

CHEMWATCH

# TECHNICAL

Week of 8 May 2026

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TECHNICAL

# CONTENTS

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## TECHNICAL

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### OCCUPATIONAL

Effect of pesticide exposure on liver function tests and serum cholinesterase levels among floriculture industry workers in Bahirdar, Ethiopia: a comparative cross-sectional study

Assessment of Early Nephrotoxicity in Workers Exposed to Trichloroethylene Using NGAL and KIM-1 Biomarkers

### CHEMICAL EFFECTS

Prenatal Exposure to Parabens: Associations with Reproductive Hormone Alterations Prenatal Paraben Exposure and Reproductive Hormones

In utero and childhood exposure to organochlorines and perfluorinated chemicals in relation to sperm aneuploidy in adulthood

### ENVIRONMENTAL RESEARCH

Potentially toxic elements in surface waters across the El Fuerte River, Mexico: chemical speciation, environmental and health risk assessment

Environmentally relevant concentrations of nanoplastic increase the accumulation and endocrine effects of BDE47 and its metabolite 6-OH-BDE-47 in male frogs' testes

Transformation and transport: Polyvinyl chloride microplastics modulate fipronil accumulation and toxicity in zebrafish

### PHARMACEUTICAL/TOXICOLOGY

Inorganic arsenic and its methylated metabolites induce pulmonary immunosuppression via the p62-Keap1-Nrf2 positive feedback loop-mediated M2 macrophage polarization

Short-term exposure to a mixture of tributyltin plus cadmium disrupts the female reproductive organs, inducing ovarian follicular depletion and metabolic offspring outcomes in rats

High-lipid diets exacerbate copper-induced hepatointestinal toxicity in yellow catfish (*Pelteobagrus fulvidraco*) via gut microbiota dysbiosis

# TECHNICAL

## OCCUPATIONAL

### Effect of pesticide exposure on liver function tests and serum cholinesterase levels among floriculture industry workers in Bahirdar, Ethiopia: a comparative cross-sectional study

Scientific reports 2026 May 03 · 3 May 2026

Workers in the floriculture industry are exposed to various classes of pesticides through skin contact, inhalation, and ingestion. Although many pesticides are known to exert hepatotoxic effects and inhibit serum butyrylcholinesterase (BChE), no prior studies have been conducted in Ethiopia, particularly in the Amhara region. This study aimed to assess the effects of pesticide exposure on liver function tests and BChE among floriculture workers in Bahir Dar, Ethiopia. A comparative cross-sectional study was conducted from February 8, 2025, to May 28, 2025, including 103 floriculture workers and 51 community controls from Zege town. Sociodemographic and clinical data were collected using structured questionnaires, and venous blood samples were analyzed using the Cobas 6000 analyzer. Data were entered in Epidata 4.7 and analyzed in SPSS version 27. Statistical analyses included chi-square/Fisher's exact tests, t-tests, ANCOVA, one-way ANOVA, and multivariate logistic regression, with significance set at  $p < 0.05$ . Floriculture workers had significantly higher ALT ( $27.71 \pm 0.44$  U/L vs.  $22.71 \pm 0.47$  U/L; MD = 4.99 [95% CI: 3.72, 6.25];  $p < 0.001$ ), AST ( $28.46 \pm 0.68$  U/L vs.  $20.69 \pm 0.73$ ; MD = 7.77 [95% CI: 5.98, 9.74];  $P < 0.001$ ), TBL ( $0.831 \pm 0.028$  mg/dL vs.  $0.362 \pm 0.030$  mg/dL; MD = 0.47 [95% CI: 0.39, 0.55];  $P < 0.001$ ), and DBL ( $0.298 \pm 0.012$  mg/dL vs.  $0.170 \pm 0.021$  mg/dL; MD = 0.13 [95% CI: 0.09, 0.17];  $p < 0.001$ ) compared with controls. Conversely, total protein ( $7.385 \pm 0.058$  g/dL vs.  $7.624 \pm 0.098$  g/dL; MD = -0.24 [95% CI: -0.46, -0.01];  $p = 0.038$ ), albumin ( $4.255 \pm 0.052$  g/dL vs.  $4.623 \pm 0.088$  g/dL; MD = -0.37 [95% CI: -0.57, -0.17];  $p < 0.001$ ), A/G ratio ( $1.394 \pm 0.040$  vs.  $1.580 \pm 0.067$ ; MD = -0.19 [95% CI: -0.34, -0.03];  $p = 0.018$ ), and BChE ( $5105 \pm 141$  U/L vs.  $6699 \pm 239$  U/L; MD = -1594 [95% CI: -2142.66, -1045.39];  $p < 0.001$ ) were significantly lower among floriculture workers compared to control. Workers with more than 10 years of exposure showed markedly elevated ALT, AST, TBL, DBL, and globulin, while reduced albumin, A/G ratio, and BChE with < 5 years and 5-10 years ( $p < 0.05$ ). Overall, prolonged pesticide exposure was associated with significant alterations of liver function and BChE among floriculture workers.

