a 0.343 mmol/L rise in 1-h glucose (95% CI: 0.179-0.507); BKMR revealed an approximately linear increase in glucose levels with mixture exposure. Across all mixture models, BPS consistently emerged as the primary contributor, with the highest weights in WQS (up to 0.542) and posterior inclusion probabilities approaching 1.0 in BKMR. Our study demonstrates that exposure to bisphenol mixtures significantly impairs mid-pregnancy glucose metabolism, with BPS and BPF serving as the predominant risk contributors. These findings underscore the necessity of mixture-based exposure assessment and support the inclusion of BPA substitutes in future public health regulations. https://doi.org/10.1016/j.fct.2026.116212

Environmental Toxicants and Polycystic Ovary Syndrome: Implications for a Metabolic-Ovarian Framework (PMOS) - A Systematic Review,
Ibibama, J. O., Madaki, P. D., Aliche, K. A., Frazzoli, C. and Orisakwe, O. E., *Biological Trace Element Research*, 2026.

Background: Polycystic ovary syndrome (PCOS) is increasingly recognized as a systemic condition characterized by intricate endocrine and metabolic dysregulation. Emerging evidence suggests that environmental contaminants may drive its pathogenesis, supporting an expanded conceptualization of the disorder as polycystic metabolic-ovarian syndrome (PMOS). This systematic review aimed to evaluate the association between exposure to potentially toxic elements (PTEs) and endocrine disruptors and the reproductive, hormonal, and metabolic clinical features of PCOS. Methods: Following PRISMA guidelines, a systematic search was conducted across PubMed, Embase, Scopus, and Web of Science for human observational studies published up to 31 December 2025. Methodological quality was appraised using the Newcastle-Ottawa Scale (NOS). Due to heterogeneity in biological matrices and toxicant classes, a narrative synthesis was performed. Results: Fourteen high-quality studies (NOS scores 8-9) from seven countries met the inclusion criteria. The evidence consistently linked exposure to endocrine disrupting chemicals specifically bisphenol A (BPA) and phthalates-with hyperandrogenism, altered luteinizing hormone/follicle-stimulating hormone ratios, and impaired ovarian reserve. Furthermore, heavy metals (cadmium, lead, mercury, arsenic) and emerging rare earth elements were significantly elevated in women with PCOS across multiple matrices (serum, urine, and follicular fluid). These exposures were strongly associated with metabolic disturbances, including insulin resistance, obesity indices, and lipid abnormalities, mediated predominantly by oxidative stress pathways and impaired antioxidant defences. Conclusion: Current observational evidence suggests associations between exposure to selected environmental toxicants and endocrine-metabolic features of PCOS. However, heterogeneity in exposure assessment, biological matrices, PCOS diagnostic criteria, and confounder adjustment limits causal interpretation. Future prospective studies with repeated biomonitoring are needed. https://doi.org/10.1007/s12011-026-05218-y

Microplastics-PFAS interactions in environmental matrices: quantitative evidence for antagonistic and synergistic toxicity,
Kuriyal, N. and Nandan, A., *Critical Reviews in Toxicology*, May 28 2026, Vol. 56, no. 5, p. 364-388.

Microplastics (MPs) and per- and polyfluoroalkyl substances (PFAS) increasingly co-occur across aquatic, terrestrial, and biological systems, yet their combined toxicological behavior remains poorly resolved. To address this gap, we conducted a systematic review and meta-analysis of 47 controlled experimental studies encompassing aquatic organisms, soil invertebrates, and mammalian models. Using standardized mean differences (Hedges' g) under a random-effects REML framework, supported by subgroup analysis, meta-regression, and bias diagnostics, we quantitatively compared biological responses induced by MPs, PFAS, and their co-exposure. PFAS produced the strongest and most consistent toxic effects across systems (g = 4.71; 95% CI: 2.32-7.09), with pronounced impacts on oxidative balance, hepatic function, immune regulation, and reproductive performance. MPs also induced widespread biological stress, though with lower and more variable intensity (g = 2.19; 95% CI: -0.29-4.66), affecting oxidative, reproductive, and inflammatory pathways. Notably, co-exposure to MPs and PFAS frequently resulted in attenuated or antagonistic responses rather than additive toxicity (g = -2.98; 95% CI: -8.89-2.93), suggesting that PFAS adsorption onto MP surfaces can reduce bioavailability and tissue uptake. However, synergistic effects emerged under specific conditions, particularly at high PFAS concentrations, with small or weathered particles and prolonged exposure durations. Dose response relationships were evident across exposure categories, with aquatic species displaying the greatest sensitivity. Overall, these findings demonstrate that mixture toxicity is context-dependent and cannot be inferred from single-compound data alone. This study provides a quantitative foundation for advancing mixture-aware environmental risk assessment and regulatory frameworks.
https://doi.org/10.1080/10408444.2026.2686292

Association between gut microbiota composition and liver dysfunction in school-aged children exposed to triclosan,
Li, A. J., Li, M. L., Xu, W., Zhang, Y. H., Yin, S. L., Yu, Y. T., Zheng, S. F., Hu, Y. F. and Song, M. Y., *Journal of Environmental Sciences*, Sep 2026, Vol. 167, p. 308-316.

Growing epidemiological evidence implicate endocrine-disrupting chemicals (EDCs) in the development of liver dysfunction across populations. However, the effects of mixed EDC exposure on children's liver function remains critically limited. This study investigated the association between co-exposure to three prevalent EDCs-bisphenol S, triclosan (TCS), and methylparaben-and liver function, as measured by the aspartate aminotransferase/alanine aminotransferase ratio (AST/ALT), in school-aged children. Using g-computation and Bayesian kernel machine regression, we found consistent positive associations between the EDCs mixture and elevated AST/ALT levels, with TCS emerging as the primary contributor. Stratification by median TCS exposure revealed significant gut microbiota compositional differences between high-and low-exposure groups. Mediation analysis identified Desulfovibrio as a key microbial mediator, explaining 48.5 % of the TCS-associated effect on AST/ALT. Our findings suggest that EDC co-exposure, particularly to TCS, may promote subclinical liver injury in children, potentially mediated through gut microbiome alterations. These results highlight the need for longitudinal studies to confirm these associations and elucidate the underlying mechanisms. https://doi.org/10.1016/j.jes.2025.10.026

Longitudinal associations of per- and polyfluoroalkyl substances with metabolic syndrome from adolescence to early adulthood: A 10-year prospective cohort study,
Lin, C. Y., Lee, H. L. and Su, T. C., *Environmental Pollution*, Oct 15 2026, Vol. 407.

Per: and polyfluoroalkyl substances (PFAS) have been implicated in metabolic dysregulation; however, longitudinal evidence linking PFAS to metabolic syndrome (MS) across the transition from adolescence or young adulthood into early adulthood is still lacking. We analyzed 516 participants aged 12-30 years in the Young Taiwanese Cohort (YOTA) with baseline examinations in 2006-2008 and follow-up in 2017-2019. Eleven plasma PFAS and MS components were quantified at both time points. We used joint models that simultaneously included baseline PFAS and long-term PFAS changes to evaluate associations with follow-up MS and its components. A PFAS exposure index was derived as the mean of standardized ln-PFAS. The baseline PFAS exposure index was not associated with MS or its components. Among individual baseline PFAS, linear PFOA was inversely associated with impaired fasting glucose, and both linear PFOA and PFHxS were inversely associated with elevated blood pressure. In contrast, the PFAS exposure index change was inversely associated with follow-up MS (OR 0.36, q = 0.050) and follow-up MS waist (OR 0.22, q < 0.001). Among individual PFAS changes, follow-up MS was inversely associated with changes in linear PFOA, PFHxS, and PFDoA, and MS waist was inversely associated with changes in linear PFOA, linear PFOS, PFNA, PFDA, PFUdA, and PFDoA. The associations between PFAS exposure index change with MS and MS waist were robust across sensitivity analyses, including qgcomp. Analyses of continuous metabolic traits showed concordant patterns for waist circumference and HDL-C. Exploratory backward models further suggested that adiposity change may influence measured PFAS change. In this Taiwanese cohort, higher Delta PFAS exposure index was associated with lower odds of MS and abdominal obesity, but the findings are consistent with reciprocal coupling rather than a protective effect of PFAS exposure. Studies with three or more repeated measurements are needed to clarify causal direction and underlying mechanisms. https://doi.org/10.1016/j.envpol.2026.128772

Exposure to bisphenols and phthalates and arterial stiffness in women of reproductive age: a New York City cohort analysis,
Ling, R., Seok, E., Liu, M. L., Mehta-Lee, S., Chen, Y., Hausvater, A., Urbina, E., Trasande, L. and Kahn, L. G., *Environment International*, Aug 2026, Vol. 214.

Purpose: This study aimed to examine associations of bisphenol and phthalate exposure with arterial stiffness among women of reproductive age. Methods: Participants include 637 adult women enrolled in the New York University Children's Health and Environment Study (NYU CHES), a prospective pregnancy cohort. The concentrations of eight bisphenols and 22 phthalate metabolites were quantified in up to three spot urine samples. Primary outcomes included brachial artery distensibility (BrachD) and carotid-femoral pulse wave velocity (cfPWV) as measures of peripheral and central arterial stiffness, respectively, along with blood pressure assessed at repeated study visits up to seven years from exposure measures. Associations between averaged, log2-transformed, creatinine-standardized chemical concentrations and subclinical vascular outcomes of interest were examined using linear mixed-effects models. Results: In covariate-adjusted models, a doubling of bisphenol A was positively associated with BrachD (0.09%/ mmHg, 95% confidence interval [CI] = 0.02, 0.16), a doubling of bisphenol S was negatively associated with mean arterial pressure (MAP) (-0.52 mmHg, 95% CI =-0.98,-0.05), a doubling of di(2-ethylhexyl) phthalate (DEHP) metabolites was negatively associated with diastolic blood pressure (DBP) (-0.81 mmHg, 95% CI =-1.38,-0.23) and MAP (-0.83 mmHg, 95% CI =-1.45,-0.21), and a doubling of antiandrogenic phthalate metabolites was associated with 0.62 mmHg lower DBP (95% CI =-1.22,-0.03) and 0.66 mmHg lower MAP (95% CI =-1.30,-0.02). Conclusion: These findings suggest that routine exposure to bisphenols and DEHP may be associated with vasorelaxation, potentially related to these chemicals' estrogenic and/or antiandrogenic effects. https://doi.org/10.1016/j.envint.2026.110420

Association between endocrine-disrupting chemicals and thyroid cancer: a systematic review and meta-analysis,
Liu, Q. H., Deng, X., Zhang, L., Xiao, D., Li, J. and Li, Y. L., *Toxicology Research*, Aug 2026, Vol. 15, no. 4.

This study comprehensively evaluated the epidemiological links between five classes of endocrine-disrupting chemicals-bisphenols, polychlorinated biphenyls, organochlorine pesticides, phthalates, and per- and polyfluoroalkyl substances-and thyroid cancer risk, aiming to lay an evidence-based foundation for clarifying environmental etiology and informing public health policy. A systematic search of databases including PubMed/MEDLINE, Cochrane Library, Web of Science, and Embase was conducted up to July 22, 2025. Meta-analysis was performed using Stata 17.0, with subgroup analyses based on geographical region, pathological subtype, and covariate adjustment for factors like BMI and sex. From 1,365 initially identified articles, 22 were included, encompassing 22,916 cases. The meta-analysis revealed that PAE metabolites MEHP, MEOHP, and MEHHP had significant positive associations with thyroid cancer risk. In contrast, the PFAS perfluorooctanoic acid showed a significant negative association. No significant associations were found for bisphenols, polychlorinated biphenyls, or organochlorine pesticides. Subgroup analyses suggested that region, pathological subtype, sample type, and covariate adjustment may influence the observed associations. In conclusion, specific PAE metabolites significantly increase thyroid cancer risk, representing potential new environmental risk factors, whereas the negative correlation with PFOA requires further mechanistic investigation. The findings highlight the need to strengthen monitoring and intervention regarding PAE exposure and to promote individualized risk early warning based on pathological typing. https://doi.org/10.1093/toxres/tfag055

Urinary phthalate metabolites and impaired cardiorespiratory fitness in a non-diabetic young U.S. population: metabolite-specific associations and age- and sex-related patterns,
Lun, Y., Jiang, R., Yu, S., Guan, X. and Wang, F., *Journal of Hazardous Materials Advances*, 2026/08/01/ 2026, Vol. 23, p. 101526.

Phthalates are ubiquitous endocrine-disrupting chemicals, but their associations with cardiorespiratory fitness (CRF) in youth remain unclear. We analyzed 2,753 non-diabetic participants from the National Health and Nutrition Examination Survey (NHANES) 1999-2004 to examine associations between urinary phthalate metabolites and CRF, defined using estimated maximal oxygen consumption (VO2 max) from submaximal treadmill testing. We used survey-weighted logistic regression, generalized additive models, an environmental risk score (ERS), Nand extreme gradient boosting (XGBoost) to evaluate individual, nonlinear, cumulative, and complementary model-interpretation patterns. In the overall population, higher mono-cyclohexyl phthalate (MCP) concentrations were significantly associated with lower odds of CRF impairment (OR = 0.60, 95% CI: 0.44, 0.82). Higher mono-n-octyl phthalate (MOP) concentrations were nominally associated with higher odds of CRF impairment (OR = 1.34, 95% CI: 1.02, 1.76). In stratified analyses, nominal associations with CRF impairment included positive associations for mono-ethyl phthalate (MEP) in adult females (OR = 1.91, 95% CI: 1.12, 3.24) and mono-isononyl phthalate (MNP) in adult males (OR = 1.67, 95% CI: 1.09, 2.56) and female adolescents (OR = 1.80, 95% CI: 1.05, 3.07), as well as an inverse association for MCP in adult males (OR = 0.50, 95% CI: 0.26, 0.94). A nonminal nonlinear interaction was observed between MNP and MOP, and XGBoost supported age-related heterogeneity in the MEP association. The ERS analysis did not identify a significant cumulative association. Overall, urinary phthalate metabolites showed metabolite-specific and age- and sex-related patterns in relation to CRF impairment, with the strongest statistical support for the overall MCP association and a need for confirmation in prospective studies with improved exposure assessment and external validation.
https://doi.org/10.1016/j.hazadv.2026.101526

Modern Determinants of Earlier Menarche and Mental Health: A Translational Review for Clinicians,
Marano, G., D'abate, C., Sorrenti, G., Traversi, G., Mazza, O. and Mazza, M., *Children*, Aug 12 2026, Vol. 13, no. 8, p. 1068.

Background: Over the past decades, the global onset of menarche has progressively advanced, driven by complex interactions between metabolic, psychosocial, and environmental factors. Beyond its reproductive significance, menarche represents a critical developmental milestone that coincides with increased vulnerability to mental health difficulties. Contemporary determinants such as childhood obesity, early-life adversity, and exposure to endocrine-disrupting chemicals (EDCs) may accelerate pubertal timing while simultaneously shaping adverse psychological trajectories during adolescence. Objectives: This review aims to provide a translational and clinically oriented synthesis of modern determinants of earlier menarche and their associations with mental health outcomes in girls and adolescents, highlighting implications for early identification, prevention, and integrated clinical care. Methods: We conducted a narrative review mapping a scoping literature search conducted using PubMed/Medline, Scopus, and PsycINFO, focusing on literature from the last decade (2016-2026), with emphasis on systematic reviews, meta-analyses, and longitudinal cohort studies. Evidence was synthesized across biological, psychosocial, and environmental domains. Results: Accumulating evidence indicates that higher childhood adiposity, psychosocial stress, and EDC exposure are consistently associated with advanced pubertal timing. Earlier menarche correlates with an elevated risk of depressive symptoms, anxiety, self-harm, and body image-related distress, although effect sizes vary across populations. Emerging longitudinal data suggest that the

peri-menarcheal period represents a critical window for symptom exacerbation. These associations appear to be mediated by biological mechanisms (e.g., hormonal shifts, hypothalamic-pituitary-adrenal (HPA) axis reactivity) and social processes including peer-comparison dynamics and premature sexualization. Conclusions: Earlier menarche acts as a biopsychosocial sentinel event rather than an isolated gynecological milestone. Integrating mental health screening and anticipatory guidance into pediatric and gynecological care is essential for early risk detection. A multidisciplinary, equity-oriented approach is required to address both individual vulnerability and broader environmental determinants. https://doi.org/10.3390/children13081068

Prenatal and childhood exposure to common plasticizers and risk-taking behavior in young adolescents,
Meerts, L., Ghassabian, A., Liu, M. L., Trasande, L., Tiemeier, H., White, T. and El Marroun, H., *Environmental Research*, Sep 15 2026, Vol. 306.

Background: Emerging evidence suggests endocrine disrupting chemicals, including bisphenols and phthalates, may affect behavioral development in children and adolescents. Risk behavior constitutes a potentially sex hormone sensitive behavioral construct. Here, we examined longitudinal associations of phthalate and bisphenol exposure with performance-based tasks and self-reported risk-taking behaviors. Methods: Within a population-based birth cohort in the Netherlands, urinary bisphenols and phthalate metabolite concentrations were measured in women during pregnancy (three times, n = 1379) and in children (once at 6 years, n = 775). At 10 years, child risk-taking behavior was assessed with the computerized experimental Columbia Card Task (CCT). At 14 years, adolescents completed a computerized self-assessment of real-life risk-taking behaviors. Linear regression and hurdle models adjusted for confounders were applied in the whole sample and stratified by sex at birth. Results: After multiple testing correction, no associations in all children or in boys were found for prenatal or childhood phthalate exposure with the average CCT-score. In girls, prenatal mono-isobutyl phthalate was associated with a higher average CCT-score, indicting more risk-taking (B per 10-fold increase in creatinineadjusted average prenatal levels = 2.10, 95% CI: 0.69,3.52). No associations were observed for bisphenol exposure nor for self-reported risk-taking. Conclusions: Prenatal phthalate exposure was associated with more risk-taking in an experimental task at 10 years-of-age in girls only. The task reflects risky decision-making, which may be a hormonally sensitive construct. Risky decision making potentially precedes real-life risk-taking, which was captured by the self-reported measure and was limited in this young sample. https://doi.org/10.1016/j.envres.2026.125140

Pesticide Exposure and Gynecological Cancers: A Review of Epidemiological Evidence and Mechanistic Pathways,
Mirasheh, M., Shahabinia, Z., Mazidimoradi, A., Soleimani, T., Allahqoli, L. and Salehiniya, H., *Diseases*, Jul 29 2026, Vol. 14, no. 8, p. 273.

Environmental exposure to pesticides is considered a risk factor for gynecological cancers, contributing to their onset and progression through various mechanisms. This narrative review investigates the role of different pesticides in the development and progression of ovarian, endometrial, and cervical cancers, and provides a comprehensive synthesis of epidemiological findings along with molecular mechanisms, highlighting carcinogenic pathways, conflicting findings, and potential therapeutic implications. Relevant epidemiological and experimental studies published between 2000 and 2025 were identified through searches of major scientific databases. Various pesticides, particularly organochlorines, contribute to gynecological cancers through mechanisms such as estrogen mimicry, DNA damage, oxidative stress, increased pro-inflammatory cytokines, and disruption of cellular signaling pathways. However, most studies found no significant

association between certain pesticides, such as Triazines and Atrazine, and gynecological cancers. Furthermore, some pesticides, like Carbendazim, exhibit a dual role; while carcinogenic, they can also serve therapeutic purposes in cancer treatment when utilized with nanotechnology. Overall, pesticide exposure is a significant factor in the development and progression of women's cancers, although the strength of the evidence varies across pesticide classes. Despite numerous studies, contradictory findings necessitate further research to clarify causal relationships.
https://doi.org/10.3390/diseases14080273

Evaluating the Effects of Pesticides Residues on Seminal Fluid Antioxidants Status and Semen Parameters among Males with Infertility at a Tertiary Hospital in Nigeria,
Oladosu, W. O., Jimoh, S. O., Ismaila, O., Alayo, A. M., Gbagirimojo, G. A., Adewale, O. R. and Okesina, A. B., *Indian Journal of Occupational and Environmental Medicine*, Apr-Jun 2026, Vol. 30, no. 2, p. 123-128.

Aims:To evaluate the impacts of seminal concentrations of selected pesticide residues on SFA parameters and seminal antioxidants in men with abnormal sperm parameters.Settings and Design:An analytical cross-sectional study of consenting infertile male patients, with at least one abnormal seminal fluid analysis (SFA) parameter, at a tertiary Health Centre.Methods and Materials:Between July 2022 and June 2023, 130 men with abnormal sperm parameters and 130 with normal sperm parameters were studied as controls. Semen samples were collected, treated, and centrifuged. Analyses included pesticide residues (GC/MS), Vitamins C and E (spectrophotometry), and enzyme levels (ELISA), with data analyzed using SPSS 20.0.Result:Subjects showed significantly higher seminal pesticide levels and lower antioxidant levels than controls. Sperm count and motility were positively linked to antioxidant status and negatively linked to pesticide levels, except for Carbamate. Seminal levels of Malathion (13.57 +/- 2.68 vs 3.54 +/- 1.81 mu g/ml, P < 0.001), DDT (24.03 +/- 1.93 vs 13.65 +/- 1.75 mu g/ml, P < 0.001), Permethrin (28.59 +/- 3.87 vs 3.82 +/- 1.78 mu g/ml, P < 0.001), and Carbamate (35.75 +/- 1.52 vs 17.75 +/- 3.90 mu g/ml, P < 0.001) were significantly higher among subjects than controls, respectively. Also, seminal levels Vitamin C (21.74 +/- 2.30 vs 54.55 +/- 2.78 mg/100 ml, P < 0.001), Vitamin E (5.62 +/- 2.17 mg/100 ml vs 14.73 +/- 1.62 mg/100 ml, P < 0.001), GPx (74.28 +/- 2.63 vs 136.14 +/- 2.79 IU/L, P < 0.001), and CAT (102.28 +/- 1.44 vs 185.57 +/- 1.24 IU/L, P < 0.001) were significantly lower among subjects than controls.Conclusion:Human exposure to various types of pesticides, including herbicides and insecticides used in various daily activities at homes, farmlands, and agricultural storage sites, negatively affects semen parameters and semen antioxidant status.
https://doi.org/10.4103/ijoem.ijoem_119_24

Associations of urinary phthalate metabolite and replacement chemical concentrations with gestational blood pressure,
Parra, K. L., Preston, E. V., Quinn, M. R., Williams, P. L., Hacker, M. R., Mcelrath, T. F., Cantonwine, D. E., Calafat, A. M., Cook Botelho, J., Wylie, B. J., Powe, C. E., Seely, E. W. and James-Todd, T., *Journal of the Endocrine Society*, 2026, Vol. 10, no. 9.