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### Assessment of Early Nephrotoxicity in Workers Exposed to Trichloroethylene Using NGAL and KIM-1 Biomarkers

Journal of applied toxicology : JAT 2026 May 02 · 2 May 2026

This study evaluated early renal effects of chronic occupational exposure to mixed organic solvents among automotive spray painters by integrating urinary trichloroacetic acid (TCA) as an internal exposure marker with serum neutrophil gelatinase-associated lipocalin (NGAL) and kidney injury molecule-1 (KIM-1) as early tubular injury biomarkers. In this cross-sectional comparison of exposed workers and controls, urinary TCA was quantified as a surrogate of trichloroethylene metabolism, and serum NGAL and KIM-1 were measured alongside routine biochemical indices, creatinine, and estimated glomerular filtration rate (eGFR). Exposed workers showed significantly elevated NGAL and KIM-1 despite within limits creatinine and eGFR. TCA demonstrated strong linear associations with NGAL and KIM-1, while other solvent metabolites showed weaker relationships. Findings indicate that chronic solvent exposure produces measurable subclinical tubular injury, underscoring the limitations of creatinine-based monitoring and supporting the incorporation of NGAL and KIM-1 into occupational health surveillance.

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## CHEMICAL EFFECTS

### Prenatal Exposure to Parabens: Associations with Reproductive Hormone Alterations Prenatal Paraben Exposure and Reproductive Hormones

Annales d'endocrinologie 2026 May 01 · 1 May 2026

**BACKGROUND:** Parabens are ubiquitous in the environment due to their extensive use in food, personal care products and pharmaceuticals. Owing to their estrogenic properties, they are classified as suspected endocrine-disrupting chemicals.

**MATERIAL AND METHODS:** A total of 154 pregnant women living in Algiers were recruited. Umbilical cord blood samples were collected immediately after delivery. Reproductive hormones (LH, FSH, testosterone, progesterone, estradiol and prolactin) were quantified by electrochemiluminescence. Methylparaben (MP), ethylparaben (EP), and propylparaben (PP) were measured using liquid chromatography coupled with tandem mass spectrometry.

**RESULTS:** MP, EP and PP were detected in 88.3%, 74.0% and 85.1% of samples, respectively, with mean concentrations of  $1,420 \pm 1,085$ ,  $1,193 \pm 1,196$ , and  $1,402 \pm 1,308$  ng/mL. Significant negative associations were found between MP and both FSH ( $\beta = -0.051$ ) and estradiol ( $\beta = -1173.862$ ). MP and PP concentrations were inversely associated with testosterone level ( $\beta = -0.248$  and  $\beta = -0.239$ , respectively). EP concentrations showed a significant negative correlation with prolactin ( $\beta = -854.906$ ). Sex-stratified analyses revealed distinct patterns. In male neonates, MP was inversely associated with FSH and EP was negatively associated with prolactin. In female neonates, MP was inversely associated with estradiol and testosterone, while PP was also negatively associated with testosterone. No significant associations were observed between parabens and luteinizing hormone or progesterone in either sex.

**CONCLUSION:** Prenatal exposure to parabens is associated with alterations in reproductive hormone levels, which may have adverse consequences for newborn health.

**OBJECTIVES:** This study aimed to characterize paraben exposure profiles in umbilical cord blood and to evaluate associations between paraben concentration and reproductive hormone level in pregnant women.

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## In utero and childhood exposure to organochlorines and perfluorinated chemicals in relation to sperm aneuploidy in adulthood

Environmental health : a global access science source 2026 May 02 · 2 May 2026

**BACKGROUND:** Sperm chromosomal abnormalities are linked to infertility and may be caused by endocrine disrupting chemical exposures during development.