Some phthalates and their replacements are endocrine disrupting chemicals (EDCs) found in consumer products and linked to hypertensive disorders of pregnancy (HDP). Few studies have evaluated associations between EDCs and gestational blood pressure.To evaluate associations between EDC biomarkers with gestational blood pressure.The Environmental Reproductive and Glucose Outcomes study (N = 338) measured urinary concentrations of 18 phthalate and replacement metabolites and systolic (SBP) and diastolic (DBP) blood pressure at 3 pregnancy study visits (median: 12, 19, and 26 weeks' gestation). Individual metabolite concentrations and summary measures based on common source or mechanism (eg, ΣPersonal care products [ΣPCP]) were log2-

transformed, specific-gravity corrected, and modeled continuously and in quartiles. We estimated covariate-adjusted associations of individual metabolites and, summary measures with blood pressure using linear regression, and with HDP using log-binomial regression, assessing metabolite mixtures using Bayesian Kernel Machine Regression.HDP prevalence was 12.7%. Higher concentrations of monoethyl phthalate (MEP) and ΣPCP were most consistently associated with SBP and DBP elevations at visit 3. Higher concentrations of MEP (Q4 vs Q1) were associated with 4.59 mmHg higher SBP (95% CI: 0.04, 9.15), and higher ΣPCP (Q3 vs Q1) were associated with 5.78 mmHg (95% CI: 1.59, 9.97). Higher MEP and ΣPCP (Q4 vs Q1) were positively linked to DBP elevations of 4.51 to 4.70 mmHg. Other phthalate biomarkers were associated with blood pressure at specific time points.Certain urinary phthalate biomarkers, particularly MEP and ΣPCP, were associated with higher blood pressure levels in pregnancy, with implications for HDP. https://doi.org/10.1210/jendso/bvag158

Aromatic amine antioxidants (AAs) and p-phenylenediamine quinones (PPD-Qs) exposure, thyroid hormones, and cognitive function in older adults: Association and potential mediation analyses,
Peng, Y., Gao, Y., Zhu, Z., Cheng, L., Chen, C., Zhao, H. and Li, H., *Environment International*, 2026/09/01/ 2026, Vol. 215, p. 110494.

Aromatic amine antioxidants (AAs) and p-phenylenediamine quinones (PPD-Qs) are novel environmental contaminants derived from tire wear, with emerging neurotoxic concerns, but epidemiological evidence linking them to cognitive health is lacking. This study investigated associations between exposure to these chemicalsand cognitive performance in older adults, and explored the potential mediating role of thyroid hormones. We measured urinary concentrations of nineteen AAs and six PPD-Qs in 439 older adults from Shenzhen, China, and assessed cognitive function using the Mini-Mental State Examination (MMSE). Higher urinary levels of specific compounds, such as N-(1,3-dimethylbutyl)-N'-phenyl-p-phenylenediamine-quinone (6PPD-Q), 4-(cyclohexylamino) diphenylamine-quinone (CPPD-Q), and 4-phenylaminodiphenylamine-quinone (DPPD-Q), were significantly associated with lower MMSE scores and increased risks of cognitive impairment. Mixture analyses further indicated that combined exposure to these chemicals was linked to poorer cognitive performance, with 6PPD-Q and CPPD-Q as the most significant contributors. Additionally, specific thyroid hormones were associated with both chemical exposures and cognitive outcomes. Exploratory mediation analysis suggested that thyrotropin (TSH) and total triiodothyronine (TT3) may mediate the association between DPPD-Q exposure and poorer performance in specific cognitive domains. These findings provide the first epidemiological evidence that AAs and PPD-Qs exposure may be associated with cognitive decline in the elderly, potentially via disruption of thyroid hormone homeostasis. These observations warrant confirmation in longitudinal studies. https://doi.org/10.1016/j.envint.2026.110494

Joint associations between gestational environmental chemical mixtures and child behavioral outcomes,
Puvvula, J., Hwang, W. T., Mccandless, L., Xie, C. C., Braun, J. M., Vuong, A. M., Schisterman, E. F., Shinohara, R. T., Oulhote, Y., Booij, L., Seguin, J. R., Bouchard, M. F., Till, C., Zidek, A., Borghese, M. M., Fisher, M., Fraser, W. D., Muckle, G., Yolton, K., Cecil, K. M., Ashley-Martin, J., Arbuckle, T. E., Lanphear, B. and Chen, A. M., *Environment International*, Aug 2026, Vol. 214.

Background: Gestational exposure to environmental chemicals contributes to adverse neurodevelopmental outcomes in children. We aim to evaluate the joint associations between gestational chemical biomarkers and behavioral functioning in preschool-aged children. Methods: We pooled data from 695 mother-child dyads (female, n = 353; male, n = 342) enrolled in the Health

Outcomes and Measures of the Environment (HOME) & Maternal-Infant Research on Environmental Chemicals (MIREC) Studies. We assessed concentrations of 29 EDC biomarkers at gestation and assessed the child's behavior and executive functioning at 3 years using the Behavioral Assessment System for Children (BASC-2) and Behavior Rating Inventory of Executive Function-Preschool Version (BRIEF-P). Higher scores on these instruments indicate adverse behavioral outcomes. We applied quantile g-computation to estimate joint associations, adjusting for covariates. Findings: Every quartile increase in chemical biomarker mixture was associated with a 0.84-point higher behavior symptom index score (95% CI:-0.89, 2.57), 1.38-point higher externalizing behavior score (95% CI:-0.65, 3.41), and 1.34-point higher internalizing behavior score (95%CI:-0.97, 3.65); these associations were imprecise with confidence intervals including null. Although the interaction by child sex remained null (p = 0.69) for internalizing behavior scores, gestational chemical mixture was associated with higher internalizing behavior scores in males (Psi: 3.59; 95%CI: 0.42, 6.75), but not in females. We found evidence of sex-specific trends for working memory scores (interaction p = 0.02), with an imprecise positive trend in males (Psi: 3.04; 95%CI:-0.46, 6.55) and a negative trend in females. Interpretation: Gestational exposure to a mixture of environmental chemicals showed suggestive but imprecise associations with internalizing problems and poorer working memory among preschool-age males, but not females. Associations were more pronounced in the HOME Study, which includes participants with higher concentrations for many chemical biomarkers, than in MIREC, raising the possibility that pooled estimates reflect cohort-specific exposure contexts. Sex-specific patterns, particularly for working memory, need further investigation, and our findings should be interpreted cautiously given the imprecision of subgroup estimates.
https://doi.org/10.1016/j.envint.2026.110383

Per- and polyfluoroalkyl substances and the risk of thyroid and breast cancer: Evidence from a meta-analysis and computational toxicology,
Shen, H.-Y., Jin, M.-H., Wu, Z.-C., He, T.-T. and Gao, C.-J., *Toxicology and Applied Pharmacology*, 2026/09/01/ 2026, Vol. 514, p. 117933.

Per- and polyfluoroalkyl substances (PFAS) are persistent environmental pollutants with endocrine-disrupting and carcinogenic effects, but epidemiological evidence linking PFAS exposure to cancer remains inconsistent. We conducted a meta-analysis to systematically evaluate associations between PFAS exposure and risks of thyroid and breast cancer. Relevant literature published between January 2003 and May 2025 was retrieved from Web of Science and PubMed. The analysis showed that the pooled odds ratios (ORs) for perfluorooctanoic acid (PFOA) and perfluorohexane sulfonate (PFHxS) in relation to thyroid cancer were 0.87 (95% CI: 0.77–0.98) and 0.84 (95% CI: 0.75–0.94), respectively. In contrast, the pooled OR for overall PFAS exposure and breast cancer was 1.04 (95% CI: 1.00–1.09, I2 = 62.2%), indicating no statistically significant association but suggesting a borderline trend. Notably, PFAS exposure was inversely associated with invasive breast cancer (OR = 0.45, 95% CI: 0.26–0.76). Computational toxicology analyses indicated that PFAS may interact with endocrine- and growth factor-related pathways involving EGFR, AKT1, and ESR1, providing supportive but hypothesis-generating mechanistic context. This study highlights specific PFAS compounds that may be associated with altered risks of thyroid and breast cancer. Future high-quality prospective studies with standardized exposure assessments are warranted to clarify the potential carcinogenic effects of PFAS. https://doi.org/10.1016/j.taap.2026.117933

Phenolic environmental estrogens exposure, urinary metabolites, and gestational diabetes mellitus: A meet-in-metabolite analysis in pregnant women,
Shen, J., Meng, H., Mao, K., Zhang, Y., Hua, W., Huo, Q., Li, W., Ma, H., Li, C., Liang, Y., Huang, Q. and Zhang, Q., *Journal of Environmental Sciences*, 2026/08/27/ 2026, p. 105725.

Phenolic environmental estrogens (PEEs) are a common class of endocrine-disrupting chemicals widely found in daily products. They competitively bind to estrogen receptors, thereby disrupting endocrine function. PEEs appear to play a role in the development of gestational diabetes mellitus (GDM). Earlier findings by our group indicated that 2-tert-octylphenol (2-t-OP) showed a positive correlation with GDM whereas nonylphenol (NP) showed an inverse association. However, the specific molecular mechanisms underlying this relationship remained largely unexplored. To address this gap, we performed comprehensive urinary PEEs quantification and metabolomic profiling in a cohort of 387 pregnant mothers, employing a meet-in-metabolite analysis to identify potential metabolic biomarkers between PEEs exposure and GDM. Our analysis uncovered 11 key metabolites that showed significant associations with both PEEs and GDM. Among these, the abundances of 8 metabolites (kynurenic acid, 11-Ketoetiocholanolone, N-Acetylneuraminic acid, α-Linolenoyl ethanolamide, uric acid, butylparaben, uracil, and N6,N6,N6-Trimethyl-L-lysine) were higher, while 3 (3'-Sialyllactose, thiosulfate, and phosphoroselenoic acid) were lower in the women with higher level of PEEs exposure. Notably, we found that 3'-Sialyllactose, uric acid, 11-Ketoetiocholanolone and phosphoroselenoic acid may impact the positive association of 2-t-OP with GDM, while 3'-Sialyllactose, 11-Ketoetiocholanolone and phosphoroselenoic acid appear to mediate the negative association for NP. Considering the relevant biological pathways, we propose that 2-t-OP and NP may inversely influence GDM risk by interfering with purine, steroid hormone and amino acid metabolism in pregnancy. By shining a light on these metabolic disruptions, our findings open up new avenues for understanding how PEEs exposure contribute to GDM.
https://doi.org/10.1016/j.jes.2026.105725

Sex- and Trimester-SpecificAssociations of PrenatalCo-Exposure to Organophosphate Esters and Phthalates with Preschoolers'Trajectories of Co-Occurring ADHD and ASD Symptoms: Cord Blood MetabolomicStudy in the Ma'anshan Birth Cohort,
Wang, X., Tong, J., Lu, M. J., Luo, L., Liu, Y., Huang, Q. Z., Lv, P., Zheng, Y. M., Gan, H., Geng, M. L., Tao, S. M., Tao, X. Y., Yan, S. Q., Gao, G. P., Wu, X. Y., Huang, K., Cao, Y. X., Gao, H. and Tao, F. B., *Environmental Science & Technology*, Aug 11 2026, Vol. 60, no. 31, p. 21465-21479.

Although neurotoxic, prenatal exposure to organophosphate esters (OPEs) and phthalic acid esters (PAEs) and their effects on preschoolers' autism spectrum disorder (ASD) and attention-deficit hyperactivity disorder (ADHD) cotrajectories and underlying metabolic mechanisms remain unclear, we aimed to elucidate these links. Maternal urinary OPEs/PAEs were measured in 3040 dyads from the Ma'anshan Birth Cohort across three trimesters. Child ADHD/ASD symptom scale scores were assessed at ages 3, 5, and 6, and cotrajectories were identified using group-based multitrajectory modeling. Single-pollutant models revealed that bis(2-ethylhexyl) phosphate (BEHP) across pregnancy was positively associated with high-score trajectories (HST) (OR = 1.20, 95% CI: 1.06, 1.37), whereas bis(2-butoxyethyl) phosphate (BBOEP) exhibited U-shaped associations. Second-trimester diphenyl phosphate (DPHP) (OR = 1.13, 95% CI: 1.01, 1.26), BEHP (OR = 1.14, 95% CI: 1.04, 1.24), and monobutyl phthalate (OR = 1.15, 95% CI: 1.00, 1.32) were positively associated with HST. First-trimester DPHP exhibited a positive correlation with moderate-score trajectories and HST in girls, while bis(1-chloro-2-propyl) phosphate across pregnancy was inversely associated with HST in boys (p(sex-int) < 0.05). No mixed effects were detected. BBOEP across pregnancy was negatively associated with ADHD symptoms, whereas BEHP was positively associated. BEHP, monomethyl phthalate, and mono-(2-ethyl-5-oxohexyl) phthalate were positively associated with ASD symptoms, whereas dibutyl phosphate and monoethyl phthalate were negatively associated (p < 0.05). Cord blood metabolomics identified pyrimidine, biotin, lysine, cysteine, and methionine metabolism as key mediators of OPE-induced cotrajectories, and purine metabolism mediated PAEs' effects (p < 0.05). This study highlights OPE/PAE neurotoxicity and reveals novel cord metabolomic insights.
https://doi.org/10.1021/acs.est.6c01402

Environmental Endocrine-Disrupting Chemicals and Metabolic Dysfunction-Associated Steatotic Liver Disease,
Zhao, Y. W., He, H. F., Zeng, J. and Zhang, J. J., *Liver International*, Jul 9 2026, Vol. 46, no. 8.

Background and Aims Metabolic dysfunction-associated steatotic liver disease (MASLD), formerly termed non-alcoholic fatty liver disease (NAFLD), represents the most prevalent chronic liver condition worldwide, affecting approximately 25%-30% of the global adult population. Endocrine-disrupting chemicals (EDCs)-including phthalates, bisphenols, per- and polyfluoroalkyl substances (PFAS), persistent organic pollutants (POPs) and heavy metals-are ubiquitous environmental contaminants with established metabolic toxicity. However, the quantitative association between EDC exposure and MASLD prevalence remains incompletely characterised. We conducted a systematic review and meta-analysis to synthesise the current epidemiological evidence on this association. Methods We systematically searched PubMed, Embase, Web of Science and the Cochrane Library from inception through January 2026, following the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 guidelines (PROSPERO: CRD420261298624). Studies were eligible if they reported quantitative associations between measured EDC exposures and MASLD/NAFLD/metabolic-associated fatty liver disease (MAFLD) outcomes in human populations. Two investigators independently screened records, extracted data and assessed methodological quality using a customised Newcastle-Ottawa Scale (NOS). Pooled odds ratios (ORs) with 95% confidence intervals (CIs) were calculated using random-effects models. Heterogeneity was assessed via the I2 statistic. Funnel plot asymmetry and Egger's test were used to evaluate publication bias. Sensitivity analyses were performed using leave-one-out methods. Results Of 427 records identified, 42 studies encompassing diverse global populations met inclusion criteria and were incorporated into the quantitative synthesis. The meta-analysis demonstrated that: Phthalates: mono (2-ethyl-5-carboxypentyl) phthalate (MECPP; weighted mean difference [WMD]: 2.40, 95% CI: 0.71-4.09), mono (2-ethyl-5-hydroxyhexyl) phthalate (MEHHP; WMD: 2.46, 95% CI: 0.81-4.10) and monobenzyl phthalate (MBzP; WMD: 1.84, 95% CI: 0.10-3.59) showed significant positive associations with MASLD prevalence; pooled analysis of phthalate mixtures yielded an overall OR of 1.17 (95% CI: 1.13-1.21; I2 = 41.4%). Bisphenols: pooled analysis demonstrated a significant positive association with MASLD (OR: 1.29, 95% CI: 1.09-1.50; I2 = 73.0%), with bisphenol A (BPA; OR: 1.32, 95% CI: 0.92-1.72), bisphenol S (BPS; OR: 1.47, 95% CI: 0.96-1.98) and bisphenol F (BPF; OR: 1.19, 95% CI: 0.77-1.61) contributing to this effect. PFAS: the overall pooled effect was OR 0.95 (95% CI: 0.91-1.00; I2 = 22.9%). POPs: the overall OR was 1.30 (95% CI: 1.22-1.37; I2 = 29.2%), with organochlorine pesticides (OCPs; OR: 1.30, 95% CI: 1.19-1.42) and polychlorinated biphenyl (PCB)/OCP combinations demonstrating significant associations. Heavy metals/metalloids: mercury (Hg) demonstrated the strongest positive association (OR: 1.60, 95% CI: 1.15-2.04; I2 = 87.3%), followed by lead (Pb; OR: 1.40, 95% CI: 1.02-1.78) and cadmium (Cd; OR: 1.30, 95% CI: 1.09-1.51; I2 = 79.2%); the overall metals pooled OR was 1.26 (95% CI: 1.16-1.37; I2 = 79.9%). Methodological quality assessed by NOS ranged from 6 to 9 points, with the majority of studies scoring >= 7. Conclusions This meta-analysis of observational studies suggests that environmental exposure to several major EDC classes-including phthalates, bisphenols, persistent organic pollutants and certain heavy metals-is associated with a higher prevalence of MASLD, although the magnitude and consistency of associations vary substantially across chemical classes and individual compounds. Findings for PFAS were heterogeneous and compound-specific. Given the predominantly cross-sectional nature of the available evidence and the considerable between-study heterogeneity, causal inference cannot be drawn. These findings should be interpreted as hypothesis-generating and underscore the need for prospective multi-pollutant studies and mechanistic investigations to clarify the role of EDCs in MASLD pathogenesis. https://doi.org/10.1111/liv.70793

Environmental exposome study for prostate cancer in elderly men from Western China: preliminary findings from a case–control study in the WeEndPd cohort,
Zhou, X., He, Y., Jin, Y., Peng, Z., Zeng, B., Zhang, Z., Wu, Y., Wei, C., Mo, Z., Cheng, J., Chen, Y., Wang, F., Lu, G., Tao, K., Yang, Z., Wu, Y., Zhang, W., Zhuang, C., Guo, W., Li, G., Liu, Y., Huang, Y., Gao, R., Zhan, Y., Zou, X., Yang, L., Wei, Q., Wei, X. and Qiu, S., *BMJ Open*, 2026, Vol. 16, no. 7, p. e110957.

Objectives To investigate the association between exposure to specific endocrine-disrupting chemicals (EDCs), air pollutants and the risk of localised prostate cancer (PCa) and to evaluate the combined effect of mixed EDC exposures.Design A case–control study.Setting Secondary care; a single tertiary hospital in Western China.Participants A total of 580 patients with histologically confirmed localised PCa who underwent radical prostatectomy between May 2021 and June 2023 were included as cases. They were matched 1:2 with 1160 cancer-free controls from the same cohort on age (±5 years) and date of biological sample collection. Key exclusion criteria were a history of other malignancies, androgen deprivation therapy prior to surgery or acute urinary infection. Primary and secondary outcome measures The primary outcome was the odds of PCa associated with individual and mixed exposures. Primary exposures were urinary concentrations of nine bisphenols (BPs) and nine phthalate metabolites (quantified via high-performance liquid chromatography–tandem mass spectrometry) and estimated ambient air pollution exposure (sulphur dioxide (SO2) and $PM_{2.5}$) based on geocoded residential addresses.Results A total of 580 cases and 1160 controls were included in the final analysis. Exposure to the highest quartile of sulphur dioxide (SO2) over 5 years (9.37–28.79 µg/m3) was significantly associated with PCa, yielding an OR of 1.65 (95% CI 1.08 to 2.72; p<0.001) compared with the lowest quartile (3.61–6.89 µg/m3). Urinary concentrations of multiple phthalate metabolites (MECPP, MEHHP and MEHP) and BPs (bisphenol A (BPA) and BPZ) were significantly higher in cases (all p<0.05). Weighted quantile sum (WQS) regression analysis demonstrated a significant positive association between the mixture of EDCs and PCa risk (WQS OR=1.26, 95% CI 1.20 to 1.33; p<0.001).Conclusions Exposure to SO2 and specific EDCs (both individually and as a mixture) is significantly associated with localised PCa.Trial registration number ChiCTR1900024623.Data are available upon reasonable request. Data are available upon reasonable request from the corresponding author.
https://doi.org/10.1136/bmjopen-2025-110957

Toxicité sur l'homme

PFAS. An everlasting challenge,
BfR2GO - The Science Magazine of the German Federal Institute for Risk Assessment, juin 2026.
PFAS, which are widely used as industrial chemicals, are proving to be long-lasting "forever chemicals" in the environment. The BfR is investigating the extent to which certain PFAS can affect health. https://www.bfr2go.de/en/issue-1-2026/pfas/

The impact of endocrine disrupting chemicals diethylstilbestrol and ketoconazole on lipid storage in in vitro matured bovine cumulus-oocyte complexes†,
Asimaki, K., Van De Chris, H. a. L., Wubbolts, R., Jansen, J. W. A., Helms, J. B., Aardema, H., Van Majorie, B. M. D. and Gadella, B. M., *Biology of Reproduction*, 2026.

Endocrine disrupting chemicals (EDCs) impact female fertility, and their widespread presence demands deeper understanding of their effects on reproduction. In vitro maturation of bovine cumulus-oocyte complexes (COCs) serves as a relevant reproductive toxicology model aligned with the 3R principle. Given the link between lipid metabolism and COC quality, the possibility that lipid

droplets (LDs) in cumulus cells (CCs) and overall COC lipid content are susceptible to EDC disruption was tested. COCs were exposed to diethylstilbestrol (DES; estrogen receptor agonist) and ketoconazole (KTZ; steroidogenesis inhibitor), during in vitro maturation. Neutral lipid dyes were used for LD visualization (count, size, area, and clustering) in CCs. Lipidomic analysis using liquid chromatography-mass spectrometry was conducted separately on oocytes and CCs. Of all tested DES concentrations (i.e., 10-9 M, 10-7 M, 10-5 M), 10-5 M DES reduced the total LD area per CC, associated with decreased LD count and increased size. KTZ at all tested concentrations (i.e., 10-8 M, 10-7 M, 10-6 M) reduced LD size and enhanced clustering. Lipidomic analysis revealed the selective depletion of cholesteryl esters in CCs exposed to 10-5 M DES or 10-6 M KTZ, with minimal changes in other neutral lipid classes. Thus EDCs can disrupt lipid storage and LD dynamics in bovine COCs. We discuss these new findings with our previous work: (i) DES-induced steroidogenesis while (ii) in contrast KTZ blocked steroidogenesis in CCs. Together, the data highlight the COC lipid metabolism as a novel EDC target, warranting further study due to potential implications for female fertility.Diethylstilbestrol and ketoconazole disrupt lipid storage and lipid droplet dynamics in bovine COCs in vitro, highlighting the need to clarify EDC impacts on COC lipid metabolism and potential fertility consequences. https://doi.org/10.1093/biolre/ioag119

A Network Toxicology Framework for Identification of Immune System Disruption by Per- and Polyfluoroalkyl Substance (PFAS) Mixture: In Silico Analysis,
Baralic, K., Vidic, K., Maric, D., Zivanovic, J., Djordjevic, A. B., Curcic, M., Bulat, Z., Antonijevic, B. and Dukic-Cosic, D., *Journal of Xenobiotics*, Jun 19 2026, Vol. 16, no. 3.

Per- and polyfluoroalkyl substances (PFAS) are persistent, chemically stable compounds widely used in daily life. Perfluorooctanoic acid (PFOA), perfluorononanoic acid (PFNA), perfluorohexanesulfonic acid (PFHxS), and perfluorooctanesulfonic acid (PFOS) were identified as the most relevant PFAS due to their prevalence and toxicity. This study aimed to investigate the immunotoxic mechanisms of a mixture of these PFAS using an in silico approach. Comparative Toxicogenomic Database (CTD), GeneMANIA, CytoHubba (Cytoscape), ToppGene Suite, and Metascape were used for the analysis. A total of 65 immune-related genes were identified as common to all four PFAS, with IFNG, TNF, IL1B, IL6, TYK2, CD3E, CASP8, VAV1, ARHGAP4, and CARD11 emerging as key hub genes. CTD phenotype analysis indicated immune dysregulation, with decreased humoral and adaptive immune responses in humans and tissue-specific modulation of B- and T-cell activity in mice, while no immune-related phenotypes were observed for PFNA. Network analysis identified functional modules associated with apoptotic and immune signaling, endothelial cell migration and angiogenesis, and shared inflammatory and viral response pathways. Disease enrichment analysis associated PFAS with autoimmune disorders (rheumatoid arthritis, asthma), metabolic conditions, and cardiovascular diseases (experimental diabetes, hypertensive disease). These results highlight PFAS involvement in immune modulation, cytokine signaling, and disease susceptibility.
https://doi.org/10.3390/jox16030115

Goutte et facteurs environnementaux,
Bardin, T., Letavernier, E., Resche-Rigon, M. and Richette, P., *Bulletin de l'Académie Nationale de Médecine*, 2026/08/07/ 2026.

Résumé Le rôle de l'environnement dans la pathogénie de goutte est l'objet d'un intérêt croissant, motivé par l'absence d'explication complète de la maladie par les facteurs génétiques et alimentaires, qui amène à chercher d'autres facteurs étiologiques. Les pics de pollution atmosphériques se caractérisent par une concentration accrue en microparticules toxiques, capables d'activer l'inflammasome NLRP3 et ont été identifiés par plusieurs études comme facteurs déclenchants des crises de goutte. De nombreux polluants sont associés à l'hyperuricémie, comme le

montrent un certain nombre d'intoxications accidentelles ou professionnelles et surtout de multiples études en population générale. Il en est ainsi des polluants organiques persistants, qui incluent les dioxines et produits apparentés (qui ont en commun de se lier au récepteur cellulaire AHR), les PFASS, les pesticides organochlorés, et les retardateurs de flamme bromés. Nos travaux expérimentaux montrent que le récepteur AHR augmente la réabsorption tubulaire rénale d'urate, pouvant expliquer l'hyperuricémie induite par les dioxines. D'autres polluants sont associés à l'hyperuricémie : hydrocarbones polycycliques aromatiques (qui se lient à AHR), certains phtalates et bisphénols. Le plomb et le cadmium sont aussi associés à l'hyperuricémie. Beaucoup de ces toxiques sont aussi des produits carcinogènes, des perturbateurs endocriniens ou sont associés au syndrome métabolique, très fréquent chez les goutteux. Les perturbations du climat, qui augmentent la diffusion de ces polluants, et la persistance, malgré les mesures prises, de la pollution sont donc un enjeu majeur pour la santé en général, mais aussi pour la goutte, dont ils pourraient expliquer l'augmentation de fréquence au cours des dernières décennies. The role of the environment in the pathogenesis of gout is the subject of growing interest, motivated by the lack of a complete explanation for the disease based on genetic and dietary factors, which has led to a search for other etiological factors. Peaks in air pollution are characterized by increased concentrations of toxic microparticles capable of activating the NLRP3 inflammasome and have been identified by several studies as triggers for gout attacks. Many pollutants are associated with hyperuricemia, as shown by a number of accidental or occupational intoxications and multiple studies in the general population. This is the case for persistent organic pollutants, which include dioxins and related products (which share the common feature of binding to the AHR cell receptor), PFASs, organochlorine pesticides, and brominated flame retardants. Our experimental work showed that the AHR receptor increased renal tubular reabsorption of urate, which may explain the hyperuricemia induced by dioxins. Other pollutants are associated with hyperuricemia: polycyclic aromatic hydrocarbons (which bind to AHR), phthalates, and bisphenols. Lead and cadmium are also associated with hyperuricemia. Many of these toxins are also carcinogens, endocrine disruptors, or are associated with metabolic syndrome, which is very common in gout patients. Climate change, which increases the spread of these pollutants, and the persistence of pollution despite the measures taken, are therefore a major challenge for health in general, but also for gout, and could explain the increased frequency of gout observed in recent decades.
https://doi.org/10.1016/j.banm.2026.05.034

Endocrine Disruptors and Gynecological Malignancies,
Baroutis, D., Katsianou, E., Koukoumpanis, K., Fragiskos, I., Sindos, N., Sindos, M. and Daskalakis, G., *Diagnostics*, Jul 2026, Vol. 16, no. 13.

Background/Objectives: Endocrine-disrupting chemicals (EDCs) interfere with hormonal homeostasis and have been implicated in gynecological malignancy pathogenesis. This narrative review synthesizes current evidence regarding EDC exposure and breast, endometrial, ovarian, and cervical cancers, examining molecular mechanisms, epidemiology, and diagnostic and clinical implications. Methods: We conducted a literature review using PubMed/MEDLINE, Embase, Scopus, and Cochrane databases through April 2026, including systematic reviews, meta-analyses, prospective cohorts, case-control studies, and mechanistic investigations examining EDC-cancer associations. Methodological quality was appraised using the Newcastle-Ottawa Scale and AMSTAR-2, with overall certainty of evidence rated using the GRADE framework. Results: Major EDC classes-bisphenol compounds, phthalates, polychlorinated biphenyls, organochlorine pesticides, and per- and polyfluoroalkyl substances-demonstrate carcinogenic potential through estrogen receptor modulation, epigenetic alterations, oxidative stress, and oncogenic signaling disruption. Breast cancer shows the strongest evidence, with prenatal and early-life DDT/DDE exposure associated with up to a 3.7-fold increased risk. Endometrial cancer demonstrates associations with

xenoestrogen mixtures exhibiting non-monotonic dose-responses, whereas ovarian and cervical cancers show emerging but limited associations. Common mechanisms include receptor crosstalk, epigenetic dysregulation with transgenerational effects, oxidative genomic instability, metabolic reprogramming, and cancer stem cell enrichment. Conclusions: Evidence supports EDC contributions to gynecological malignancy through convergent pathways, though causal inference remains constrained by observational epidemiology, long latency periods, and challenges in characterizing real-world mixture exposures. Diagnostic and prevention strategies should integrate EDC exposure into risk-prediction models, leverage multi-omics biomarkers for early detection, and emphasize exposure reduction during critical developmental windows alongside regulatory reform. https://doi.org/10.3390/diagnostics16132116

Sex-specific cellular and molecular mechanisms of PFAS-induced reproductive toxicity,
Bhagwan, A. S., Gowtham, A., Kumar, D. and Kaundal, R. K., *Toxicology*, Nov 2026, Vol. 526.

Per- and polyfluoroalkyl substances (PFAS) are persistent environmental contaminants widely detected in water, food, consumer products, and household dust, resulting in ongoing human exposure and bioaccumulation. Accumulating evidence increasingly implicates PFAS in reproductive dysfunction. Although PFAS-associated reproductive toxicity has been widely reviewed, critical knowledge gaps persist regarding the sex-specific cellular and molecular mechanisms mediating these effects and the reproductive toxicity of emerging replacement PFAS. Increasing epidemiological, in vivo, and in vitro evidence indicates that PFAS disrupt reproductive health through multiple mechanisms common to both sexes, including oxidative stress, mitochondrial dysfunction, endocrine perturbation, inflammation, epigenetic modifications, and dysregulation of cell survival pathways. However, emerging data reveal sex-specific differences in susceptibility and molecular responses. In males, PFAS specifically disrupt steroidogenesis, the blood-testis barrier (BTB), induce germ cell apoptosis, and impair spermatogenesis, leading to reduced sperm quality and fertility. In females, PFAS exposure perturbs ovarian homeostasis by impairing folliculogenesis, oocyte maturation, steroid hormone biosynthesis, and granulosa/cumulus cell function, thereby contributing to diminished ovarian reserve and reproductive dysfunction. Distinct alterations in hormone signaling, lipid metabolism, nuclear receptor activation, and epigenetic regulation mediate these sex-specific effects. Beyond gonadal toxicity, PFAS exposure during pregnancy disrupts placental development and endocrine signaling, contributing to placental dysfunction, adverse pregnancy outcomes, and developmental programming associated with metabolic, neurodevelopmental, and immune abnormalities in offspring. This review presents current epidemiological and mechanistic evidence to delineate shared and sex-specific pathways underlying reproductive toxicity induced by both legacy and emerging PFAS. The findings provide an updated perspective on PFAS-induced reproductive toxicity and have implications for future risk assessment and targeted interventions. https://doi.org/10.1016/j.tox.2026.154535

IntegratedProteomic and Phosphoproteomic ProfilingReveals Mechanisms of Bisphenol A Induced Placental Toxicity,
Biswas, A., Saha, S., Kharbanda, N. and Maiti, T. K., *Chemical Research in Toxicology*, 2026.

The global industrialization and rapid urbanization have elevated the risk of toxic pollutant exposure, which affects human health, especially during pregnancy. Pregnant mothers are daily exposed to bisphenol A (BPA), a common plastic leachate and a prominent toxic pollutant present in our environment. BPA acts as an endocrine-disrupting chemical (EDC) by altering feto-placental homeostasis. This persistent and potent exposure to BPA during gestation can trigger placental damage, affecting trophoblast cell function and survival. BPA even disrupts specific signaling cascades by altering post-translational protein phosphorylation. However, this BPA-mediated

dysregulation of signaling nodes in the early trimester placenta remains unexplored. Therefore, this study investigates the global proteome changes in post-BPA-exposed extravillous trophoblast (EVT) cells, which revealed a BPA-mediated dynamic regulation of phospho-proteome signatures and their associated kinases. Further inspection shows that the altered phosphorylation of c-JUN (S63) and GSK3 alpha (Y279) is associated with BPA toxicity in EVTs and the placenta. This altered phosphorylation affects cellular signaling downstream, imparting damage upon the growing feto-placental unit. This highlights an altered phosphorylation-mediated mechanism of BPA toxicity in the placenta, which can cause the onset of adverse pregnancy outcomes. Data are available via ProteomeXchange with the identifiers PXD074780 and PXD080006.
https://doi.org/10.1021/acs.chemrestox.6c00191

Chemical exposures as risk factors for the development of thyroid cancer,
Caretti, R., Arain, H. A. and Chen, H. R., *Oncologist*, Sep 2026, Vol. 31, no. 9.

Background The incidence of thyroid cancer (TC) has risen in recent decades, prompting investigation of environmental contributors beyond traditional risk factors.Summary This narrative review examines the association between TC and chemical exposures including per- and polyfluoroalkyl substance (PFAS), polychlorinated biphenyls (PCBs), polybrominated diphenyl ethers (PBDEs), phthalates, phenols, nitrates, and polycyclic aromatic hydrocarbons (PAHs). Several studies have shown a link between PFAS and PCB exposure and TC, particularly papillary thyroid cancer. Nitrates, even at currently accepted safe exposure levels have also been shown to have an association with TC. Findings for phthalates, PBDEs, phenols, and PAHs are inconsistent and less studied.Conclusion Current evidence supports that an association exists between select chemical exposures and TC development, but study limitations prevent any direct link to causation. Further research is needed to guide clinical practice and public health policy.
https://doi.org/10.1093/oncolo/oyag299

Endocrine disruptors and male reproductive health: mechanisms, epigenetics, and clinical perspectives,
Concepción-Zavaleta, M. J., Villanueva-De La Cruz, J. P., Rodríguez-Soto, J. C., Fuentes-Mendoza, J., Coronado-Arroyo, J., Concepción-Urteaga, L., Quiroz-Aldave, J. and Paz-Ibarra, J., *Revista Internacional De Andrologia*, Jun 2026, Vol. 24, no. 2, p. 1–14.

Male infertility, a pressing public health issue, is increasingly linked to exposure to endocrine-disrupting chemicals (EDCs), such as phthalates, bisphenol A (BPA), organochlorines, pesticides, as well as natural EDCs such as phytoestrogens. This review synthesizes mechanistic and translational evidence on how EDCs impair male reproductive function through multifaceted pathways. EDCs disrupt the hypothalamic-pituitary-gonadal (HPG) axis, induce cytotoxic effects in Leydig and Sertoli cells, and impair steroidogenesis and spermatogenesis, leading to clinically significant reductions in sperm concentration, motility, and morphology. Emerging data highlight transgenerational effects via epigenetic modifications, including altered DNA methylation and microRNA expression, which may perpetuate infertility across generations. We critically evaluate epidemiological and experimental studies, revealing consistent associations between EDC exposure and adverse reproductive outcomes, such as cryptorchidism, testicular cancer, and idiopathic infertility. Notably, prenatal and perinatal exposure windows exhibit heightened vulnerability, with lifelong consequences. Despite progress, key gaps persist in understanding low-dose mixture effects, interindividual susceptibility, and the interplay between EDCs and lifestyle factors (e.g., oxidative stress, psychosocial stressors). Clinically, this review underscores the need for enhanced biomonitoring to quantify real-world EDC exposures and their interactions, mechanistic research elucidating dose-response relationships and non-monotonic effects, and evidence-based policies to

regulate EDCs in consumer products, agriculture, and healthcare. We propose a translational framework integrating high-throughput toxicology, epigenetic biomarkers, and clinical interventions to mitigate risks. Addressing these challenges is urgent to safeguard male reproductive health amid rising global infertility rates. Future research should also explicitly consider the role of natural endocrine-disrupting compounds, particularly phytoestrogens, in shaping male reproductive outcomes. https://doi.org/10.22514/j.androl.2026.015

Prenatal PFAS exposure and offspring health: evidence review and implications for intergenerational risk assessment,
Deng, B., Xia, X. and Sun, X. X., *Frontiers in Public Health*, Jul 16 2026, Vol. 14.

Prenatal exposure to per- and polyfluoroalkyl substances (PFAS) represents a critical developmental concern because several PFAS cross the placenta and may perturb biological programming during sensitive windows. Although epidemiological and experimental studies have linked prenatal exposure to several measured PFAS, primarily legacy perfluoroalkyl acids such as perfluorooctanoic acid (PFOA), perfluorooctanesulfonic acid (PFOS), Perfluorohexane Sulfonic Acid (PFHxS), and Perfluorononanoic Acid (PFNA), to diverse offspring outcomes, the strength of evidence differs substantially across compounds and health domains. Current human evidence is strongest for impaired vaccine antibody responses and altered growth or metabolic trajectories in studies dominated by legacy PFAS, whereas evidence for other congeners, short-chain PFAS, ether-based alternatives, and fluorotelomer compounds remains sparse. Mechanistically, placental transfer, nuclear receptor perturbation, thyroid hormone transport disruption, mitochondrial stress, immune modulation, and epigenetic reprogramming may jointly contribute to developmental susceptibility. We further discuss how emerging PFAS alternatives and real-world mixture exposures challenge single-chemical and adult-centered assessment paradigms. Finally, we outline a conceptual framework for developmental hazard assessment, in which exposure-window characterization, congener-specific toxicokinetics, human-relevant models, multi-omics biomarkers, and PBPK/PBTK modeling are positioned as complementary components rather than as a fully operational assessment system. https://doi.org/10.3389/fpubh.2026.1892134

Chronic Monobutyl Phthalate Exposure Promotes Anaplastic Thyroid Cancer Progression Through Inflammatory Signaling Dysregulation: Integrated Transcriptomic and Network Toxicology Analyses,
Deng, Y., Tan, S., Wei, J., Guo, X., Hu, L., Qu, X. and Zhou, J., *Biomedicines*, 2026, Vol. 14, no. 8, p. 1755.
https://doi.org/10.3390/biomedicines14081755

Chronic low-dose of BPS and PFOS alter adipogenic programming and impair insulin responsiveness in human adipocytes,
Gaggi, G., Di Credico, A., Bibbò, S., Marini, A., Di Baldassarre, A. and Ghinassi, B., *Journal of Endocrinological Investigation*, 2026/08/26 2026.

obesity is a major global health concern tightly linked to insulin resistance and type 2 diabetes. Environmental exposure to endocrine-disrupting chemicals (EDs), including bisphenols (BPs) and perfluoroalkyl substances (PFs), has been implicated in metabolic dysfunction, yet the impact of chronic low-dose co-exposure on human adipocyte development and insulin responsiveness remains poorly defined. Here, we evaluated bisphenol S (BPS) and perfluorooctane sulfonate (PFOS), alone or combined, in human adipose-derived stem cells undergoing adipogenic differentiation.
https://doi.org/10.1007/s40618-026-03030-y

Neuroendocrine Disruption of Reproduction by Bisphenols and Phthalates: From Molecular Mechanisms to Reproductive Outcomes,
Gherman Lencu, C. C., Gerdanovics, C. A., Orășan, O. H., Iancu, D. M., Gerdanovics, A., Perne, M. G., Milaciu, M. V., Negrean, V., Dobrotă, I. R. and Alexescu, T. G., *International Journal of Molecular Sciences*, 2026, Vol. 27, no. 16, p. 7311. https://www.mdpi.com/1422-0067/27/16/7311

Implications of Endocrine-Disrupting Chemicals for Human Health and Effective Methods for Prevention and Reduction,
Gherman-Lencu, C. C., Alexescu, T. G., Muresanu, C., Gerdanovics, C. A., Milaciu, M. V. and Iancu, D. M., *Toxics*, Jun 12 2026, Vol. 14, no. 6.

Endocrine-disrupting chemicals (EDCs) are a heterogeneous group of exogenous compounds capable of interfering with hormonal homeostasis and endocrine-regulated physiological processes. Their widespread occurrence in food, water, air, consumer products and industrial materials has raised increasing concern regarding their contribution to chronic disease burden. This review synthesizes current evidence on the exposure characteristics, molecular mechanisms, health effects, and prevention strategies related to major EDC classes, including bisphenol A and phthalates, dioxins and polychlorinated biphenyls, per- and polyfluoroalkyl substances, pesticides, and brominated flame retardants. Evidence indicates that EDCs may act through receptor-mediated signaling, altered hormone synthesis and metabolism, oxidative stress, mitochondrial dysfunction, immune modulation, and epigenetic mechanisms, with effects that may vary according to dose, timing, sex, age, and developmental susceptibility. Reported health outcomes include metabolic and cardiovascular disorders, reproductive dysfunction, hormone-dependent cancers, thyroid disruption, immune dysregulation, and adverse developmental effects. Although complete avoidance is unrealistic, exposure reduction and risk mitigation can be achieved through coordinated individual, clinical, environmental, and regulatory interventions. A life-course approach is essential to limit the health burden associated with endocrine disruption. https://doi.org/10.3390/toxics14060515

Per- and Polyfluoroalkyl Substances Exposure and Ischemic Heart Disease: Emerging Evidence from the Literature,
Gorini, F., Tonacci, A., Palazzo, M., Bustaffa, E., Minichilli, F. and Borghini, A., *Antioxidants*, Jun 5 2026, Vol. 15, no. 6.

Ischemic heart disease (IHD) is a chronic and progressive condition characterized by reduced blood flow, mainly due to atherosclerosis. It is currently the leading cause of mortality among cardiovascular diseases. In recent years, per- and polyfluoroalkyl substances (PFAS), a group of ubiquitous and highly persistent environmental contaminants, have emerged as potential risk factors for IHD. PFAS are well-established endocrine disruptors and have been associated with hypercholesterolemia, hypertriglyceridemia, and insulin resistance. Despite the limited number of epidemiological studies and inconsistent findings from occupational settings, accumulating evidence suggests that elevated exposure to certain PFAS compounds may increase the risk of IHD and vascular dysfunction, including processes related to atherosclerosis development, sometimes with dose-response relationships and sex-specific patterns. Mechanistic evidence supports this link, indicating that PFAS exposure induces molecular and cellular alterations relevant to cardiovascular pathophysiology, including increased oxidative stress and vascular inflammation, and disruption of lipid metabolism. In addition, PFAS may affect epigenetic regulation, telomere length, and mitochondrial DNA copy number, which are emerging biomarkers associated with atherosclerosis and IHD and may indicate early cardiovascular vulnerability. Future research integrating innovative approaches and advanced analytical techniques may help address current knowledge gaps and

clarify the mechanistic pathways linking PFAS exposure to clinical cardiovascular outcomes. https://doi.org/10.3390/antiox15060718

Bisphenol A and cancer: a critical review of the epidemiologic literature with an emphasis on exposure assessment,
Guha, N., Osborne, G., Marder, M. E., Sandy, M. S., Sun, M., Firchow, N. and Cogliano, V. J., *Environmental Health*, Jul 22 2026, Vol. 25, no. 1.

Background Bisphenol A (BPA) is a high production volume chemical that has been used for decades in numerous consumer and industrial applications. Daily exposure to BPA is likely; it is readily detected in >90% of the general population despite being rapidly metabolized and excreted. BPA's toxicity, including endocrine disrupting activity, has sparked public health concern. We comprehensively reviewed the epidemiologic literature on the carcinogenicity of BPA and highlighted exposure assessment considerations that impact study interpretation. Methods Multiple biomedical databases were searched through February 2025 for peer-reviewed cancer epidemiology studies that assessed associations with BPA exposure. Studies of the following designs (or a variant) were included: cohort, case-control, cross-sectional. A detailed bias assessment was conducted with guidance from the Report on Carcinogens Handbook and the International Agency for Research on Cancer Monographs Preamble. Results Of the 139 records identified, 43 epidemiological studies were reviewed; all were conducted in the general population. We focused the review on cancers of the breast and prostate because they had the highest number of cohort or case-control studies, but all cancer sites were summarized in the appendix for completeness. Generally, there were mixed results for associations with BPA. Interpretation was hampered by the high potential for exposure measurement error and the inability to characterize past BPA exposure, necessary to assess cancer outcomes with long latency. The majority of studies relied on biomonitoring using short-term biomarkers of recent BPA exposure, and measured BPA in urine at a single time point post-diagnosis; this failed to capture the critical time window of susceptibility, rule out reverse causation, or characterize temporal variation in BPA exposure. Conclusions The epidemiologic evidence was inadequate to evaluate the carcinogenicity of BPA - mainly due to exposure measurement error and misclassification, limited number of studies by cancer site, and heterogeneity in the results and study methodology. The inadequate evidence base cannot rule out potential carcinogenicity of BPA in humans. Future studies conducted within highly exposed occupational cohorts, with prospective and longitudinal collection of quantitative exposure data (e.g., BPA levels) and assessment of cancer outcomes or acute endpoints involved in established mechanisms of carcinogenesis (e.g., receptor mediated effects) are likely to be informative. https://doi.org/10.1186/s12940-026-01306-7

Endocrine and cardiovascular consequences of plasticiser exposure: a narrative review from the thyroid- cardiac axis perspective,
Halder, D., P.V, V., Mitra, T., Karthikeyan, D., Anil Kumar, A. H. S., S.N, M. S., Mishra, S. and Janardhanan, R., *Frontiers in Pharmacology*, 2026–August–28 2026, Vol. Volume 17 - 2026.