**METHODS:** Aneuploidy was determined in semen samples obtained from 96 Faroese young men aged 22-24 years who were members of a birth cohort created in 1986-1987. Their current and previous serum as well as cord blood were analyzed for DDE, major PCB congeners (118, 138, 153, and 180), and PFAS (PFOA, PFOS, PFNA, PFDA, and PFHxS). Incidence rate ratios between the exposures and the risk of an extra sex chromosome in adult sperm were assessed as indication of meiotic errors. The mixture effect for overall exposures (PCBs and/or PFASs) was estimated as the change in the percentage of each type of disomy for a doubling of the exposures for two individuals within the same smoking status and abstinence time group.

**RESULTS:** Higher concentrations of organochlorines in cord blood and in serum at ages 7, 14 years and 22 years were associated with increased proportions of chromosomal disomies. The PCB concentration in cord blood was associated mainly with having an extra Y chromosome (p-value: 0.006), while PFAS concentrations at adulthood were consistently associated with XX18 and YY18 disomies (p-values < 0.05).

**DISCUSSION:** These findings provide new evidence that fetal and subsequent chemical exposures can have enduring influence into adulthood on the formation of male germ cells.

**OBJECTIVES:** We examined whether exposure to organochlorine compounds (OC), including polychlorinated compounds (PCBs), and perfluorinated compounds (PFASs) measured repeatedly since birth predicted sperm chromosomal abnormalities in young adulthood.

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### ENVIRONMENTAL RESEARCH

## Potentially toxic elements in surface waters across the El Fuerte River, Mexico: chemical speciation, environmental and health risk assessment

Environmental pollution (Barking, Essex : 1987) 2026 Apr 30 · 30 Apr 2026

El Fuerte River (EFR) is one of Mexico's main rivers, and its basin supports agricultural, mining, industrial, and municipal activities that have been associated with contamination by potentially toxic elements (PTEs). This study evaluated As, Pb, Cd, Cu, Zn, Fe, and Mn along the EFR during the dry and rainy seasons. Pollution risk of PTEs was assessed using the heavy metal pollution index (HPI), heavy metal evaluation index (HEI), and health risk indicators (hazard quotient (HQ), hazard index (HI), and total carcinogenic risk (TCR)). This novel methodological framework provides a detailed understanding of pollution dynamics and risks in a subtropical river that has not been assessed previously. For both seasons, concentrations ( $\mu\text{g L}^{-1}$ ) were: 2.11-24.44 (As), <DL-0.62 (Pb), <DL-1.44 (Cd), <DL-8.04 (Cu), <DL-270.68 (Zn), 2.26-241.79 (Fe), and 0.55-1021 (Mn), showing an increasing

trend downstream. Speciation revealed that Cd, Zn, and Mn were mainly present as free ions, whereas Pb and Cu as carbonate complexes. The main sources of PTEs were associated with weathering upstream and agricultural and municipal activities downstream. Human exposure via ingestion was the most important pathway for PTEs. The results indicate that children were more vulnerable compared to adults. Arsenic was identified as the primary driver of non-carcinogenic risk via ingestion pathway. HQderm values for both adults and children indicate that PTEs studied do not pose a threat via dermal absorption. TCRs for As were found in moderate-risk range ( $1 \times 10^{-4} < \text{TCR} < 1 \times 10^{-3}$ ) at all sites and scenarios.

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## Environmentally relevant concentrations of nanoplastic increase the accumulation and endocrine effects of BDE47 and its metabolite 6-OH-BDE-47 in male frogs' testes

Journal of environmental sciences (China) 2026 Jun:164:768-778 · 1 Jun 2026

Emerging pollutants such as polystyrene-nanoplastics (PS-NPs) and polybrominated diphenyl ethers (PBDEs) are a threat to human health and ecological safety. However, NP-mediated PBDE exposure and toxicity remain poorly understood. Herein, male frogs (*Pelophylax nigromaculatus*) were exposed to 2,2',4,4'-tetrabromodiphenyl ether (BDE-47) and 6-hydroxy-2,2',4,4'-tetrabromodiphenyl ether (6-OH-BDE-47) alone or in combination with PS-NPs for 30 days to explore the carrier toxicity effect and mechanism of co-exposure. Transcriptome data verified that both BDE-47 and 6-OH-BDE-47 could mediate the steroid hormone synthesis signaling pathway to induce reproductive toxicity. The presence of PS-NPs increased the bioconcentration of BDE-47 and 6-OH-BDE-47 by 54.36 % and 51.49 %, respectively, and aggravate the damage of the testes. To investigate the mechanism underlying the disrupted steroidogenesis, changes in hormones of the hypothalamic-pituitary-gonadal (HPG) axis and gene expression involved in synthesis and transformation of sex hormones were assessed. AR, StAR, CYP11A1 and HSD3B1 were all downregulated, while mRNA expression levels of CYP19A1 and ESR1 were upregulated, accompanied by a decrease in testosterone and an increase in oestradiol. The results demonstrated that PS-NPs can exacerbate the estrogen-like effects of BDE-47 and 6-OH-BDE-47 in male frogs, highlighting the potential risks posed by these pollutants in aquatic ecosystems.