Endocrine-disrupting chemicals particularly plasticisers such as phthalates, bisphenols and their emerging substitutes, are increasingly recognised as the main environmental toxicants with significant implications for thyroid and cardiovascular health. Due to their widespread use in consumer products, food packaging, medical devices, cosmetics and industrial materials, chronic human exposure occurs via ingestion, inhalation, dermal absorption and parenteral administration. Increasing evidence indicates that these compounds disrupt the hypothalamic-pituitary-thyroid axis by interfering with thyroid hormone synthesis, transport, receptor signalling, iodine homeostasis and endocrine feedback regulation. These disruptions contribute to thyroid autoimmunity, hypothyroidism, and broader endocrine imbalance. Because tfundihyroid hormones are vital for

cardiovascular health, plasticiser-related thyroid problems can indirectly lead to cardiovascular issues, including hypertension, endothelial dysfunction, dyslipidaemia, arrhythmias, atherosclerosis and heart failure. Mechanistically, these effects are mediated by oxidative stress, mitochondrial dysfunction, inflammatory signalling, ion-channel disruption, metabolic dysregulation and epigenetic modifications. Integrated thyroid-cardiac crosstalk is increasingly recognised as a critical mechanistic axis linking environmental EDC exposure to the progression of cardiometabolic disease. The review highlights the emerging biomarkers and advanced microfluidic and organ-on-chip technologies as promising tools for early detection and mechanistic evaluation of plasticiser-induced thyroid-cardiac dysfunction. Collectively, this review also highlights the critical need for long-term human studies, combined exposure assessments and translational diagnostic approaches to enhance our understanding and alleviate the escalating public health burden of environmental plasticiser exposure. https://doi.org/10.3389/fphar.2026.1876287

Living in a chemical cocktail: real-world mixture exposures and their impact on ovarian function, Halloran, K. M., Zhou, Y. R. and Padmanabhan, V., *Journal of Reproduction and Development*, 2026 2026, Vol. 72, no. 3, p. 539–552.

Poor reproductive health is related to ovarian function, which may manifest as early/late puberty, premature ovarian insufficiency, and/or early menopause, and is increasingly observed worldwide. Mounting evidence implicates exposure to endocrine disrupting chemicals (EDCs) in these outcomes. EDCs are now ubiquitous in the environment, resulting in chronic low-level exposure of humans and animals to many chemicals. These compounds perturb endogenous hormone signaling, leading to both local and systemic endocrine disruption. Consequently, the hypothalamic-pituitary-ovarian (HPO) axis represents a primary target, with downstream consequences for ovarian function. Traditional toxicological approaches often rely on acute, high-dose exposures to single compounds, which fail to capture the complex reality of low-dose exposure to diverse chemical mixtures. Recognizing this gap, recent research has started to leverage more "real-life" exposure models that better represent these environmental conditions. This review summarizes current evidence on the impact of EDCs on reproductive health with a focus on ovarian function, highlights existing animal models that capitalize on more real-life exposure scenarios, and explores critical research considerations and future directions to advance our understanding of how real-world EDC mixtures disrupt ovarian function and female reproductive health. https://doi.org/10.1262/jrd.2025-084

A critical review of the epigenetic effects of bisphenols in animal models, Hasan, A., Biswas, S., Boyd, R. I., Wu, T. T., Lu, A. S., Martyniuk, C. J., Maitra, S., Niyogi, S. and Chivers, D. P., *Journal of Hazardous Materials Advances*, Aug 2026, Vol. 23.

Bisphenols, particularly Bisphenol A (BPA) and its analogs such as Bisphenol S (BPS) and Bisphenol F (BPF), are prominent environmental contaminants due to their extensive use in consumer products. These compounds are recognized as endocrine disrupting chemicals (EDCs) and are associated with several adverse effects in humans and wildlife, including reproductive, developmental, and metabolic dysfunctions. Increasing evidence suggests a role for epigenetic mechanisms underlying prolonged or delayed toxicity due to bisphenols. This review critically examines the epigenetic effects of BPA and its analogs across different animal and cell models, including human epidemiological studies, in vitro experiments, mammalian and fish models. Key findings show that bisphenol exposure alters DNA methylation, histone modifications, and non-coding RNA expression, implicating these changes in disrupted gene expression, reproductive impairments, and transgenerational effects. Notably, bisphenols dysregulate genes associated with lipid metabolism (Srebf1, Srebf2), steroidogenesis (star, cyp11a1, cyp17a1), immune function (Foxp3), and neurodevelopment (Grin2b, IGF-1, COMT), often through hypo- or hypermethylation of CpG islands

and promoter regions. Additionally, histone modifications such as H3K9me3, H3K4me3, and H3K27me3 and altered expression of miRNAs (miR-27b-3p, miR-200) further mediate these toxic effects. The dose-dependent and tissue-specific nature of epigenetic modifications further underscores the complexity of bisphenol toxicity. Molecular docking amongst different taxa also reveals moderate binding affinities of BPA, BPF, and BPS to DNMT enzymes as well as histone proteins, suggesting direct epigenetic effects at the level of the protein. By integrating multidisciplinary research, this review elucidates the molecular pathways underlying bisphenol-induced epigenetic changes and provides critical insights into their broader environmental and health implications. Moving forward, data on comparative toxicity of BPA analogs and the interplay between different epigenetic mechanisms are needed. In addition, greater emphasis should be placed on environmentally relevant exposure conditions, including chronic low-dose exposures and chemical mixtures. Future studies should investigate potential mitigation strategies, including the reversibility of bisphenol-induced epigenetic alterations through interventions. Addressing these challenges will be essential for advancing our mechanistic understanding of bisphenol-induced epigenetic reprogramming in humans and wildlife. https://doi.org/10.1016/j.hazadv.2026.101314

Bisphenol A exposure, stroke, and endothelial-related mechanisms: Association and mechanistic exploration,
He, J. R., Chen, Y. Q., You, N. L., Wang, M., Yu, C. L., Talic, S., Koh, H., Guo, Y. M., Zhang, M. M. and Gasevic, D., *Journal of Hazardous Materials Advances*, Aug 2026, Vol. 23.

Bisphenol A (BPA) has been implicated in various health outcomes, including cardiovascular and neurological disorders. However, its association with stroke remains insufficiently understood. We first analyzed data from the National Health and Nutrition Examination Survey (NHANES) to explore associations between BPA exposure and stroke prevalence. Network toxicology approaches were then used to identify shared targets and biological pathways between BPA and stroke. Key stroke-related genes were prioritized using LASSO, Boruta, and XGBoost, and were further characterized by single-cell RNA sequencing analysis. Molecular docking analyses were performed to assess the potential interactions between BPA and the identified target proteins. In NHANES, each unit increase in log10-transformed BPA was associated with 53% higher odds of stroke (OR=1.53, 95% CI 1.11-2.09). Network toxicology identified 59 overlapping BPA-stroke genes. A consensus of machine learning algorithms prioritized three key genes: ESR1, NOS3, and F13A1. Parsimonious prediction models demonstrated good discrimination in the held-out dataset (RF AUC=0.806; SVM AUC=0.791). Transcriptomic pathway analysis revealed increased activity of angiogenesis and PI3K-Akt-mTOR signaling pathways in stroke cases. Single-cell RNA sequencing identified 11 distinct brain cell clusters, with BPA-related gene signatures enriched in endothelial cell populations across a resting-to-activated trajectory. Molecular docking suggested potential structural compatibility between BPA and ESR1, NOS3, and F13A1. RT-qPCR and western blot analyses in BPA-treated HUVECs further supported expression-level changes in selected targets genes. BPA exposure was associated with stroke prevalence and may act through endothelial-related pathways and regulatory genes. Further studies are needed to clarify causality and mechanisms.
https://doi.org/10.1016/j.hazadv.2026.101422

Les phthalates, perturbateurs endocriniens de la reproduction : une menace croissante pour la fertilité féminine. Mémoire de Master en sciences pharmaceutiques.,
Kayiranga, L., *Faculté de pharmacie et des sciences biomédicales* (2026),
Les phtalates sont des perturbateurs endocriniens largement présents dans l'environnement et leurs métabolites, tels que le MEHP, sont détectés chez la majorité des femmes. L'objectif de ce travail est d'analyser et de synthétiser les connaissances actuelles relatives aux mécanismes par lesquels les phtalates affectent la fonction ovarienne et la fertilité féminine. Ces composés altèrent la fonction

ovarienne en perturbant la folliculogenèse, la stéroïdogenèse et des voies de signalisation cellulaire clés. Il en résulte une augmentation de l'apoptose, une diminution de la réserve ovarienne et une baisse de la qualité ovocytaire.
Les données humaines et animales mettent en évidence des conséquences cliniques telles que l'anovulation, l'irrégularité des cycles, le vieillissement ovarien accéléré et un risque accru d'insuffisance ovarienne prématurée. Les études de biosurveillance confirment une exposition mondiale influencée par l'âge, le statut socio-économique et l'environnement domestique ou professionnel. Les réglementations européennes visant à limiter l'usage des phtalates soulignent l'importance de réduire l'exposition pour protéger la santé reproductive féminine.
https://thesis.dial.uclouvain.be/entities/masterthesis/a76cf0a8-ae1a-4c1c-8bdc-77728e5d755e

Comparative evaluation of bisphenol A, bisphenol F and tetramethyl bisphenol F on adipogenesis, oxidative stress and inflammation in 3T3-L1 adipocytes,
Lee, S. M., Kim, H. J., Park, J. W., Kwon, J. M., Kim, H. M., Jeong, S. H., Moon, Y. G. and Kim, H. H., *Scientific Reports*, 2026/08/25 2026.

Bisphenol A (BPA) has long been recognized as an endocrine-disrupting chemical (EDC) with obesogenic properties, prompting widespread industrial substitution with structural analogues, among which bisphenol F (BPF) and tetramethyl bisphenol F (TMBPF) have gained particular commercial prominence. However, the metabolic safety of these alternatives remains inadequately characterized. The present study systematically compared the adipogenic, pro-oxidative, and pro-inflammatory effects of BPA, BPF, and TMBPF in differentiated 3T3-L1 murine preadipocytes and employed in silico molecular docking to elucidate their interactions with key nuclear receptors. Treatment with all three compounds at concentrations of 0.01–1 μM during adipocyte differentiation induced dose-dependent increases in intracellular lipid accumulation and lipid droplet size, with TMBPF eliciting the most pronounced effect (approximately 300% of MDI control at 1 μM). Consistently, mRNA and protein expression of the master adipogenic transcription factors PPARγ and C/EBPα, together with lipid metabolism markers including SREBP-1c, FABP4, FAS, and LPL, were significantly upregulated by all three compounds in a concentration-dependent manner. TMBPF also exhibited the strongest binding affinity for the PPARγ ligand-binding domain (docking score: −8.4 kcal/mol) and the glucocorticoid receptor (GR; −8.3 kcal/mol), consistent with its potent in vitro adipogenic activity. Adipokine profiling revealed increased expression of adiponectin, leptin, and resistin, with TMBPF treatment resulting in the most prominent upregulation of AdipoR1. Furthermore, TMBPF selectively suppressed antioxidant enzyme expression (Cat, Sod2, and Gpx1) and all three bisphenols markedly elevated secretion of pro-inflammatory cytokines TNF-α, IL-6, and MCP-1. Collectively, these findings indicate that TMBPF, despite its classification as a "safer" BPA alternative, exhibits greater adipogenic and oxidative disrupting potency compared to BPA and BPF, raising concerns that warrant further in vivo and epidemiological evaluation of TMBPF, particularly given its increasing use as a food-contact coating material. https://doi.org/10.1038/s41598-026-68045-0

Endocrine disruption by emergent brominated flame retardant Decabromodiphenyl ethane (DBDPE) promotes cancer-associated characteristics in human breast cells and zebrafish xenografts,
Lépine, M., Mcdermott, A., Patten, S. A. and Plante, I., *Environmental Research*, Oct 1 2026, Vol. 307.

Reports show that at least 1000 manufactured chemicals released into the environment are associated with endocrine disruption. Among these, halogenated flame retardants are a source of concern due to their ubiquity, toxic properties, and adverse health effects. Replacing

polybrominated diphenyl ethers (PBDEs), a well-studied family of compounds whose use is currently restricted, with compounds of similar chemical structure and fire resistance, such as Decabromodiphenyl ethane (DBDPE), leads to the presumptions of comparable health effects. Because of the reported toxicity of PBDEs, the presence of DBDPE in breast milk raises concerns about its potential effects on breast cancer. We used cancerous and non-cancerous human breast cell lines, as well as a zebrafish xenograft model, to investigate the toxicity of this replacement compound. Exposure to DBDPE led to a non-significant increased expression of progesterone receptor B (PRB) at lower doses followed by a dosedependent decreasing trend, while thyroid hormone receptor α1 (THRα1) was decreased in cancer cells (T47D). The (3-catenin/Wnt pathway, associated with proliferation regulation and the mesenchymal epithelial transition, was also affected. Interestingly, while proliferation and migration were not affected in both cell lines exposed to DBDPE in classical cell culture, both capacities were increased when cells were xenografted in exposed zebrafish. These results suggest that, such as PBDEs, DBDPE can affect breast cells, and highlight the importance of testing the potential hazards of replacement molecules before their widespread use. https://doi.org/10.1016/j.envres.2026.125393

Oxidative stress-mediated DNA damage and macrophage polarization microenvironment: A novel mechanism of DCHP-induced male reproductive system impairment,
Li, Q., Yang, Y., Jiang, D., Han, X., Li, J., Wang, Y., Chao, L., Huang, C. and Fan, Z., *Reproductive Toxicology*, 2026/09/01/ 2026, Vol. 144, p. 109308.

The global decline in male fertility has emerged as a major public health challenge, with mounting evidence pointing to a close association between endocrine-disrupting chemicals (EDCs) and reproductive dysfunction. As a representative member of this class, dicyclohexyl phthalate (DCHP) exhibits strong environmental persistence and bioaccumulation potential within the mammalian reproductive system. In the present study, male ICR mice were orally administered DCHP at 10, 100, or 300 mg/kg/day for six consecutive weeks, and GC-2 mouse spermatocyte cells were exposed to 5, 20, or 50 μM DCHP for 24 h. Long-term, high-dose DCHP exposure (300 mg/kg/day) was associated with significant testicular histopathological alterations and impaired epididymal sperm parameters in mice. Both the in vivo and in vitro models revealed that DCHP treatment correlated with cell cycle disruption and proliferative arrest. Western blotting and TUNEL assays further indicated that DCHP could triggered apoptosis. As an exploratory acute ex vivo assessment, 35 human semen samples were exposed to DCHP (5, 20 and 50 μM) for 12 h, which revealed a dose-dependent reduction in sperm quality. At the mechanistic level, our data suggest a potential cascade wherein DCHP exposure is linked to oxidative stress, concomitant DNA damage, and mitochondrial dysfunction. RNA-seq analysis further highlighted the enrichment of inflammatory responses and related signaling pathways. Subsequent validation experiments showed that DCHP exposure was accompanied by an increased testicular M1 macrophage population, elevated secretion of pro-inflammatory cytokines (IL-6, TNF-α), and activation of the NF-κB pathway—changes that may collectively contribute to an unfavorable testicular microenvironment and impaired sperm quality. Overall, our findings reveal close associations between DCHP exposure and testicular injury, proposing a working model involving oxidative stress and inflammation; however, further functional studies are required to establish definitive causal relationships.
https://doi.org/10.1016/j.reprotox.2026.109308

Oxidative stress-mediated DNA damage and macrophage polarization microenvironment: A novel mechanism of DCHP-induced male reproductive system impairment,
Li, Q. N., Yang, Y., Jiang, D. N., Han, X. J., Li, J., Wang, Y. H., Chao, L., Huang, C. Z. and Fan, Z. H., *Reproductive Toxicology*, Sep 2026, Vol. 144.

The global decline in male fertility has emerged as a major public health challenge, with mounting evidence pointing to a close association between endocrine-disrupting chemicals (EDCs) and reproductive dysfunction. As a representative member of this class, dicyclohexyl phthalate (DCHP) exhibits strong environmental persistence and bioaccumulation potential within the mammalian reproductive system. In the present study, male ICR mice were orally administered DCHP at 10, 100, or 300 mg/kg/day for six consecutive weeks, and GC-2 mouse spermatocyte cells were exposed to 5, 20, or 50 mu M DCHP for 24 h. Long-term, high-dose DCHP exposure (300 mg/kg/day) was associated with significant testicular histopathological alterations and impaired epididymal sperm parameters in mice. Both the in vivo and in vitro models revealed that DCHP treatment correlated with cell cycle disruption and proliferative arrest. Western blotting and TUNEL assays further indicated that DCHP could triggered apoptosis. As an exploratory acute ex vivo assessment, 35 human semen samples were exposed to DCHP (5, 20 and 50 mu M) for 12 h, which revealed a dose-dependent reduction in sperm quality. At the mechanistic level, our data suggest a potential cascade wherein DCHP exposure is linked to oxidative stress, concomitant DNA damage, and mitochondrial dysfunction. RNA-seq analysis further highlighted the enrichment of inflammatory responses and related signaling pathways. Subsequent validation experiments showed that DCHP exposure was accompanied by an increased testicular M1 macrophage population, elevated secretion of pro-inflammatory cytokines (IL-6, TNF-alpha), and activation of the NF-kappa B pathway-changes that may collectively contribute to an unfavorable testicular microenvironment and impaired sperm quality. Overall, our findings reveal close associations between DCHP exposure and testicular injury, proposing a working model involving oxidative stress and inflammation; however, further functional studies are required to establish definitive causal relationships.
https://doi.org/10.1016/j.reprotox.2026.109308

PFAS disrupt adult hippocampal neurogenesis and synaptic remodeling associated with NOX2/ROS/Ferroptosis activation,
Li, X., Li, X., Li, C., Cao, L., Li, S., Wang, X., Du, K., Cui, Y., Ma, Y., Wang, X., Xing, X., Feng, H., Yang, C., Wang, X. and Li, C., *Environment International*, 2026/09/01/ 2026, p. 110503.

Per- and polyfluoroalkyl substances (PFAS) accumulate in the hippocampus, yet their effects on adult hippocampal neurogenesis (AHN) remain unclear. In this study, adult male mice were orally exposed to PFOA or GenX (2 or 10 mg/kg/day) for 28 days. Behavioral performance, AHN, synaptic remodeling, microglial morphology, and redox status were evaluated using behavioral assays, Nissl and Golgi staining, BrdU immunolabeling, microglial morphometric analyses, and Western blotting. PFOA exposure and high-dose GenX exposure reduced time spent and distance traveled in the center area of the open field and impaired spatial learning and memory performance in the Morris water maze, as indicated by increased escape latency and fewer platform crossings. In contrast, PFOA and low-dose GenX exposure reduced exploration of the open arms in the elevated plus maze. PFOA and GenX reduced dendritic spine density with fewer mushroom/thin spines and more stubby spines. PFOA preferentially decreased PSD95 (postsynaptic marker), whereas GenX reduced synaptophysin (presynaptic marker). In the subgranular zone (SGZ), survival and neuronal differentiation of neural stem cells were diminished, and asymmetric divisions of radial glia-like cells increased, suggesting stem-cell pool depletion. Microglia exhibited a hyper-ramified/bushy reactive phenotype. Hippocampal ROS/MDA rose, NOX2 components (GP91phox/P22phox) were upregulated, and ferroptosis defenses (SLC7A11/GLS2/GPX4) were downregulated. Collectively, PFOA and GenX disrupt hippocampal synaptic remodeling and AHN and are associated with increased oxidative stress and ferroptosis-related alterations. Differential pre- versus postsynaptic vulnerabilities may contribute to the distinct behavioral alterations observed following PFOA and GenX exposure. This work provides new insights into the neurotoxic effects of PFOA and GenX and identifies potential

pathways involved in PFAS-induced hippocampal dysfunction.
https://doi.org/10.1016/j.envint.2026.110503

Epigenetic vulnerability in endocrine disruption: bridging mechanisms, models, and environmental inequities,
Lizcano, F., Ramírez-Bohorquez, M. C., Ballesteros-Garcia, M. C., Vargas, V. and Cardenas, S., *Clinical Epigenetics*, Jun 6 2026, Vol. 18, no. 1.

This narrative mini-review examines how endocrine-disrupting chemicals (EDCs) induce persistent epigenetic alterations that shape endocrine, metabolic, and developmental trajectories across the lifespan. It addresses the emerging gap in integrating mechanistic epigenetic evidence with population-level vulnerability, particularly in underrepresented regions such as Latin America. These effects are especially critical during sensitive windows-including fetal development, childhood, and puberty-when DNA methylation, histone modifications, and non-coding RNA networks establish long-term molecular programming. While animal models have provided key mechanistic insights, their translational limitations highlight the need for human-relevant systems capable of capturing endocrine-specific and epigenetic endpoints under environmentally realistic conditions. We propose that epigenetic programming represents a central biological interface linking EDC exposure to context-dependent vulnerability. New Approach Methodologies (NAMs)-including organoids, micro-physiological systems, and computational models-offer a translational bridge aligned with Adverse Outcome Pathway frameworks. Importantly, susceptibility to endocrine disruption is shaped not only by biological factors but also by environmental, nutritional, and socioeconomic determinants. Evidence from Latin American populations-including altitude gradients, nutritional transitions, and structural inequalities-illustrates how these contextual factors interact with epigenetic mechanisms to influence metabolic and endocrine outcomes. This integrative perspective supports the development of more equitable and context-sensitive approaches to endocrine toxicology.
https://doi.org/10.1186/s13148-026-02158-1

BPA and Male Reproductive Health: Mechanistic Insights, Toxicological Implications, and the Protective Role of Antioxidants,
Maniradhan, M., *Journal of Applied Toxicology*, 2026.

Bisphenol A (BPA) is a ubiquitous endocrine-disrupting chemical commonly used in producing polycarbonate plastics and epoxy resins, resulting in widespread human exposure through various sources. BPA mimics estrogen and disrupts the hypothalamic-pituitary-testicular (HPT) axis, impairing androgen signaling, steroidogenesis, and spermatogenesis. It induces oxidative stress by promoting the excessive generation of reactive oxygen species (ROS), resulting in lipid peroxidation, DNA damage, mitochondrial dysfunction, and apoptosis of germ cells. BPA also compromises the antioxidant defense systems by downregulating key enzymes such as superoxide dismutase, catalase, and glutathione peroxidase, contributing to redox imbalance. BPA exposure during critical developmental windows is particularly concerning, as it can cross the placenta and has been detected in amniotic fluid, cord blood, placenta, and breast milk. Early-life exposure may have long-term consequences for male reproductive health. Furthermore, BPA-induced epigenetic modifications suggest the potential for transgenerational reproductive effects. Recent studies have demonstrated the ameliorative effects of various antioxidants, including alpha-lipoic acid, melatonin, selenium, vitamins, and phytochemicals, in mitigating BPA-induced testicular toxicity. These agents help to restore redox homeostasis, preserve mitochondrial integrity, and attenuate inflammation and apoptosis. This review uniquely integrates endocrine disruption, oxidative stress, mitochondrial dysfunction, and epigenetic alterations to provide a comprehensive mechanistic framework. It also highlights emerging pathways such as lipid peroxidation-mediated damage and

evaluates antioxidant interventions based on their molecular targets. A comprehensive understanding of the molecular pathways underlying BPA-induced reproductive toxicity and the mechanistic basis of antioxidant-mediated protection is imperative for developing effective interventions to protect male reproductive health. https://doi.org/10.1002/jat.70320

The hepato-exposome axis: How endocrine disruptors hijack liver receptors to drive MASLD, Martín-Grau, M., Kamstra, J. H., Hyötyläinen, T., Orešič, M., Song, Y., Veličković, N., Djordjevic, A., Milutinovic, D. V., Jackson, R. F., Van Vorstenbosch, R., Massart, J., Vidal-Puig, A. and Carobbio, S., *Journal of Hepatology*, 2026/06/22/ 2026.

Summary Metabolic dysfunction–associated steatotic liver disease (MASLD) is increasing worldwide at a pace that cannot be fully explained by obesity, diet, or genetics alone. Emerging evidence provides mechanistic insight into how defined endocrine-disrupting chemicals (EDCs) rewire hepatic transcriptional and metabolic networks relevant to MASLD. This review focuses on the molecular interfaces through which major EDC families intersect with hepatic metabolic regulation. We propose the "hepato-exposome axis" as a receptor-centric framework that maps specific EDC classes to nuclear receptors (NRs) and key metabolic pathways involved in lipid metabolism, glucose homeostasis, mitochondrial function, and inflammatory signalling that are implicated in MASLD progression. We summarise key EDC groups linked to liver disease, including organochlorine pesticides, pyrethroids, bisphenols, phthalates, organophosphate esters, and per- and polyfluoroalkyl substances (PFAS). We then integrate current understanding of hepatic metabolic and xenobiotic pathways in homeostasis and MASLD, emphasising xenobiotic-sensing and metabolic NRs (AhR, PXR, CAR, PPARs, FXR, LXRs, RXR) as convergence points for EDC action. Drawing on in vivo and in vitro studies, we show that distinct EDC families imprint overlapping molecular signatures onto pathways that control de novo lipogenesis, β-oxidation, glucose handling, antioxidant defences, and inflammatory/immune signalling, collectively fostering a pro-steatotic and pro-inflammatory hepatic milieu. We further discuss vulnerable populations, critical windows of exposure, mixture effects, and sex-specific responses, positioning EDCs as environmental modifiers of the MASLD trajectory across the life course. Finally, we outline priorities for mechanistic and translational research, including mixture toxicology, multi-omics exposome profiling, and biomarker discovery, to better quantify and mitigate endocrine disruptors' contribution to the global MASLD burden. https://doi.org/10.1016/j.jhep.2026.06.021

Fluorene-9-Bisphenol (BHPF), A Silent Endocrine Disruptor: A Comprehensive Toxicological Assessment of Molecular Mechanisms Driving Multi-Organ Toxicity, Mujeeb, S., Zaheen, S., Khan, A. M., Chiou, C.-C. and Chen, C.-C., *Journal of Hazardous Materials Advances*, 2026/08/31/ 2026, p. 101532.

Fluorene-9-bisphenol (BHPF), a structurally rigid bisphenol A (BPA) substitute widely used in polyesters, epoxy resins, polyurethanes, and food-contact plastics, has emerged as a persistent global contaminant detected in human serum, breast milk, and multiple tissues. Extensive animal and cell-based evidence, spanning mussels, zebrafish, mice, and porcine models, shows that BHPF accumulates in the liver, brain, heart, intestine, and reproductive organs, producing organ-specific and systemic damage through convergent mechanisms including oxidative stress, mitochondrial dysfunction, inflammatory and immune activation, and programmed cell death via apoptosis and ferroptosis. By antagonizing estrogen receptors (ERα, GPER) and disrupting the hypothalamic-pituitary-gonadal and gut-brain axes, BHPF impairs steroidogenesis, reproductive and neurodevelopmental function, hepatic metabolism, cardiovascular homeostasis, and intestinal-immune balance. Despite this growing toxicological evidence, human epidemiological data remain scarce, and no remediation strategy has been specifically designed for BHPF's unique

physicochemical properties; current mitigation approaches, advanced oxidation, adsorption, membrane separation, biological degradation, and surfactant-assisted techniques, are merely extrapolated from BPA-targeted technologies of uncertain transferability. Critical gaps persist regarding degradation-product toxicity and BHPF-selective adsorbent design. This review synthesizes BHPF's exposure routes, organ-level toxicities, and molecular mechanisms, evaluates adapted remediation strategies, and highlights the urgent need for human biomonitoring, mechanistic clarification, and BHPF-specific environmental management and regulatory frameworks. https://doi.org/10.1016/j.hazadv.2026.101532

Interplay between thermal contact and hormonal activity: A kinetic analysis on smartphone-induced endocrine disruption,
Onikanni, S. A., Nhung, N. T. A., Amos, O. E., Dao, T. N. P., Aribigbola, T., Olayinka, O. S., De Carvalho, D. P. and Miranda-Alves, L., *Journal of Steroid Biochemistry and Molecular Biology*, Nov 2026, Vol. 264.

Endocrine toxicology has largely overlooked persistent, behavior-linked thermal microenvironments established by smartphones, a phenomenon characterized by near-continuous human-thermal interaction. In this review, we propose a mechanistic model of the thermokinetic endocrine interface to explain how heat from smartphones might alter endocrine-relevant systems. Offering a new view on chemical interactions with consumer devices, we posit that localized thermal pulses, not continuous low-dose exposure, can accelerate the migration of polymer additives, influence desorption and diffusion dynamics, and intensify the transfer of surface chemicals to the skin. Materials science, dermal pharmacology, and endocrine signaling evidence indicate that gentle heating can alter the stratum corneum's permeability, tissue distribution, receptor mobility, and ligand-receptor residence kinetics. We hypothesize that smartphone-generated heat could act as a kinetic amplifier, repurposing intermittent contact into temporally regulated internal microdosing and altered signaling responsiveness thereby mitigating its role as a primary endocrine toxicant. Whereas current perspectives reorient risk evaluation from static external exposure benchmarks to evolving internal dose profiles, thermal exposure configurations, and biological temporal dynamics. We propose using temperature-integrated physiologically based pharmacokinetic models, warm-exposure endocrine assays, and real-world migration analytics for strategic prioritization. By incorporating thermal biology into endocrine risk assessment, new pathways linking everyday devices to hormonal disruption can be discovered. https://doi.org/10.1016/j.jsbmb.2026.107081

Evaluation of the reproductive toxicity of endocrine disruptors during early embryo development,
Parra-Forero, L. Y. and Nowak, R. A., *Biology of Reproduction*, 2026.

Because the blastocyst is a critical stage of early embryonic development, developmental abnormalities can serve as sensitive biomarkers of reproductive toxicity. This review focuses on the impact of various endocrine disrupting chemicals (EDCs) including phthalates, bisphenols, parabens, and per- and polyfluoroalkyl substances on blastocyst formation, structure, and function. Murine blastocyst development models offer a controlled and reproducible platform to study cell differentiation, implantation potential, and epigenetic regulation. In vitro systems allow simulation of internal EDC concentrations found in human biological fluids, enabling dose-response relationships that reflect real world exposure. Endocrine disrupting chemicals present in reproductive fluids such as follicular, oviductal, and endometrial fluid can impair oocyte quality and compromise embryonic development, even in morphologically normal embryos. Blastocysts exposed to EDCs during development show alterations in lineage specification markers (e.g., OCT4, SOX2, CDX2), cytoskeletal organization, and nuclear integrity, suggesting disruption of key developmental pathways. This review systematically reviews in vitro and in vivo evidence, along with proposed

mechanisms of action, for each group of EDCs. Importantly, only studies reporting blastocyst formation rates were included to ensure relevance and comparability. The synthesis identifies common toxicity patterns, potential synergistic effects, and transgenerational consequences. The findings have direct implications for human fertility, particularly in the context of assisted reproductive technologies, and highlight the need for improved regulatory frameworks and biomarker validation. Overall, the blastocyst emerges as a powerful tool for understanding and mitigating the reproductive risks associated with environmental chemical exposure. https://doi.org/10.1093/biolre/ioag154

Impact of microplastics on reproductive health: Genetic and epigenetic insights into idiopathic recurrent pregnancy loss and infertility – A Narrative Review,
Praveen Kumar, K. S., Bhat, D. and Prashant, A., *F&S Reviews*, 2026/08/12/ 2026, p. 100119.

Microplastics (MPs) have emerged as pervasive environmental pollutants with growing evidence of bioaccumulation in human tissues, including the reproductive system. Although their ecological and metabolic toxicity is well recognized, their role in human reproductive dysfunction, particularly recurrent pregnancy loss (RPL) and infertility, remains insufficiently explored. This review summarises current evidence on the genetic, epigenetic, endocrine, and immunological mechanisms underlying the associations between MP exposure and adverse reproductive outcomes reported in human observational studies; however, causal relationships remain unestablished. This review synthesizes current evidence on the genetic, epigenetic, endocrine, immunological, and molecular mechanisms through which MPs may impair reproductive health. MPs enter the body through ingestion, inhalation, and dermal exposure, subsequently accumulating in reproductive organs and placental tissues, where they may act as carriers of endocrine-disrupting chemicals. Experimental studies indicate that MP exposure promotes oxidative stress, mitochondrial dysfunction, inflammation, DNA damage, endocrine dysregulation, and altered gene expression, leading to chromosomal instability and epigenetic alterations, including DNA methylation, histone modifications, and microRNA dysregulation. Current human evidence, including placental, biomonitoring, and assisted reproductive technology (ART)-related studies, supports the biological plausibility of MP-associated reproductive toxicity, although direct clinical evidence linking MP exposure to RPL remains limited. Unlike previous reviews, this article integrates mechanistic evidence with emerging human clinical findings to provide a comprehensive framework connecting environmental exposure, genetic and epigenetic regulation, and reproductive dysfunction. Furthermore, it identifies key translational knowledge gaps and outlines future research priorities aimed at improving risk assessment, biomarker discovery, and preventive strategies for MP-associated reproductive disorders. https://doi.org/10.1016/j.xfnr.2026.100119

Environmental endocrine disruptors in cardio-oncology: emerging modifiers of cardiovascular vulnerability in patients with cancer,
Quagliariello, V., Berretta, M., D'aiuto, G., Bisceglia, I., Barbato, M., Del Toro, B., Santagata, J., Canale, M. L., Paccone, A., Inno, A., D'ambrosio, C., Oliva, S., Dessalvi, C. C., Di Matola, T., Gabrielli, D., Fabiani, I., Pagliani, L., Iliescu, C. A., Borowiec, A. and Maurea, N., *Cardio-Oncology*, Jun 19 2026, Vol. 12, no. 1.

BackgroundCardio-oncology has traditionally focused on treatment-related cardiovascular toxicity and conventional cardiovascular risk factors. However, increasing evidence suggests that environmental exposures may contribute to both cancer development and cardiovascular disease through shared biological mechanisms. Endocrine-disrupting chemicals (EDCs) are ubiquitous environmental contaminants capable of interfering with hormonal signaling, metabolic homeostasis, vascular function, and inflammatory pathways. Despite growing evidence linking EDCs

to cardiometabolic disorders and hormone-sensitive malignancies, their potential role within the cardio-oncology continuum remains largely unexplored.MethodsThis narrative review summarizes and critically discusses current experimental, translational, and epidemiological evidence regarding the potential contribution of environmental endocrine disruptors to cardiovascular risk and cancer biology. Particular attention is given to molecular pathways relevant to cancer therapy-related cardiovascular toxicity, breast cancer biology, adipose tissue dysfunction, and emerging exposomic determinants of long-term cardiovascular outcomes in patients with cancer.Main bodyMajor classes of EDCs, including bisphenols, phthalates, per- and polyfluoroalkyl substances (PFAS), persistent organic pollutants (POPs), parabens, pesticides, and other environmental contaminants, are continuously encountered through food systems, plastics, consumer products, contaminated water, and healthcare materials. These compounds influence multiple biological processes that are central to both oncogenesis and cardiovascular disease, including oxidative stress, mitochondrial dysfunction, endothelial injury, chronic inflammation, metabolic reprogramming, thrombosis, and epigenetic remodeling. In breast cancer, EDCs may modulate subtype-specific signaling pathways involving estrogen receptor activation, HER2 crosstalk, aryl hydrocarbon receptor signaling, and homologous recombination networks. In parallel, growing evidence supports associations between EDC exposure and hypertension, accelerated atherosclerosis, heart failure, metabolic syndrome, and major adverse cardiovascular events. We further discuss the hypothesis that lipophilic EDCs may accumulate within adipose depots and that dysfunctional epicardial adipose tissue could represent a local toxicological niche capable of amplifying cardiovascular vulnerability in cancer survivors, although direct evidence remains unavailable.ConclusionsEDCs should not yet be considered established cardio-oncology risk factors; however, they represent biologically plausible exposomic modifiers operating at the intersection of cancer, metabolism, and cardiovascular disease. Incorporating environmental exposures into cardio-oncology research may improve understanding of interindividual variability in cardiovascular outcomes and open new avenues for risk stratification, prevention, and survivorship care. Future prospective studies integrating exposure biomarkers, adipose tissue biology, and cardiovascular phenotyping are warranted to define the clinical relevance of EDCs in patients with cancer. https://doi.org/10.1186/s40959-026-00532-9

An integrative review of Bisphenol A (BPA): mechanistic insights into exposure dynamics, multiorgan toxicity, and biomolecular pathways,
Rabby, T., Rafi, M. T. H., Sultana, N., Saha, S., Naziha, I. J., Islam, S., Chen, U., Sumi, S. A., Hussain, M. S., Danishuddin and Haque, M. A., *Environmental Reviews*, 2026/08/21/ 2026, Vol. 34, p. 1–31.

Bisphenol A (BPA) is a highly consumed synthetic compound used in the production of polycarbonate plastics and epoxy resins, with global production exceeding six billion pounds yearly. Alarmingly, BPA leaching into food, beverages, and the environment results in hazardous exposure to humans and wildlife. The current review provides a holistic assessment of BPA, focusing on the convergence of environmental exposure dynamics, molecular toxicity pathways, and their consequences for human health and regulatory policy. By synthesizing evidence from experimental, epidemiological, and environmental studies, the current approach examines how BPA interacts with major organ systems through receptor-mediated signaling, oxidative stress, and epigenetic modulation and how these processes may contribute to multisystem outcomes, such as endocrine, metabolic, neurological, and immune alterations. Particular focus is given to emerging studies on immunological effects and the pervasiveness of BPA in environmental matrices, supported by biomonitoring data. The increasing use of BPA analogues is also critically examined in the current review, which raises concerns regarding the structural similarities and the possibility of "regrettable substitution". Although a considerable number of investigations indicate ties between exposure to BPA and negative health outcomes, inconsistent study designs and exposure contexts highlight the need for careful interpretation. By integrating mechanistic insights with real exposure patterns and

changing regulatory stances that call for increased policy enforcement, the development of alternative materials, and ongoing biomonitoring, the current review aims to provide a more coherent framework for understanding BPA-related risks and to direct future research priorities and risk management strategies. https://doi.org/10.1139/er-2025-0245

Toxic effects of perfluoroalkyl and polyfluoroalkyl substances (PFAS) on the gut microenvironment and their potential association with colorectal cancer,
Tang, X. F. and Tang, L. X., *Toxicology Mechanisms and Methods*, 2026.

Per- and polyfluoroalkyl substances (PFAS) are persistent surfactants with ingestion as a major exposure route, positioning the intestine as a primary site of contact. This narrative review integrates mechanistic toxicology, multi-omics microbiology, and human observational studies to evaluate whether PFAS-associated disruption of intestinal homeostasis could contribute to colorectal cancer (CRC). In vitro epithelial systems and animal models indicate that selected PFAS can impair barrier function through altered membrane properties and reduced tight-junction expression, increasing paracellular permeability and luminal antigen translocation. PFAS may also perturb goblet-cell secretion and mucus organization, in part through endoplasmic reticulum (ER) stress and altered autophagy, thereby facilitating mucosa-associated bacterial adherence. Stress signaling can converge on mitochondrial dysfunction and reactive oxygen species (ROS) generation that primes inflammasome activity and cytokine-mediated inflammation. In colon cell models, PFOA and PFOS have been associated with modulation of Wnt/beta-catenin signaling and downstream transcriptional programs linked to proliferative and invasive phenotypes. At the community level, exposure-associated dysbiosis includes loss of butyrate-producing taxa and disruption of bile acid pools, consistent with reduced short-chain fatty acids (SCFAs) and altered farnesoid X receptor signaling. However, epidemiologic findings remain inconsistent, including null and inverse associations that may reflect reverse causation from occult bleeding, exposure misclassification, residual dietary confounding, non-monotonic dose responses, congener heterogeneity, and species-specific toxicokinetics. We propose priorities for future work including long-lag prospective sampling, physiologically based pharmacokinetic (PBPK)-informed exposure reconstruction, and adverse outcome pathway (AOP)-anchored multi-omics endpoints for causal inference and regulation. https://doi.org/10.1080/15376516.2026.2695156

Mechanisms by which “Everywhere” and “Forever” Environmental Chemicals Impact Ovarian Function and Female Fertility,
Um, D. and Flaws, J. A., *Biology of Reproduction*, 2026.

Phthalates and per- and polyfluoroalkyl substances (PFAS) are widespread environmental contaminants that are consistently detected in follicular fluid, serum, and reproductive tissues. This review synthesizes current experimental and epidemiological evidence on the molecular and cellular mechanisms by which phthalates and PFAS impair ovarian function and female fertility. Human studies primarily identify exposure-outcome associations, whereas animal, ex vivo, and in vitro models provide most of the causal and mechanistic evidence. Phthalates and PFAS disrupt interconnected pathways involved in folliculogenesis, steroidogenesis, mitochondrial homeostasis, inflammatory signaling, and cell survival. These chemicals dysregulate pathways governing primordial follicle activation and ovarian reserve maintenance, and impair estradiol and progesterone synthesis. A central mechanistic theme by which phthalates and PFAS impair ovarian function and fertility involves mitochondrial dysfunction, which promotes oxidative stress and contributes to apoptosis. However, evidence for phthalate- and PFAS-induced ovarian necroptosis, pyroptosis, immune-cell infiltration, and inflammation-driven fibrosis remains limited and is often based on a small number of studies conducted at doses above typical human exposures. Although

mixture studies are relatively scarce, available data indicate that mixtures can perturb mitochondrial activity, steroid secretion, follicle dynamics, inflammatory signaling, and Hippo-pathway endpoints. Overall, altered folliculogenesis and steroidogenesis, mitochondrial dysfunction, oxidative stress, and apoptosis emerge as the best-supported mechanisms linking phthalate and PFAS exposure to ovarian toxicity. This review also highlights the need for exposure-relevant studies, quantitative pathology, and stronger integration of experimental mechanisms with human biomonitoring data. https://doi.org/10.1093/biolre/ioag184

Editorial overview: Endocrine disrupting chemicals and the thyroid hormone system,
Van Gerwen, M. and Goldner, W., *Current Opinion in Endocrine and Metabolic Research*, 2026/09/01/ 2026, Vol. 44, p. 100622.
https://doi.org/10.1016/j.coemr.2026.100622

Unraveling the association between triclosan and breast cancer: A multidimensional research strategy integrating machine learning, mendelian randomization, and molecular dynamics simulations,
Wu, D., Yuan, J., Xu, H., Yang, C., Pan, J., Li, G., Dai, W., Gan, J., Jiao, J. and Wu, X., *Environ Int*, Aug 6 2026, Vol. 215, no. 1873-6750 (Electronic), p. 110455.

OBJECTIVE: The association between triclosan and breast cancer remains controversial, with early studies suggesting antitumor effects and recent evidence indicating pro-carcinogenic risks, but the key targets and molecular mechanisms remain unclear. This study integrated computational toxicology, network toxicology, machine learning, Mendelian randomization, and in vitro experiments to determine the direction of triclosan's effect on breast cancer and systematically elucidate its core molecular mechanisms. METHODS: ProTox3 predicted carcinogenicity. Triclosan targets (SwissTargetPrediction/PharmMapper) were intersected with breast cancer DEGs (GSE42568/GSE70947) to build a PPI network. Three ML algorithms (RF, LASSO, SVM-RFE) with SHAP screened hub genes. Two-sample MR assessed causality; docking and 100 ns MD verified binding. MCF-7 cells were treated with triclosan to assess proliferation (CCK-8, EdU, Ki-67), colony formation, migration, cell cycle progression, and NR3C1 expression, with siRNA knockdown further confirming the mediation. RESULTS: ProTox3 predicted moderate endocrine-related carcinogenicity. Intersection of 2,732 DEGs with triclosan targets yielded 98 candidates. Five hub genes (ADH5, PDK2, NR3C1, PTGS2, FABP4) were identified; RF AUC = 0.939. MR confirmed NR3C1 inversely causal on breast cancer risk (OR = 0.730, 95% CI:0.619-0.861, P < 0.001), and high NR3C1 predicted longer survival (HR = 0.86, 95% CI:0.77-0.95, P = 0.0042). MD showed stable binding (free energy: -116.676 ± 1.895 kJ/mol). In vitro, triclosan promoted proliferation (CCK-8/EdU, P < 0.01), colony formation/migration (P < 0.01), increased S-phase (P < 0.05), and reduced NR3C1 (P < 0.05); knockdown enhanced this, confirming downregulation. CONCLUSION: Triclosan stably binds and suppresses NR3C1, removing NR3C1-dependent restraint on proliferation, colony formation, migration, and cell cycle progression. NR3C1 negatively correlates with risk and is a favorable prognostic marker. This work provides a multi-level evidence chain from binding to prognosis, clarifying triclosan's pro-cancer mechanisms and offering clues for risk evaluation and intervention of endocrine-disrupting chemical-associated breast cancer.
https://doi.org/10.1016/j.envint.2026.110455

Organophosphate flame retardants and incident type 2 diabetes: Integrating population evidence, mechanistic insights, and effect modification by healthy lifestyle,
Wu, X., Zhou, Y., Nie, Y., Xu, H., Liu, H., Cheng, M., Guo, Y., Lin, F., Tang, X., Chen, P., Wei, D., Hou, J., Huo, W., Liu, X., Li, L., Wang, C., Fan, C., Chen, H., Chen, T. and Mao, Z., *Journal of Hazardous Materials*, 2026/09/15/ 2026, Vol. 516, p. 143347.