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## Transformation and transport: Polyvinyl chloride microplastics modulate fipronil accumulation and toxicity in zebrafish

Journal of environmental sciences (China) 2026 Jun:164:308-315 · 1 Jun 2026

Microplastics (MPs) frequently co-occur with organic contaminants in aquatic environments, yet their potential role in mediating contaminant transformation remains underexplored in ecotoxicological assessments. In this study, we investigated the dual role of poly (vinyl chloride) (PVC) MPs (150-530  $\mu\text{m}$ ) as both carriers and reactive surfaces for the insecticide fipronil. Through adsorption experiments, transformation product analysis, and zebrafish dietary exposure trials, we demonstrated that PVC MPs adsorbed fipronil with a capacity of approximately 5.0  $\mu\text{g/g}$  and promoted its transformation into fipronil sulfone (66.9-343.3  $\text{ng/g}$ ) and fipronil desulfinyl (0.66-0.96  $\text{ng/g}$ ). Following 14 days of exposure to fipronil alone (50  $\text{ng/g}$ ), zebrafish accumulated 0.17  $\text{ng/g}$

fipronil and 0.30 ng/g ww (wet weight) fipronil sulfone. In contrast, co-exposure with MPs significantly increased their accumulation to 0.53 and 3.25 ng/g ww, respectively, and additionally resulted in the presence of fipronil desulfinyl (0.77 ng/g ww), which was undetectable in the fipronil-only group. Behavioral assays showed that both individual and combined exposures increased zebrafish locomotion, with synergistic effects observed during the initial 1-3 days. Moreover, combined exposure induced greater alterations in antioxidant enzyme activities than fipronil alone, highlighting the predominant role of MPs in driving oxidative stress. Principal coordinate analysis of enzyme and behavior metrics showed clear distinctions among the control, MPs, fipronil, and combined exposures, emphasizing the significant interactive effects between MPs and fipronil in aquatic organisms.

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## PHARMACEUTICAL/TOXICOLOGY

### **Inorganic arsenic and its methylated metabolites induce pulmonary immunosuppression via the p62-Keap1-Nrf2 positive feedback loop-mediated M2 macrophage polarization**

Ecotoxicology and environmental safety 2026 May 02:317:120214 · 2 May 2026

Trivalent inorganic arsenic (iAsIII) is a widespread environmental contaminant associated with adverse health outcomes, including immune dysregulation. However, the mechanisms underlying pulmonary immunotoxicity induced by iAsIII and its methylated metabolites remain unclear. In this study, acute oral exposure to iAsIII in C57BL/6 mice elicited an early immunosuppressive shift in the pulmonary microenvironment, characterized by upregulated expression of M2 macrophage phenotypic markers and reduced inflammatory cytokines. In vitro, iAsIII and its methylated metabolites, monomethylarsonous acid (MMAIII) and dimethylarsinous acid (DMAIII), promoted a phenotypic transition of murine alveolar macrophages from an M1- to an M2-like state. This shift was evidenced by decreased expression of the M1 marker iNOS and pro-inflammatory cytokines (IL-6 and IL-1 $\beta$ ), along with enhanced expression of the M2 marker ARG1 and the anti-inflammatory cytokine IL-10, with MMAIII exhibiting the strongest immunosuppressive potency. Mechanistically, iAsIII and MMAIII robustly activated the Nrf2-HO-1 pathway, whereas DMAIII elicited relatively weaker activation. Silencing of Nrf2 abolished iAsIII- and MMAIII-driven M2 polarization, indicating an essential role for Nrf2. Furthermore, iAsIII and MMAIII induced aberrant accumulation of the autophagy adaptor p62, accompanied by impaired autophagic flux, lysosomal alkalinization, and increased lysosomal membrane permeability. Knockdown of p62 attenuated Nrf2 activation and reversed the M2-like polarization phenotype. Moreover, the reduction of p62 following Nrf2 silencing further suggests a bidirectional regulatory interaction between p62 and Nrf2. Collectively, these findings demonstrate that iAsIII and MMAIII drive alveolar macrophage polarization toward an immunosuppressive M2 phenotype via a p62-Keap1-Nrf2 positive feedback loop, providing mechanistic insight into iAsIII-induced pulmonary immunosuppression.