Higher exposure to organophosphate flame retardants (OPFRs) may contribute to type 2 diabetes mellitus (T2DM), but prospective evidence, lifestyle modification, and biological plausibility remain insufficiently characterized. We conducted a nested case-control study within the Henan Rural Cohort to evaluate associations between OPFRs exposure and incident T2DM. Cox models, WQS, QGC, BKMR, and an adaptive elastic-net-based environmental risk score were used to assess single and mixed OPFRs exposure. Baseline and trajectory-based healthy lifestyle scores were applied to evaluate joint effects, and signed Wald χ^2 decomposition quantified contributions of OPFRs and lifestyle domains. Mechanistic evidence was integrated from in vitro experiments and toxicogenomic analyses, including RNA-sequencing, network toxicology, molecular docking, and GEO re-analysis, to evaluate EGFR-related signaling and build an integrative adverse outcome pathway. Higher urinary OPFRs, particularly TPHP, were associated with increased T2DM risk, and OPFRs mixtures showed a linear dose-response relationship with T2DM risk. High OPFRs exposure combined with unhealthy or deteriorating lifestyle trajectories conferred the greatest risk, with OPFRs plus lifestyle explaining 55.36% of total model χ^2. In HepG2 cells, TPHP impaired glucose consumption and downregulated EGFR, whereas EGFR overexpression alleviated the TPHP-associated abnormal glucose consumption. EGFR overexpression was confirmed at both mRNA and protein levels. The p-AKT/total AKT results provided supportive evidence of AKT-related signaling changes, whereas total GLUT2 protein abundance was not significantly altered. Molecular docking supported potential TPHP-EGFR binding affinity of −8.5 kcal/mol, and GEO analyses provided external supportive evidence that EGFR expression tended to be higher in healthier lifestyle-related conditions. These findings suggest that mixed OPFRs exposure, dominated by TPHP, was associated with higher incident T2DM risk, partly attenuated by healthier lifestyle trajectories, supporting combined exposure-reduction and lifestyle-based prevention strategies.
https://doi.org/10.1016/j.jhazmat.2026.143347

Endocrine Noise: Sex-Specific Disruption of Hypothalamic-Pituitary-Adrenal (HPA) Axis by Endocrine-Disrupting Chemicals,
Xega, V., Yang, M. H. and Liu, J. L., *Sexes*, Apr 23 2026, Vol. 7, no. 2.

Environmental chemicals are rarely considered stressors in the way that psychological or physical stressors are. Yet many endocrine-disrupting chemicals (EDCs) interact with the body's core stress response system. This review examines how EDCs alter hypothalamic-pituitary-adrenal (HPA) regulation and how biological sex influences those responses. Drawing on human epidemiological data and experimental models, we describe how EDC exposure affects cortisol dynamics, feedback sensitivity, and adrenal signaling, with a particular focus on sex-dependent outcomes. We propose the concept of endocrine noise to describe how low-dose, often mixed EDC exposures introduce persistent interference into hormone signaling without necessarily causing overt endocrine deficiency or excess. In this framework, EDCs act as chronic, low-grade stressors that reset the timing, feedback precision, and rhythmic organization of the HPA axis rather than as isolated reproductive toxicants. We argue that EDCs should be understood as chronic, context-dependent stress modifiers that reshape sex-specific "risk architectures" for affective, metabolic, and immune disorders. Recognizing sex-specific HPA architecture and endocrine noise has immediate implications for study design and regulation, including the need for sex-stratified analyses, circadian-sensitive sampling of cortisol, and risk assessments that consider how the same exposure can push female and male stress systems in divergent directions.
https://doi.org/10.3390/sexes7020022

Association between the composite dietary antioxidant index and urinary phthalate concentrations Evidence from NHANES 2011-2018,
Yang, Y. J., Ding, P., Pan, J. C., Feng, Y. H., Xin, Z. C. and Wu, C. L., *Medicine*, Jul 10 2026, Vol. 105, no. 28.

The relationship between the Composite Dietary Antioxidant Index (CDAI) and urinary phthalate concentrations has yet to be explored. This study utilized data from the 2011-2018 NHANES database. Weighted multivariable linear regression models were employed to examine the relationship between CDAI and urinary phthalate concentrations. Subgroup analyses were conducted to explore differences in associations across various subgroups. Additionally, restricted cubic spline (RCS) analyses were performed to investigate potential nonlinear relationships between CDAI and urinary phthalate concentrations. In the multivariable linear regression model, fully adjusted for confounding variables, CDAI was significantly associated with lower concentrations of high-molecular-weight (HMW) phthalates, including MCOP (beta = -0.724, 95% CI: -1.454, -0.006) and MECP (beta = -0.297, 95% CI: -0.618, -0.091). MBzP also showed a significant negative association with CDAI (beta = -0.212, 95% CI: -0.353, -0.071). For low-molecular-weight (LMW) phthalates, there was a significant association between CDAI and lower MiBP concentrations (beta = -0.136, 95% CI: -0.318, -0.046). Subgroup analyses indicated a negative correlation between CDAI and urinary phthalate concentrations in participants aged 60 years and older, as well as in male participants. Weighted RCS analysis demonstrated a linear relationship between CDAI and urinary phthalate concentrations. CDAI negatively correlated with urinary phthalate concentrations; further research is needed for validation. https://doi.org/10.1097/md.0000000000049659

The Possible Association Between Bisphenol A (BPA) and the Neuropathological Processes Characteristic of Alzheimer's Disease—A Systematic Review of the Literature,
Żebrowska-Gamdzyk, M. and Maciejczyk, M., *International Journal of Molecular Sciences*, 2026, Vol. 27, no. 15, p. 7026.
https://doi.org/10.3390/ijms27157026

Endocrine-Disrupting Pesticides as Drivers of Human Disease: Mechanistic Toxicology and Life-Course Health Effects,
Zidan, N. E.-H., Alshaal, T., Elhawat, N., Elhamalawy, O., Malhat, F. and Eissa, F., *International Journal of Molecular Sciences*, 2026, Vol. 27, no. 15, p. 6928.
https://doi.org/10.3390/ijms27156928

Evaluation de l'exposition

The transfer of per- and polyfluoroalkyl substances (PFAS) from mother to child: Comparison between maternal and cord blood in an Italian cohort,
Fornasari, A., Sech, M., Piva, E., Morini, L., Vaiano, F., Seravalli, V., Seidenari, A., Lenzi, J., Simonazzi, G., Fais, P. and Pascali, J. P., *Journal of Pharmaceutical and Biomedical Analysis*, 2026/12/15/ 2026, Vol. 281, p. 117637.

Per- and polyfluoroalkyl substances (PFAS) are persistent synthetic pollutants associated with multisystemic toxicity, including endocrine disruption and developmental risks. While maternal-fetal transmission has been documented, significant gaps remain regarding the fetal burden of emerging and short-chain alternatives. This study quantified 23 PFAS in paired maternal and cord blood samples from 120 women recruited in Italy, between 2024 and 2025 during the ELENA project (Early Life Exposure to per- and polyfluoroalkyl substaNces (PFAS) and HeAlth risks). Analysis was

performed using LC-HRMS to evaluate the single and total PFAS burden after solid-phase extraction. PFAS were detected in 99.04% of maternal and 84.8% of cord blood specimens. Legacy compounds, PFOS (99.0%), PFOA (83.7%), and PFHxS (78.8%), were the most prevalent in maternal samples. Significant associations were found between the regular use of processed food and higher levels of maternal 6:2FTS and PFHpS. While the median sums of PFAS in maternal and cord blood were 1.19 ng/mL and 0.49 ng/mL respectively, nearly 30% of women and 3% of cord blood exceeded the 2 ng/mL NASEM threshold associated with potential health risks. In general, when comparing the observation frequencies and concentration of the compounds in paired samples, the kinetic barrier of placenta has been confirmed, with no selective accumulation. The calculated Transplacental Transfer Efficiency (TTE) values suggested that chemical structure significantly dictates fetal burden. These results highlight the urgent need for biomonitoring approaches and targeted strategies to reduce maternal exposure to legacy and emerging PFAS to protect fetal development.
https://doi.org/10.1016/j.jpba.2026.117637

Exposure to Endocrine-disrupting Chemicals, Iron Metabolism, and Risk of Metabolic Dysfunction-associated Steatotic Liver Disease: A Nationwide Cross-sectional Study in China, Li, Z. Y., Zhao, Y. B., Wu, Z. Y., Wang, H. J., Hu, Y. Z., Zhang, X. F., Kang, X., Su, C., Zhang, T. and Liu, A. D., *Biomedical and Environmental Sciences*, Jun 2026, Vol. 39, no. 6, p. 677–689.

Objective This study aimed to investigate the association between exposure to mixtures of environmental endocrine-disrupting chemicals (EDCs) and metabolic dysfunction-associated steatotic liver disease (MASLD) and to assess the potential mediating role of iron metabolism. Methods A total of 6,989 adults from the China Health and Nutrition Survey (2015 cycle) were included. The serum concentrations of 22 EDCs were measured. Logistic regression, weighted quantile sum (WQS) regression, and Bayesian kernel machine regression (BKMR) models were used to evaluate the association between EDC exposure and risk of MASLD. Mediation analyses were performed to assess the mediating role of serum ferritin (SF). Results Eight EDCs were positively associated with MASLD. The WQS regression model identified six major contributors, including beta-hexachlorocyclohexane, p,p'-DDT, monoethyl phthalate, acenaphthene, perfluorooctanoic acid, and perfluoro-n-pentanoic acid, in mixture effects. The BKMR model demonstrated that higher levels of EDC mixture were associated with an increased risk of MASLD. Subgroup analyses suggested stronger correlations in males and in individuals aged < 65 years. SF was estimated to mediate 11.2%-32.1% of the association between key EDCs and MASLD. Conclusion Exposure to EDC mixtures was associated with an increased risk of MASLD, with iron metabolism playing a notable mediating role. Reducing the exposure to key EDCs may help alleviate the burden of MASLD.
https://doi.org/10.3967/bes2026.049

PFAS internal exposure levels across two generations and endocrine-metabolic biomarker associations in women from the French E3N-Generations cohort, Mancini, F. R., Perrin, C., Billmann, R., Blanché, H., Frénoy, P., Ren, X., Petitjean, G., Pommereuil, R., Cano-Sancho, G., Le Bizec, B., Antignac, J.-P. and Severi, G., *Environment International*, 2026/09/01/ 2026, Vol. 215, p. 110462.

Background Per- and polyfluoroalkyl substances (PFAS) are environmentally persistent chemicals suspected to act as endocrine-active compounds, disrupting lipid metabolism and hormonal homeostasis, while possible multigenerational effects are an emerging concern. Although legacy PFAS such as perfluorooctane sulfonic acid (PFOS) and perfluorooctanoic acid (PFOA) have been restricted in France since 2009, the impact of these measures on long-term exposure remains unclear. Objectives We aimed to (1) compare serum PFAS concentrations across two generations of adult French women and identify determinants of exposure; (2) evaluate associations between PFAS

(individually and as mixtures) and early hormonal and metabolic biomarkers; and (3) explore potential intergenerational associations between maternal PFAS exposure and adult daughters' biomarkers. Methods We analyzed serum PFAS concentrations among two generations of women from the E3N-Generations cohort (1995–1999 and 2024). Biomarkers included thyroid-stimulating hormone (TSH), testosterone, sex hormone–binding globulin (SHBG), triglycerides, and total, HDL, and LDL cholesterol. Multivariable models assessed determinants of exposure and PFAS–biomarker associations, with exploratory analyses of maternal–daughter pairs. Results Total PFAS concentrations decreased substantially over time, particularly for PFOS and PFOA, although these compounds remained the predominant PFAS detected in both generations. Sociodemographic, anthropometric, and reproductive factors were associated with PFAS serum concentrations, with generation-specific patterns. The results showed a positive cumulative association between PFAS and testosterone and a negative association with TSH and HDL cholesterol, while associations with other lipid biomarkers were heterogeneous. No consistent associations were observed between maternal PFAS concentrations and adult daughters' biomarkers. Conclusions Despite regulatory-driven declines, PFAS exposure remains widespread and associated with endocrine and metabolic biomarkers in adult women. These findings are consistent with the need for strengthened, class-based PFAS regulation and dedicated studies to rigorously evaluate potential multigenerational associations. https://doi.org/10.1016/j.envint.2026.110462

Contribution ofConsumer Products to Emerging ChemicalExposures in a Preconception Cohort Study,
Zhou, J. Q., Lin, E. Z., Wesselink, A. K., Schildroth, S., Willis, M. D., Hatch, E. E., Wise, L. A. and Pollitt, K. J. G., *Environmental Science & Technology*, Jul 28 2026, Vol. 60, no. 29, p. 20197–20212.

In the United States (U.S.), 15% of couples attempting pregnancy experience infertility, and 20% of pregnancies end in miscarriage. Environmental chemical exposures, particularly endocrine-disrupting chemicals (EDCs) from consumer products, may contribute to reproductive health challenges. We mailed wearable passive samplers (Fresh Air wristbands) to 132 female participants across 39 U.S. states and 24 of their male partners in the Pregnancy Study Online (PRESTO) to characterize exposure among couples trying to conceive between January and October 2021. Wristbands were analyzed using thermal desorption gas chromatography-high-resolution mass spectrometry with targeted and nontargeted approaches. Nontargeted analysis revealed exposure to 491 chemicals, predominantly benzenoids (36.9%) and organoheterocyclic compounds (18.3%). Health hazard assessment identified 107 high-risk EDCs and 47 compounds predicted to have medium-to-high impacts on reproductive outcomes, including galaxolide, salicylates, and phthalates. Targeted analysis quantified 43 chemicals, revealing universal exposure to polycyclic aromatic hydrocarbons and phthalates. Lifestyle factors influenced exposures: frequent sunscreen users had up to 3.1 times higher ultraviolet filter exposures, while dog owners had 1.2-2.6 times higher exposure to fragrance compounds. Among cohabitating couples, few similarities were observed, despite shared environments. The findings demonstrate that consumer products contribute to complex exposure mixtures with reproductive health implications during preconception. https://doi.org/10.1021/acs.est.5c18677

Non-destructive extraction and characterization of human pulmonary microplastics and the associated endocrine disrupting chemicals,
Zhu, T., Zhang, X. Y., Qiu, M. T., Chen, K. Z. and Guo, X. J., *Environmental Pollution*, Oct 15 2026, Vol. 407.

Inhalation exposure to microplastics (MPs) and associated plastic additives has raised growing public health concerns, although their distribution and potential health impacts in human lungs

remain poorly understood. To minimize interference from lipids and collagen fibers, particulate matter (PM) was first extracted from lung tissues using a non-destructive ultrasonic treatment and sequential centrifugation (USC) method, followed by obtaining the microplastic extract (MPE) using ultrasound and density separation (UDS), and subsequent detection of EDCs via GC-MS. This approach enabled characterization of both pulmonary MPs and associated EDCs. Sixteen polymer types were identified, with nylon (27.3%), polyester (10.7%), and polyurethane (7.8%) being the most abundant, alongside 71 EDCs, including phthalic acid esters (PAEs), organophosphate esters (OPEs), and polycyclic aromatic hydrocarbons (PAHs). Smokers and individuals over 60 years old showed significantly higher pulmonary MP abundances ($P < 0.01$). EDC concentrations were higher in MPE than in surrounding lung tissues, particularly for PAEs, which showed elevated enrichment factor MPs (EFMs range: 16.4-101; mean: 50.1). The average concentration of pulmonary MPs, estimated via PAEs under an upper-bound scenario, was 7.35 mg/kg. The heterogeneous distribution of MPs and additives within pulmonary tissues may have implications for localized exposure, although the biological effects remain to be further investigated. These findings underscore the importance of characterizing MPs and associated contaminants to better assess potential health risks. https://doi.org/10.1016/j.envpol.2026.128705

Méthodes

Predicting the immunotoxic potential of chemical mixtures based on their endocrine activity profiles via computational modeling and in vitro bioassays,
Braun, G., Hilscherová, K., Van Brummelen, S., Pieters, R., Smutná, M., Wojtysiak, N. and Escher, B. I., *Environment International*, Aug 2026, Vol. 214.

Exposure to endocrine disrupting chemicals (EDCs) is a known threat to the environment and human health, because EDCs can interfere with many developmental processes. As immune cells rely on signaling pathways than can be impacted by EDCs, the disruption of endocrine processes exhibits the potential to directly or indirectly adversely alter immune response. We employed a high-throughput in silico workflow to identify EDCs that are likely to alter endocrine and metabolic processes with direct or indirect immunotoxic potential. Initially included 83,693 chemicals were reduced to a priority list of 6,620 and matched with blood concentrations as reported in human biomonitoring studies. Mixtures in representative concentration ratios were prepared for five main groups of EDCs with five chemicals per mixture, covering per-and polyfluorinated compounds, halogenated pesticides, phthalates, phenols, and polycyclic aromatic hydrocarbons, and one mixture containing all 25 chemicals. Single compounds and mixtures were tested in high-throughput bioassays covering potential indirect modulators of immune response such as endocrine signaling through activation of estrogen receptor, androgen receptor, glucocorticoid receptor and aryl hydrocarbon receptor, thyroid hormone signaling through agonism and antagonism on the thyroid hormone receptor, thyroperoxidase inhibition, and transthyretin binding, or more direct endpoints such as pro-inflammatory signaling through the activation of nuclear factor kappa-light-chainenhancer of activated B cells, as well as redox and metabolic imbalance via oxidative stress response and mitochondrial disruption. 19 out of 33 bioactive mixtures confirmed concentration additivity across all tested endpoints within 3-fold deviation, indicating a clear relevance of commonly detected EDCs mixtures as potential immunomodulators.
https://doi.org/10.1016/j.envint.2026.110413

High-throughput Virtual Screen of Endocrine-disrupting Chemicals Identifies Disruptors of EGFR Signaling,
Jesikiewicz, L. T., Marathe, R., Sepehri, B., Demissie, R., Lee, H., Veiga-Lopez, A. and Villegas, J. A., *bioRxiv*, 2026, p. 2026.08.05.743028.

Chemical exposures during pregnancy are linked to an increased risk of pregnancy complications that contribute significantly to maternal and infant morbidity and mortality and can lead to long term health consequences for both the mother and the offspring. The placenta, a central regulator of pregnancy health, is a direct target of environmental toxicants. Epidermal growth factor receptor (EGFR), highly expressed in the placenta, regulates proliferation, migration, invasion, fusion, and cellular bioenergetics. To identify compounds of environmental concern with potential for EGFR-disrupting activity, we optimized a high-throughput virtual screening protocol for the identification of EGFR inhibitors and achieved enrichment factors of EF1% = 10.09, EF5% = 3.86, and EF10% = 3.0 in a benchmarking dataset. We applied this protocol to screen the Collaborative Estrogen Receptor Activity Prediction Project database, finding that top-scoring compounds were enriched for aromatic and fused-ring chemical classes, including dyes. Kinase activity assays revealed that two out of thirteen selected compounds, Vat Red 32 and Reactive Red 136, inhibited EGFR kinase activity with micromolar IC50 values. Additionally, pose refinement with molecular dynamics simulations characterized the binding interactions of Reactive Red 136 within the EGFR kinase domain, and functional assays in HTR-8/SVneo placental trophoblast cells showed that Reactive Red 136, but not Vat Red 32, partially attenuated EGF-mediated cell migration despite both compounds inhibiting EGFR kinase activity. Together, this study has generated an enriched dataset of candidate environmental EGFR modulators, with experimental validation confirming enrichment for EGFR-disrupting activity among the selected compounds. These results provide a valuable resource for toxicological studies.Competing Interest StatementThe authors have declared no competing interest. https://doi.org/10.64898/2026.08.05.743028

High-throughput analysis method for the comprehensive assessment of per-and polyfluoroalkyl substances, bisphenols, organophosphate esters, phthalates and personal care product additives in indoor dust,
Li, J. F., Wen, D. F., Li, Q. S., Yang, M. M., Zhu, Y. Y., Lin, Z. Q., Li, M. H., Li, L., Lai, X. T., Zhu, Q. S., Sun, C. J. and Zhao, Z. H., *Journal of Chromatography A*, Sep 27 2026, Vol. 1785.

Endocrine disrupting chemicals (EDCs) widely exist in indoor environments and warrant close scrutiny due to their potentially adverse health impacts. However, comprehensive evaluation of their single and combined health effects is hampered by the lack of high-throughput detection methods. Therefore, we developed a rapid UHPLC-MS/MS method for simultaneous quantification of 41 EDCs across 5 categories in just 10 min. This streamlined protocol includes a simple ethyl acetate extraction of only 50 mg dust samples, followed by nitrogen-assisted drying and reconstitution in 90% acetonitrile, ensuring minimal processing time while maintaining robustness. All 41 EDCs exhibited good linearity (R-2 > 0.99) and recovery rates ranging from 70.22% to 129.86%. The method achieved LODs (limits of detection) of 0.0001-0.4747 ng/mL and LOQs (limits of quantification) of 0.0002-1.5823 ng/mL, demonstrating high sensitivity. Method validation and real dust sample analysis confirmed the reliability of this approach in assessing human exposure of indoor EDCs. This study established an efficient, high-throughput analytical strategy, providing a novel tool for future research on the health risks posed by complex EDCs mixtures in indoor environments. https://doi.org/10.1016/j.chroma.2026.467270

Per- and Polyfluoroalkyl Substances (PFAS) in Michigan: A Novel High-Throughput Method with Liquid Chromatography Tandem Mass Spectrometry (LC-MS/MS) Analysis of 39 PFAS Compounds in Human and Bovine Dried Blood Spots (DBS),
Morrison, J. M., Lockwood-O'brien, S. Y., Bielicki, C. A., Carmack, D. J., Feeley, J. R., Karrer, T. A. and Geiger, M. J., *Toxics*, 2026, Vol. 14, no. 9, p. 753.
https://doi.org/10.3390/toxics14090753

Validated SPE-HPLC-MS/MS analysis of steroid hormones in H295R cell culture medium: chromatographic separation and ESI response in the presence of potential endocrine-disrupting chemicals,
Sadutto, D., Manna, L., Tait, S., Coppola, L., Lori, G., D'ettorre, G. and Bossù, E., *Analytical and Bioanalytical Chemistry*, 2026.

A sensitive and robust SPE-HPLC-MS/MS method was developed and validated for the simultaneous quantification of five steroid hormones in H295R cell culture medium. Chromatographic separation of the target steroids and selected potential endocrine-disrupting chemicals (EDCs) was achieved using a ZORBAX Eclipse Plus C18 column (1.8 & micro;m, 95 & Aring;, 50 & times; 2.1 mm). Sample preparation was performed by solid-phase extraction employing hydrophilic-lipophilic balanced polymeric reversed-phase sorbent cartridges. Extraction recovery was evaluated at three concentration levels, tailored to each steroid, and exceeded 80% for all analytes. Intra- and inter-day precision, expressed as relative standard deviation (RSD), ranged from 1 to 12%. Method limits of quantification were in the range of 8 and 73 pg mL(-)1, enabling reliable detection of steroids present at low endogenous levels. In addition to method validation, the ESI response of steroids was systematically evaluated in the presence of the selected compounds. Under the reference chromatographic gradient, signal variations were generally limited, indicating stable and robust ionization performance during LC-ESI-MS/MS analysis. The validated method was successfully applied to the high-throughput analysis of 285 H295R cell culture medium samples, generating a dataset of 1425 individual hormone measurements obtained within a broader framework investigating steroidogenic responses to individual potential EDCs. The results confirmed the capability of the analytical method to reliably monitor hormone fluctuations across a broad concentration range, demonstrating its suitability for routine, large-scale sample analysis in complex in vitro systems. https://doi.org/10.1007/s00216-026-06688-8

Anogenital distance as a cross-species biomarker of fetal androgen action: Origins, translation, and implications for reproductive toxicology,
Svingen, T., *Reproductive Toxicology*, Sep 2026, Vol. 144.

Anogenital distance (AGD) is a dose-responsive biomarker of disrupted fetal androgen signalling. Because AGD remains relatively stable after birth, its measurement provides a retrospective indicator of fetal androgen action and has proven valuable in toxicity research, particularly for identifying chemical exposures with antiandrogenic properties. Although initially described as an anatomical trait for sexing neonatal animals, rodent toxicity studies in the 1990s established AGD as an androgen-sensitive endpoint following in utero exposure. Importantly, reduced AGD was consistently associated with a broader spectrum of male reproductive abnormalities, including cryptorchidism and hypospadias, collectively reflecting impaired masculinization during development. The identification of a distinct masculinization programming window (MPW) subsequently provided a mechanistic framework for interpreting AGD variation. Human studies have since confirmed that AGD captures aspects of prenatal endocrine disruption, including phthalate exposure, supporting its use as a translational biomarker linking experimental models, epidemiology, and regulatory toxicology. This review traces key developments in the conceptual

evolution of AGD, from a sexually dimorphic anatomical trait used for visual sex determination to a formalized biomarker applied in animal and human studies of endocrine disruption. https://doi.org/10.1016/j.reprotox.2026.109293

Mathematical model for thyroid hormone regulation in foetal rat brain, Thompson, C. V., Leedale, J. A., Charlton, A., Webb, S. D. and Currie, R., *Computational Toxicology*, Sep 2026, Vol. 39.

During early pregnancy, significant perturbations to thyroid hormone activity in the mammalian foetal brain can lead to adverse neurodevelopmental outcomes. Endocrine-disrupting chemicals have the potential to cause these perturbations by interfering with normal systemic thyroid hormone regulation. This represents a complex multi-scale problem involving both whole-body (systemic) and tissue-based (local) regulation of thyroid hormones. Understanding whether biological barriers such as the blood-brain barrier and localised transcriptional feedback systems can resist systemic changes in thyroid hormones is critical. We developed a novel mathematical model describing thyroid hormone regulation dynamics in the foetal rat brain and used it to predict the effects of three chemicals (propylthiouracil, sodium phenobarbital, and methimazole) on foetal brain thyroid hormone concentrations. The model predicted changes in foetal brain thyroid hormones within two-fold accuracy based on measured changes in systemic foetal thyroid hormone levels. The model also demonstrates that maternal (dam) systemic thyroid hormone levels can be used to predict these effects during gestation. Mathematical modelling of thyroid hormone regulation in the foetal brain can improve the prediction and understanding of the dynamic effects of endocrine-disrupting chemicals on neurodevelopment and support developmental neurotoxicity risk assessment. https://doi.org/10.1016/j.comtox.2026.100442

Adverse outcome pathway network-based approach for the evaluation of thyroid hormone system disruption for human health and environmental regulatory hazard assessment, Vergauwen, L., Tait, S., Bajard, L., Buławska, N., Gutleb, A. C., Hessel, E., Hilscherová, K., Holbech, H., Kowalska, D., Novák, J., Papaioannou, N., Renieri, E., Roncaglioni, A., Sarigiannis, D. A., Sosnowska, A., Van Den Brand, A., Jacobs, M. N. and Knapen, D., *Environment International*, 2026/09/01/ 2026, Vol. 215, p. 110451.

Thyroid hormones (THs) are essential for growth, development, and metabolism across vertebrates. Thyroid hormone system disruption (THSD), as one of the mechanisms of endocrine disruption (ED), is widely recognised as a critical concern for both human and environmental health. Important challenges remain in the availability and regulatory application of methods for THSD assessment. Here we discuss recent methodological advances in the area of THSD, including validation efforts for in vitro assays and THSD-sensitive endpoints as additions to existing Organisation for Economic Co-operation and Development (OECD) Test Guidelines using fish early-life stages. We propose a modular adverse outcome pathway (AOP) network-based framework integrating mechanistic and effect-level information to support regulatory hazard assessment for both human and environmental health under a One Health paradigm. The framework summarises essential elements of the THSD AOP network relevant to human and environmental health and methods were mapped to these mechanism and effect components. In a case study on resorcinol, a chemical of regulatory interest relevant for both human and environmental health, we demonstrate how emerging methods, including in vitro and fish eleutheroembryo (developmental stage between hatching and the onset of independent feeding) assays as new approach methods (NAMs), complement traditional data to strengthen weight-of-evidence for ED classification. By mapping gaps and priorities, this work advances the integration of NAMs into chemical safety assessment, supporting a future update of OECD Guidance Document 150. These efforts align with European Union

initiatives to gradually phase out animal testing for chemical hazard assessment and implement Next Generation Risk Assessment. https://doi.org/10.1016/j.envint.2026.110451

Agenda

Deux appels à projets de recherche pour mieux connaître les risques sanitaires liés à l'environnement et au travail,
(juillet 2026),
L'Anses lance ce 9 juillet 2026 deux appels à projets de recherche dans le cadre du Programme national de recherche environnement-santé-travail (PNR EST) : l'un généraliste, sur les thèmes santé-environnement et santé-travail, l'autre spécifique aux effets potentiels des radiofréquences sur la santé. L'enjeu : inciter les scientifiques à s'emparer de questions de recherche encore peu étudiées mais considérées comme d'importance majeure pour la santé humaine et les écosystèmes. Seront retenus en priorité les projets traitant de thématiques qui ont été identifiées comme prioritaires au regard des enjeux sanitaires et sociaux pour la population générale et dans le monde du travail. Il s'agit notamment : des substances chimiques, comme les perturbateurs endocriniens, les PFAS (per- et polyfluoroalkylés) et les produits phytopharmaceutiques, de la caractérisation de l'exposome, des micro et nanoplastiques, etc... Les scientifiques intéressés par les appels sont invités à soumettre des lettres d'intention présentant leurs projets d'ici le 22 septembre 2026 pour l'appel à projets général https://www.anses.fr/fr/content/deux-appels-projets-de-recherche-pour-mieux-connaitre-les-risques-sanitaires-lies-1

Webinaire « Pesticides : effets sur la santé de l'Homme et des écosystèmes »,
ANSES (24 septembre 2026),
À l'occasion des 20 ans du Programme national de recherche en Environnement Santé Travail (PNR EST), l'Anses organise un troisième rendez-vous d'une série de webinaires, consacré ce mois-ci aux pesticides et les effets sur la santé de l'Homme et des écosystèmes.
Ce webinaire, sur le thème « Pesticides : effets sur la santé de l'Homme et des écosystèmes », permettra de discuter de l'apport de ces recherches à l'évaluation des risques et à l'élaboration des politiques publiques.
Il se tiendra jeudi 24 septembre 2026 de 11h à 12h30.
https://survey.anses.fr/SurveyServer/s/DFRV/pesticides/questionnaire.htm
https://toute-la-veille-acteurs-sante.fr/245065/seminaire-pesticides-effets-sur-la-sante-de-lhomme-et-des-ecosystemes-en-ligne/

Webinar - Glyphosate Science: Cancer, neurotoxicity & endocrine disruption,
Collaborative for Health and Environment (CHE) (USA) (6 octobre 2026),
The Global Glyphosate Study, a multi-pronged effort bringing together a wide range of experts to address multiple health endpoints, has led to the publication of key research results on the carcinogenicity of glyphosate, as well as yielding information on other health endpoints. The Seattle Glyphosate Symposium, held in March 2026, culminated in the publication of a scientific Consensus Statement on the urgent need to address health harms of glyphosate. In the context of this scientific consensus, in June 2026 the US Supreme Court held that US states may not require warning labels about carcinogenicity of glyphosate because the US Environmental Protection Agency does not require such a warning. In this webinar, Dr. Daniele Mandrioli will discuss recent findings from the Global Glyphosate Study. Dr. Lianne Sheppard will discuss the findings presented at the March symposium, which was attended by 70 leading researchers from around the world. She will also share the science and recommendations highlighted in the Consensus Statement.
https://www.healthandenvironment.org/che-webinars/97164

Actualité, société, politique et évaluation du caractère PE

Avis de l'Agence nationale de sécurité sanitaire de l'alimentation, de l'environnement et du travail relatif aux travaux d'expertise réalisés en 2025 par l'Agence concernant l'évaluation et l'expertise des substances et produits cosmétiques régis par le règlement (CE) n°1223/2009
ANSES (18 juin 2026),
Depuis le 1er janvier 2024, l'Anses est en charge des missions de vigilance et d'expertise relatives aux produits cosmétiques et de tatouage (article L1313-1 du Code de la Santé Publique). Les missions d'expertise couvrent 3 axes :
• l'évaluation des dangers des substances et des risques associés à l'exposition aux produits utilisés, dans le cadre du règlement européen relatif aux produits cosmétiques et/ou des réglementations chimiques transversales (REACh et CLP),
• l'appui à la Direction générale de la concurrence, de la consommation, et de la répression des fraudes (DGCCRF) dans ses missions de surveillance du marché et de police sanitaire pour ces produits,
• le pilotage d'études d'exposition à ces produits et à leurs substances.
https://www.anses.fr/system/files/COSMETIQUES-2026-MPEX-0023.pdf

Endocrine Society and ESE Submit Joint Comments on Whether EDCs Should be Included Within Scope of Directive 2004/37/EC - Society Letters,
ENDOCRINE SOCIETY (juillet 2026),
The European Society of Endocrinology (ESE) and the Endocrine Society welcome the opportunity to contribute to ECHA's evaluation of whether endocrine-disrupting chemicals (EDCs) should be included within the scope of Directive 2004/37/EC on the protection of workers from risks related to exposure to carcinogens, mutagens or reprotoxic substances at work (CMRD).
https://www.endocrine.org/advocacy/society-letters/2026/edc-comments

Exposition au cadmium : pourquoi il faut s'inquiéter,
INSERM (31 aout 2026),
Si le cadmium a fait l'actualité récemment, sa présence dans nos aliments est bien connue depuis 2011. Dans un nouveau rapport, l'Agence nationale de sécurité sanitaire de l'alimentation, de l'environnement et du travail (Anses) alerte sur les niveaux alarmants d'imprégnation des Français à ce métal cancérogène, trois à quatre fois plus élevés par rapport aux autres Européens. Pourquoi sommes-nous plus exposés ? Quels sont les effets sur la santé, et comment s'en prémunir ? Trois experts répondent à nos questions. https://www.inserm.fr/actualite/exposition-au-cadmium-pourquoi-il-faut-sinquieter/

Les métiers de la beauté,
Travail et sécurité, mars 2026, no. 879, p. 12–25.
Coiffeurs, esthéticiennes, prothésistes ongulaires ou tatoueurs : les métiers de la beauté exposent à de nombreux risques professionnels souvent méconnus. Troubles musculosquelettiques, utilisation de produits chimiques, stations debout prolongées ou pression liée à la relation client… Dans un secteur dominé par les très petites entreprises, la prévention des risques au travail constitue un enjeu essentiel pour préserver la santé des professionnels et la pérennité des salons.
https://www.inrs.fr/media.html?refINRS=TS879page12

Nouvelle version de QSAR Toolbox disponible,
ECHA (2026),
QSAR Toolbox 4.9 est sorti en juillet. La dernière version inclut de nouvelles bases de données, des capacités de métabolisme améliorées, des outils de prédiction mis à jour et des flux de travail supplémentaires. Co-détenue par l'ECHA et l'OCDE, QSAR Toolbox est une ressource clé pour la toxicologie réglementaire. Il soutient des évaluations basées sur les données et contribue à réduire le besoin de tests sur les animaux.
https://qsartoolbox.org/wp-content/uploads/2026/07/Toolbox-4.9-Release-Notes.pdf
https://qsartoolbox.org/

Podcast - Bisphenols and the Thyroid,
ENDOCRINE SOCIETY (aout 2026),
What is BPA? Where is it found? And what do we know about the possible health risks regarding exposure to it? These questions and more are explored in depth in an ENDO 2026 abstract titled, "Bisphenol A, and Alternative Bisphenols S, F, Increase Proliferation and Growth of Thyroid Follicular Epithelial Cells and Disrupt Thyroid Histology."
https://endo2026.endocrine.org/fsPopup.asp?efp=RkVJT0NBS1UyNTg5OA&PresentationID=1830636&rnd=5.961686E-02&mode=presInfo
Host Aaron Lohr talks with the author of that abstract, Kailey Caroland, a cancer biology PhD candidate in the Weiss Lab at Vanderbilt University School of Medicine.
https://www.endocrine.org/podcast/enp115-bisphenol-a

Podcast - La SST expliquée - Les perturbateurs endocriniens *IRSST* (janvier 2026),
Qu'est-ce qu'un perturbateur endocrinien? Dans quels milieux de travail ces substances sont-elles présentes et quels effets peuvent-elles avoir sur le corps humain? Cet épisode répond à toutes ces questions et vous montre comment les milieux de travail mettent en place différents mécanismes pour réduire ou éliminer l'exposition à ces substances. Sabrina Gravel, professionnelle chercheuse et Pamela Prud'homme, professionnelle de recherche, nous éclairent.
https://rss.com/podcasts/lasstexpliquee/2455817/

Podcast - La SST expliquée - Les pesticides,
IRSST (janvier 2026),
Il existe de nombreux pesticides utilisés pour contrôler les nuisances, mais il est tout aussi essentiel de s'en protéger. Comment ces produits pénètrent-ils dans le corps humain et, surtout, quelles mesures concrètes peut-on mettre en place en milieu de travail pour réduire l'exposition ? Dans cet épisode Caroline Jolly, professionnelle chercheuse et Michèle Bouchard, professeure titulaire, Département de santé environnementale et santé au travail et titulaire de la Chaire d'analyse et de gestion des risques toxicologiques répondront à ces questions en vous présentant les principaux modes d'absorption et les bonnes pratiques de prévention.
https://rss.com/podcasts/lasstexpliquee/2455827/

Quand Annie Genevard, ministre de l'agriculture, invite BASF à faire blanchir l'un de ses herbicides,
Le Monde 4 juin 2026.
S'adressant à une assemblée de producteurs de blé, la ministre a encouragé le producteur d'un herbicide, classé perturbateur endocrinien, à présenter des « méthodes d'évaluation alternatives » pour obtenir une autorisation de mise sur le marché.
https://www.lemonde.fr/politique/article/2026/06/03/quand-annie-genevard-ministre-de-l-agriculture-invite-basf-a-faire-blanchir-l-un-de-ses-herbicides_6696665_823448.html?

Retour de l'acétamipride : « Le vote des sénatrices et sénateurs constituera un véritable test pour nos institutions démocratiques ». Tribune.,
Le Monde, 29 juin 2026.
Les représentants des principales associations de patients et des sociétés savantes médicales et scientifiques rappellent, dans une tribune au « Monde », l'état de la science sur la toxicité de l'acétamipride et du flupyradifurone, alors que le Sénat débat de la possible réintroduction de ces pesticides en France. https://www.lemonde.fr/idees/article/2026/06/29/retour-de-l-acetamipride-le-vote-des-senatrices-et-senateurs-constituera-un-veritable-test-pour-nos-institutions-democratiques_6716870_3232.html?search-type=classic&ise_click_rank=1

Targeting Epigenetic Dysregulation: Antioxidants as Countermeasures Against EDC-Induced Reproductive Toxicity,
Feng, Y., Chen, D. K., Wu, J. J., Peng, X. W. and Mei, S. Q., *Antioxidants*, Jun 2 2026, Vol. 15, no. 6.

Ubiquitous environmental endocrine-disrupting chemicals (EDCs), including bisphenols, phthalates, and heavy metals, pose a severe and persistent threat to mammalian reproductive health worldwide. Oxidative stress acts as the pivotal mediator which drives epigenetic dysregulation in germ cells upon EDC exposure, including aberrant DNA methylation, abnormal histone post-translational modifications and dysregulated non-coding RNA networks. EDC-induced oxidative stress damages endogenous antioxidant defense systems and inactivates key epigenetic regulators, forming a self-reinforcing cycle of redox imbalance and epigenetic dysregulation, which ultimately leads to impaired gametogenesis, reduced fertility, and transgenerational reproductive abnormalities. This review summarizes current evidence indicating that multiple antioxidants, including melatonin, vitamin C, resveratrol, and epigallocatechin gallate, alleviate EDC-induced reproductive toxicity by targeting epigenetic dysregulation. Their protective effects encompass scavenging excessive reactive oxygen species, activating endogenous antioxidant signaling cascades, restoring activity of epigenetic enzymes, and rectifying aberrant histone modification profiles, contributing to the maintenance of epigenetic homeostasis in germ cells. This review clarifies the intrinsic mechanistic link among EDC exposure, oxidative stress, epigenetic dysregulation and reproductive toxicity, which provides a theoretical basis for formulating reproductive health protection strategies against EDC exposure and guides the exploration of clinical epigenetic biomarkers. https://doi.org/10.3390/antiox15060704

Unmet Information Needs of Spanish Female Breast Cancer Survivors on Chemical Pollutants: A Cross-Sectional Mixed-Method Study,
García-Molina, L., Navarro-Matillas, B., Riquelme-Gallego, B., Molina-Fernández, M. Z., Expósito, J., Arrebola, J. P. and Martin-Olmedo, P., *Toxics*, May 23 2026, Vol. 14, no. 6.

Breast cancer (BC) remains the most prevalent malignancy among women. Prolonged exposure to chemical pollutants has been identified as one of its potential risk factors. Despite increased access to health information, important informational gaps persist regarding chemical exposure. This concern is particularly pronounced among female BC survivors due to their greater vulnerability. This study investigated unmet information needs related to chemical exposure among female BC survivors from Granada (Spain). The data were collected through 150 semi-structured interviews and focus groups between March 2023 and April 2024 and analyzed using the Framework Method. Most participants demonstrated limited awareness regarding BC and its potential association with chemical exposure. Nevertheless, 78.67% of them expressed concerns regarding the issue after diagnosis and anxiety surrounding the lack of reliable information from trusted sources on

recurrence prevention. These findings highlight the need for public health communication that informs and empowers protective behavior. https://doi.org/10.3390/toxics14060456

The global research landscape and frontiers of endocrine-disrupting chemicals-induced vascular toxicity: A bibliometric and visualization analysis,
Kang, T. Y., Zhang, F. Z., Zheng, J. H., Yong, X. and Ismail, N. A., *Eurasian Journal of Medicine and Oncology*, 2026 2026, Vol. 10, no. 3.

Introduction: Endocrine-disrupting chemicals (EDCs) have attracted increasing attention as potential contributors to vascular toxicity. Objective: This study aimed to characterize the global research landscape, knowledge structure, and emerging trends in this field. Methods: A total of 911 publications indexed in the Science Citation Index Expanded of the Web of Science Core Collection from 1997 to 2025 were analyzed using bibliometric and visualization methods to map collaboration patterns and knowledge evolution. Results: The United States leads in both publication output and citation impact. Knowledge mapping indicates that an "exposure-mechanism-endpoint" framework is widely adopted in the literature, with "oxidative stress" appearing as a highly connected keyword linking environmental exposures and vascular outcomes. Trend analysis further suggests a shift in research focus from traditional persistent organic pollutants to emerging contaminants associated with modern lifestyles, alongside an increasing focus on early-life exposures and susceptible populations. Conclusion: Research on EDC-related vascular toxicity is evolving toward greater emphasis on emerging contaminants, critical exposure windows, and mechanistic exploration. These trends may help inform future research directions and risk assessment strategies.
https://doi.org/10.36922/ejmo026050050

PFAS: challenges and scientific perspectives in human health risk assessment,
Kowalczyk, J., Abraham, K., August, C., Boeddinghaus, R. S., Chan, J. C. Y., Eckhardt, A., Fletcher, T., Freeling, F., Göckener, B., Halldórsson, T. I., Herzke, D., Hoogenboom, R. L. a. P., Jakobsson, K., Jung, C., Madia, F., Monien, B., Numata, J., Ramhøj, L., Schoeters, G., Sommerkorn, K., Trier, X., Tsiros, P., Unkelbach, C., Van Leeuwen, S. P. J. and Buhrke, T., *Archives of Toxicology*, 2026/08/25 2026.