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## Short-term exposure to a mixture of tributyltin plus cadmium disrupts the female reproductive organs, inducing ovarian follicular depletion and metabolic offspring outcomes in rats

Reproductive toxicology (Elmsford, N.Y.) 2026 Apr 30:143:109253 · 30 Apr 2026

Tributyltin (TBT) and cadmium (Cd) are environmental pollutants that separately cause reproductive problems. However, the reproductive effects of exposure to a mixture of TBT plus Cd are not well known. We hypothesized that exposure to a mixture of TBT plus Cd (TBT + Cd) changes reproductive function. Female rats were treated with the TBT + Cd mixture daily for 15 days, after which tissue chemical deposition, histology, hormone levels, inflammation, collagen deposition, and protein expression in the ovary and uterus were assessed. Increased tin (Sn) and Cd levels were identified in the sera and livers of TBT + Cd rats. TBT + Cd rats displayed abnormal estrous cycles, reduced serum estrogen and testosterone levels, reduced ovarian reserve and antral follicles, corpora lutea, and total healthy follicle numbers compared to controls, suggesting ovarian follicular depletion. Uterine hypertrophy, inflammation and high collagen deposition were noted in TBT + Cd rats. TBT + Cd rats had increased ovarian and uterine mast cell numbers compared to controls, suggesting inflammation. An increase in estrogen receptor alpha (ER $\alpha$ ) and progesterone receptor (PR) positive cells was noted in the ovaries and uteri of TBT + Cd rats. Prenatal TBT + Cd exposure altered serum glucose and triglyceride levels in the offspring, which may contribute to an unhealthy developmental environment. Collectively, these data indicate that TBT + Cd exposure changes female reproductive function, contributing to ovarian follicular depletion, uterine inflammation and fibrosis, and metabolic abnormalities in the prenatal exposure offspring.

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## High-lipid diets exacerbate copper-induced hepatointestinal toxicity in yellow catfish (*Pelteobagrus fulvidraco*) via gut microbiota dysbiosis

Journal of environmental sciences (China) 2026 Jun:164:729-740 · 1 Jun 2026

Copper (Cu)-contained chemicals and high-lipid diets are commonly applied in aquaculture, both of them are capable of negatively affecting fish health via complicated mechanisms, yet their microbiota-mediated interactions remain unclear. This study investigated their combined effects on yellow catfish (*Pelteobagrus fulvidraco*). A control (Con), high-lipid (HL), and HL + antibiotics (HLA, deplete intestinal microbiota) diets were fed fish for nine weeks, and half fish underwent acute Cu exposure (0.8 mg/L) during week nine (assigned as ConCu, HLCu, and HLACu, respectively). Results showed the HL feeding impaired growth but not Cu exposure. The HL and HLA groups showed exacerbated hepatic vacuolization, oxidative stress (reduced antioxidant enzymes, elevated malondialdehyde (MDA), and inflammation (upregulated  $\text{tnf}\alpha$ ,  $\text{il1}\beta$ , and  $\text{nfk}\beta$  expression). Cu exposure worsened these effects in all groups, further reducing intestinal villus length and tight junction genes ( $\text{zo1}$  and  $\text{occludin}$ ) expression while activating mitogen-activated protein kinase (MAPK)-related inflammation. Hepatic Cu accumulation followed  $\text{HLACu} > \text{HLCu} > \text{ConCu}$  ( $P < 0.05$ ), linked to upregulated Cu transporters ( $\text{ctr1}$  and  $\text{ctr2}$ ) and metallothionein ( $\text{mt2}$ ) expression. Microbiota analysis revealed high-lipid diet and Cu exposure caused microbiota dysbiosis, reducing *Plesiomonas* and *Pseudomonas* abundances, which amplified copper toxicity. This study demonstrates that high-lipid diets intensify Cu-induced hepatointestinal toxicity via oxidative and inflammatory pathways and microbiota dysbiosis, highlighting microbiome-driven Cu metabolism as a key mechanism in fish toxicity.

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