To review recent advances in research on per- and polyfluoroalkyl substances (PFAS) and outline priority steps for risk assessment in consumer health protection, the German Federal Institute for Risk Assessment (BfR) organized the 'International PFAS Conference' in Berlin in October 2025. Building on the European Food Safety Authority's (EFSA) opinion in 2020, global research activities on PFAS have intensified. The conference was attended by 200 participants from 18 countries and covered topics such as analytical methods, human exposure, toxicokinetics, toxicity, and future perspectives. Given that there are more than 21,000 different PFAS in use, discussions highlighted the need for further data collection and a basis for prioritizing substances. Robust exposure assessment requires improved analytical methods combined with newly developed predictive tools to quantify, identify, and make better use of non-target data. Hazard characterization may benefit from the combined use of classic experimental data sets, epidemiological data, and new approach methodologies (NAMs) data. The participants emphasized the continuous need for refined data and proposed a systematic consolidation of global data on shared data platforms, accompanied by corresponding guidelines for data usage. In the final panel discussion, effective risk communication was identified as a critical challenge, necessitating clear and consistent messaging for the public and policymakers. The conference concluded with five key recommendations for future health risk assessments: prioritizing PFAS; improving analytical methods; promoting generation of robust data and data gap filling; promoting open science, including the development of shared data infrastructure and cross-institutional knowledge exchange; and strengthening risk communication.

These recommendations aim to support transparent, evidence-based and effective PFAS consumer health risk management in the decades ahead. https://doi.org/10.1007/s00204-026-04523-8

The Impact of Endocrine Disruptors in Cosmetic Products: A Systematic Review, Popa, F., Stanescu, C., Zavtoni, A. M. and Zavtoni, M., *Cosmetics*, Jun 2 2026, Vol. 13, no. 3.

Nowadays, technology is developing so rapidly that it implicitly drives economic growth by increasing the productivity of artificial products. Endocrine disruptors (EDs) are increasingly recognized as emerging contaminants in cosmetic formulations, raising concerns about their ability to alter hormonal homeostasis through long-term, low-dose exposure. The objective of this systematic review is to synthesize current evidence on the presence, mechanisms of action, and health effects of endocrine-disrupting chemicals commonly found in cosmetic formulations, as well as existing regulatory frameworks and gaps in consumer protection. A systematic search across major scientific databases for studies published between 2000 and March 2026 was conducted in accordance with PRISMA guidelines, yielding 102 eligible studies. The most commonly identified endocrine disruptors were parabens, phthalates, benzophenones, and triclosan. Evidence shows that these compounds can mimic or block endogenous hormones, disrupt estrogenic, androgenic, and thyroid pathways, and contribute to reproductive, metabolic, and developmental disturbances. Several studies reported bioaccumulation in human tissues and measurable internal exposure with higher vulnerability in pregnant women and adolescents. Current evidence supports a biologically plausible and clinically relevant association between exposure to cosmetic-derived endocrine disruptors and adverse health outcomes. Despite regulatory progress, major gaps persist in long-term exposure assessment, mixture toxicity, and cumulative real-world risk. Strengthening surveillance, harmonizing international regulations, and encouraging safer cosmetic formulations remain essential to reducing population exposure. Although regulatory measures have been introduced, comprehensive protocols for the use of endocrine disruptors in cosmetics are still under development; additional targeted research is required to refine health risk assessments and strengthen regulatory frameworks. https://doi.org/10.3390/cosmetics13030141

Toxicité sur les animaux

Polystyrene nanoplastics disrupt mouse placenta development in a sex-dependent manner, Alahmadi, H., Harbolic, A., De Oliveira-Cordova, C., Reynolds, R., Jojy, M., Potts, C., Doan, S., Mathur, T., Islam, M. S., Andrade, M. J., Smith, Q., Stapleton, P. A., Mitra, S. and Warner, G. R., *Reproductive Toxicology*, 2026/10/01/ 2026, Vol. 145, p. 109331.

Plastic production has been increasing exponentially. Throughout their lifespan, plastics degrade into smaller particles that accumulate in our bodies and the environment. Recent studies found these plastic particles can cross the placental barrier and reach the fetus. However, the impact of plastic particles on placental function is still unknown. We hypothesized that nanoplastics would disrupt placental growth and function, specifically focusing on transforming growth factor beta (TGFβ) signaling. To understand the impact of plastic particles on the placenta, we orally exposed pregnant CD-1 mice to 50 nm or 200 nm polystyrene plastic particles from gestation day 8 to day 15 at a human-relevant concentration of 5 mg/kg/day. After euthanasia on day 15, placenta and fetus weights were recorded, and tissues were prepared for histomorphology and gene expression analysis. We observed a statistically significant decrease in the area of the decidua in the placentas for the 200 nm treatment group and a borderline significant decrease in decidua area for the 50 nm treatment group compared to control. However, when separated by sex, only the male decidua were significantly decreased in the 200 nm group. Gene expression analysis of key signaling factors in the TGFβ pathway identified increased expression of Smad2 and Smad3, which may be

suppressing estrogen receptor signaling. Overall, both particle sizes disrupted placenta structure and signaling in a sex-dependent manner. https://doi.org/10.1016/j.reprotox.2026.109331

Acute exposure to di(2-ethylhexyl) phthalate or diisononyl phthalate leads to increased inflammation and oxidative stress in the uteri of mice,
Andrus, A. R., Parra-Forero, L. Y., Sexton, C. N., Liu, K. C., Rush, S. M., Laws, M. J., Flaws, J. A. and Nowak, R. A., *Reproductive Toxicology*, Sep 2026, Vol. 144.

Phthalates are a class of synthetic compounds, known as endocrine-disrupting chemicals, widely used as plasticizers in consumer products, including personal care items, medical devices, and food packaging. Two common phthalates, di(2-ethylhexyl) phthalate (DEHP) and diisononyl phthalate (DiNP), have been associated with adverse effects on female reproductive health. This study investigated the effects of acute DEHP and DiNP exposure on uterine inflammation and oxidative stress in adult female CD-1 mice. Mice were orally dosed for 10 days with vehicle control, DEHP (20 & micro;g/kg/day, 200 & micro;g/kg/day, or 200 mg/kg/day), or DiNP (20 & micro;g/kg/day, 100 & micro;g/kg/day, or 200 mg/kg/day). Uteri were collected during diestrus for histological and gene expression analyses. Quantitative PCR (qPCR) showed that DEHP (20 and 200 & micro;g/kg/day) and DiNP (200 mg/kg/day) increased expression of inflammasome-related genes (Il18, Il1 beta, and Nlrp3). DiNP at 200 mg/kg/day also increased Il10 expression. Oxidative stress genes revealed DEHP increased Prdx2 expression at all doses without affecting Sod1, Cat, or Gpx1. However, DiNP increased Prdx2 at 20 & micro;g/kg/day but reduced Sod1, Cat, and Gpx1 at higher doses. Histological analysis revealed that high-dose DiNP reduced outer myometrium thickness and luminal epithelial cell height, while DEHP only affected the cell height at the highest dose. Macrophage and other mononuclear phagocytic cell infiltration increased with DEHP (20 and 200 & micro;g/kg/day) and all doses of DiNP. while cell proliferation was only changed in DEHP (200 & micro;g/kg/day). Together, these findings demonstrate that acute exposure to DEHP and DiNP induces uterine inflammatory and oxidative stress responses, with distinct dose-dependent effects for each phthalate. https://doi.org/10.1016/j.reprotox.2026.109306

Assessing Metabolic and Reproductive Consequences of Prenatal Paracetamol Exposure in Female CD1 Mice,
Entezari, B., Bozdag, D., Ince-Erguc, E., Buhur, A., Tomruk, C. S., Sabuncuoglu, S., Yavasoglu, A. and Gurer-Orhan, H., *Journal of Applied Toxicology*, 2026.

Paracetamol is a widely used analgesic during pregnancy; however, emerging evidence suggests it may act as an endocrine/metabolism disrupting compound. Using an F1 female CD1 mouse model, this study scrutinized the long-term metabolic and reproductive consequences linked to intrauterine paracetamol exposure. Pregnant dams were administered paracetamol at three dose levels (Cmax/10, Cmax, and Cmax & times; 10) during a critical window of organogenesis (gestational days 7.5-16.5). Offspring were monitored until 17 weeks of age to assess growth, glucose homeostasis, lipidomic profiles, and ovarian histology. Prenatal exposure did not significantly affect total body weight but induced a dose-dependent increase in peritoneal adipose tissue mass, particularly in the Cmax & times; 10 group. Metabolic assessments revealed impaired glucose tolerance in the Cmax/10 group and increased insulin sensitivity across all dose groups. Adipose tissue gene expression analysis demonstrated upregulation of adipogenic markers (Ppar gamma, Lpl, Fasn), while untargeted lipidomics revealed consistent dysregulation of lipid species, including lysophosphatidylcholines (LPCs), phosphatidylcholines (PCs), sphingomyelins (SMs), and triacylglycerols (TGs), indicating altered lipid storage and inflammatory signaling. Histological evaluation showed a marked reduction in total follicle number and increased follicular atresia following paracetamol exposure. Degenerative changes in oocytes and granulosa cells were

observed, and multi-oocyte follicles (MOFs) were detected exclusively in the Cmax/10. Collectively, these findings demonstrate that intrauterine paracetamol exposure induces persistent developmental reprogramming in female offspring, characterized by visceral adiposity, disrupted lipid homeostasis, and depletion of the ovarian reserve. Notably, several endpoints exhibited non-monotonic dose-response relationships, underscoring the sensitivity of developing systems to even subtherapeutic paracetamol exposure. https://doi.org/10.1002/jat.70367

Seminal Vesicle Abnormalities and Exploratory miR-664-5p and FOXO Findings in Aged Mice Following Long-Term Butyl Benzyl Phthalate Exposure,
Hwang, S., Kang, H. B., Kim, D. H., Kim, H. H. and Park, M. H., *Antioxidants*, 2026, Vol. 15, no. 8, p. 1021.
https://doi.org/10.3390/antiox15081021

Enduring autism-like phenotypes and deregulated hypothalamic prosocial peptides after early-life exposure to indoor flame retardants in male C57BL/6 mice,
Kozlova, E. V., Gonzalez, G. M., Denys, M. E., Bishay, A. E., Gutierrez, R., Reid, J., Krum, J. M., Lampel, G., Luvsanravdan, N., Rabbani, K. M., Tu, J. R., Campoy, L., Anchondo, L. M., Luna, C. N., Olomi, D. S., Monarrez, E., Carrillo, V., Tran, J. D., Platt, D., Korde, Y., Chinthirla, B. D., Blaibel, M., Kim, S., Chompre, G., Phillips, A. L., Stapleton, H. M., Henkelmann, B., Schramm, K. and Curras-Collazo, M. C., *Journal of Neuroendocrinology*, Aug 5 2026, Vol. 38, no. 8.

Polybrominated diphenyl ethers (PBDEs) are neuroendocrine-disrupting chemicals that produce adverse neurodevelopmental effects. PBDEs have been implicated as risk factors for autism spectrum disorder (ASD), which is characterized by abnormal psychosocial functioning and is commonly comorbid with cognitive deficits and sensory abnormalities. Here, we used a mouse model with translationally relevant exposure to establish direct causal evidence that maternal transfer of a commercial mixture of PBDEs, DE-71, produces ASD-relevant behavioral and neuroendocrine deficits in male offspring. C57Bl6/N mouse dams were exposed to a commercial PBDE mixture, DE-71, via oral administration of 0 (vehicle control, VEH/CON), 0.1 (L-DE-71), or 0.4 (H-DE-71) mg/kg b.w./day for 10 weeks, spanning from 3 weeks prior to gestation through the end of lactation at postnatal day (PND) 21. Mass spectrometric analysis indicated a dose-dependent transfer of PBDEs (in ppb) to brains of F1 male offspring at PND 30, with reduction in levels by PND 110. DE-71-exposed adult F1 male offspring displayed ASD-relevant abnormal neurobehavioral phenotypes, including impaired social novelty preference (SNP) despite intact general sociability and exaggerated repetitive behavior. There were also milder effects on long-term social recognition memory (SRM). DE-71-exposed mice also displayed altered olfactory discrimination of social odors without altered odor preference for non-social odors, impaired novel object recognition memory, and reduced open field habituation relative to VEH/CON. However, no changes were observed in sensorimotor, anxiety-, or depressive-like behaviors. At the molecular level, DE-71-exposed males displayed deregulated gene markers of prosocial neuropeptides, oxytocin, vasopressin, and PACAP systems in hypothalamic and forebrain regions. Oxt was upregulated in the paraventricular nucleus (PVN); Avp was upregulated in the PVN and bed nucleus of the stria terminalis (BNST) but downregulated in the lateral septum (LS); Avp1ar and Adcyap1 were upregulated in the BNST; and Adcyap1r1 was upregulated in the PVN, supraoptic nucleus (SON), and BNST. Peripheral OXT was increased in L-DE-71 males, while no changes in plasma AVP were observed. These findings demonstrate that developmental PBDE exposure produces enduring behavioral and neuroendocrine phenotypes that resemble core domains of ASD, which may result from early neurodevelopmental reprogramming within central social and memory networks. https://doi.org/10.1111/jne.70237

Gene-environment interaction in hypospadias: DEHP exposure aggravates DNAH8-related disruption of testicular steroidogenesis,
Li, Y., Huang, J. C., Peng, Z. W., Yu, M. M., Yin, X. Q., Li, J. Y., Guo, C. M., Wang, Y. P., Huang, Y. C., Chen, F., Lyu, Y. and Ding, Y., *Ecotoxicology and Environmental Safety*, Sep 1 2026, Vol. 322.

Hypospadias is a prevalent congenital malformation of the male external genitalia with ill-defined pathogenesis. The rising incidence of isolated cases cannot be explained by genetic factors alone, and its multifactorial etiology suggests that genetic susceptibility is often exacerbated by environmental factors, aligning with 'two-hit' hypothesis. Here, we investigated the combined effects of the hypospadias risk gene DNAH8 and prenatal exposure to the endocrine-disrupting chemical di-(2-ethylhexyl) phthalate (DEHP). Fetal wild-type and Dnah8-knockout (KO) mice were exposed to DEHP (0, 250, or 500 mg/kg/day) to evaluate urethral morphology, androgen dynamics, and testicular phenotypes. Dnah8 KO fetuses exposed to low-dose DEHP (250 mg/kg/day) exposure exhibited aggravated urethral fusion defects at E16.5. By E18.5, this co-exposure abolished the compensatory developmental mechanisms typically observed in Dnah8 KO mice, resulting in a 35.0% incidence of hypospadias, comparable to the 36.6% incidence induced by high-dose DEHP (500 mg/kg/day) alone. This combined exposure induced persistent testicular steroidogenic dysfunction, characterized by declining testosterone levels and significant reductions in Sertoli and fetal Leydig cells (FLCs). Proteomic analysis confirmed marked disruptions in oxidative stress and steroid biosynthesis pathways. Mechanistically, DEHP and its active metabolite, mono-(2-ethylhexyl) phthalate, induced oxidative stress, reduced cell viability, and inhibited the migration of mesenchymal progenitor cells (MPCs), compounding DNAH8 deficiency-induced MPCs differentiation delay. This impaired MPC trajectory led to deficient FLCs, irreversible disruption of testosterone synthesis, and hypospadias. These findings elucidate a novel DNAH8-DEHP interaction, establish a physiologically relevant in vivo model, and highlight early oxidative stress in progenitor cells as a critical node in hypospadias pathogenesis. https://doi.org/10.1016/j.ecoenv.2026.120531

Involvement of HOXA3 and chaperone-mediated autophagy in DEHP-induced ferroptosis of mouse Leydig cells via regulating GPX4 expression,
Liu, Y., Guan, C., Xu, L., Yang, J., Wang, J., Yang, S. and Chen, J., *Cell Death & Disease*, 2026/08/24 2026.

Impaired Leydig cell function leads to low testosterone levels, which is associated with male reproductive developmental defects and functional disorders. Exposure to di(2-ethylhexyl) phthalate (DEHP), an endocrine-disrupting chemical, can cause damage to Leydig cells, yet the underlying mechanism remains unclear. In this study, we found that DEHP exposure decreased serum testosterone levels and triggered ferroptosis of mouse Leydig cells by inhibiting GPX4 expression. Subsequently, mono(2-ethylhexyl) phthalate (MEHP), the primary active metabolite of DEHP, was shown to suppress the transcription of Gpx4 gene in TM3 cells by downregulation of HOXA3, which bound to the −264 to −258 region (RE3) of Gpx4 gene promoter. Concurrently, MEHP promoted HSC70 to recognize and degradate GPX4 protein via chaperone-mediated autophagy (CMA). Furthermore, both overexpression of HOXA3 and inhibition of CMA significantly abrogated MEHP-induced ferroptosis of TM3 cells through upregulating the expression of GPX4. Ultimately, tannic acid (TA), a natural polyphenol, was identified to increase the expression of GPX4 and inhibit MEHP-induced ferroptosis of TM3 cells, thereby alleviating DEHP-caused testicular damage in mice through the upregulation of HOXA3 and inhibition of CMA. https://doi.org/10.1038/s41419-026-09214-x

Bisphenol S induces hepatic steatosis and insulin resistance through AMPK inhibition and endoplasmic reticulum stress,
Sepúlveda-Fragoso, V., Barreto-Reis, E., Carvalho-Laureano, T. D., Stockler-Pinto, M. B., Frantz, E. D., De Carvalho, D. P., Palomer, X., Barroso, E., Vázquez-Carrera, M., Miranda-Alves, L. and Magliano, D. C., *Food and Chemical Toxicology*, Sep 2026, Vol. 215.

Bisphenol S (BPS), a common substitute for bisphenol A, has emerged as a widespread environmental contaminant, yet its metabolic effects remain poorly understood. Increasing evidence links BPS exposure to metabolic dysfunction-associated steatotic liver disease (MASLD), although the underlying mechanisms are unclear. Here, we investigated the metabolic effects of chronic BPS exposure (4, 25, and 50 mu g/kg/day for 12 weeks) in C57BL/6 mice and evaluated its impact on human Huh-7 hepatocytes. In vivo, exposure to 25 mu g/kg/day exhibited obesogenic potential, while all doses induced insulin resistance and promoted hepatic micro- and macrovesicular steatosis. BPS triggered endoplasmic reticulum (ER) stress and suppressed AMP-activated protein kinase (AMPK) phosphorylation, disrupting hepatic lipid and glucose metabolism. Consistently, BPS exposure in Huh-7 cells induced ER stress, reduced AMPK activity, and impaired insulin-stimulated Akt phosphorylation, indicating decreased insulin sensitivity. Notably, pharmacological activation of AMPK attenuated BPS-induced inhibition of insulin signaling, identifying AMPK as a key mediator of BPS-driven metabolic dysfunction in hepatocytes. Collectively, these findings demonstrate that BPS promotes hepatic steatosis and insulin resistance by activating ER stress and inhibiting AMPK, highlighting BPS as a potential environmental contributor to MASLD and related metabolic disorders. https://doi.org/10.1016/j.fct.2026.116222

Paternal DEHP exposure causes male offspring glycometabolism disorders via sperm miR-10a-5p,
Sun, J., Shan, D., Wang, J., Di, Q. and Xu, Q., *Ecotoxicology and Environmental Safety*, 2026/09/01/ 2026, Vol. 322, p. 120594.

Di (2-ethylhexyl) phthalate (DEHP) is an endocrine disruptor commonly present in environment, with metabolic disturbances and reproductive toxicity. DEHP remains one of the most widely utilized plasticizers, is still consumed in large quantities with limited prospects for complete replacement in short term. Consequently, human population is inevitably at risk of DEHP exposure. Previous studies have revealed the adverse effects of maternal perinatal DEHP exposure on offspring glucose homeostasis, however, whether paternal pre-pregnancy DEHP exposure disrupts offspring glucose metabolism through epigenetic inheritance remains unclear. In this study, we aimed to investigate the transgenerational epigenetic mechanism of abnormal glycometabolism in paternal DEHP exposed offspring, and to identify potential intervention targets. Here, male rats are administrated DEHP (0.1, 0.5 and 1.0 mg/kg body weight/d) by gavage for 15 weeks and then mated with females to produce offspring. This study finds that paternal DEHP exposure impaired glycometabolism in adult male offspring across generations. Mechanistically, paternal DEHP exposure down-regulated miR-10a-5p expression in the F0 generation sperm and offspring pancreatic islets, then led to Klf11-dependent β cell dysfunction and insulin synthesis reduction. Further evidence confirmed that miR-10a-5p bound Klf11 mRNA 3'-untranslated region (3'- UTR) and repressed its expression. Therefore, DEHP induced miR-10a-5p reduction resulted in Klf11 disinhibition, which functions as a transcriptional suppressor of Kit and Ins2 genes expression, eventually exacerbating glycometabolism disorders. Ultimately, our findings reveal that long-term DEHP exposure induced glycometabolism disorders exhibits transgenerational genetic characteristics and identify miR-10a-5p/Klf11 axis as a potential molecular target to mitigate offspring metabolic risk associated with paternal environment exposures. https://doi.org/10.1016/j.ecoenv.2026.120594

Chronic Di(2-ethylhexyl) phthalate and diisononyl phthalate exposure impairs uterine endometrial glycogen metabolism in mice,
Zarei, P., Raneri, I., Thapa, G., Andrus, A., Ibrahim, S., Laws, M. J., Flaws, J. A., Nowak, R. A. and Dean, M., *Reproductive Toxicology*, 2026/10/01/ 2026, Vol. 145, p. 109337.

Phthalates are associated with several reproductive disorders in women and reduce fertility in mice. They are also known to impair hepatic glycogen metabolism. Glucose is a crucial nutrient for the uterus, and glycogen buffers glucose concentration in the endometrium. The objective of this study was to investigate how long-term exposure to di(2-ethylhexyl) phthalate (DEHP) and diisononyl phthalate (DiNP) alters glycogen metabolism in the murine endometrium. Six-week-old female mice were fed chow containing vehicle or DEHP or DiNP at 0.15, 1.5, and 1500 parts per million (ppm) ad libitum for 9 months. Uteri were collected at diestrus. DEHP significantly reduced glycogen levels in the glandular epithelium (GE) and luminal epithelium (LE). In the stroma, both 1.5 and 1500 ppm groups had significantly lower glycogen. In the DiNP-treated mice, all three concentrations significantly decreased glycogen in GE, LE, and stroma. Neither phthalate altered mRNA levels of hexokinase1 (Hk1), glycogen synthase 1 (Gys1), glycogen phosphorylase M (Pygm), or glucose-6-phosphatase 3 (G6pc3). Immunohistochemistry showed that both phthalates increased HK1 levels in the stroma but not the epithelium. DEHP and DiNP (1500 ppm) increased PYGM in GE, LE, and stroma. DiNP (1500 ppm) significantly lowered G6PC3 in LE compared to all other groups. In the GE, both 1.5 and 1500 ppm DiNP decreased the immunostaining of G6PC3 compared to control and 0.15 ppm DiNP. Our results show that phthalates alter endometrial glycogen levels and expression of key enzymes. These findings are consistent with altered glycogen metabolism, which could alter endometrial glucose metabolism. https://doi.org/10.1016/j.reprotox.2026.109337