CHEMWATCH

Bulletin Board

Contents

JUN. 13, 2025

(click on page numbers for links)

CONTACT US

subscribers@chemwatch.net
tel +61 3 9572 4700
fax +61 3 9572 4777

1227 Glen Huntly Rd
Glen Huntly
Victoria 3163 Australia

CHEMICAL EFFECTS

Dynamic QSAR modeling for predicting in vivo genotoxicity and inflammation induced by nanoparticles and advanced materials: a time-dose-property/response approach

2025-06-06

Predicting the health risks of nanoparticles (NPs) and advanced materials (AdMa) is a critical challenge. Due to the complexity and time-consuming nature of experimental testing, there is a reliance on in silico methods such as quantitative structure-activity relationship (QSAR), which, while effective, often fail to capture the dynamic nature of material activity over time-essential for reliable risk assessment. This study develops dynamic QSAR models using machine learning to predict toxicological responses, such as inflammation and genotoxicity, following pulmonary exposure to 39 AdMa across various post-exposure time points and dose levels. By incorporating exposure time, administered dose, and material properties as independent variables, we successfully developed time-dose-property/response models capable of predicting AdMa-induced in vivo genotoxicity in bronchoalveolar lavage fluid cells, lung and liver tissue, and inflammation in terms of neutrophil influx into the lungs of mice. Key factors driving AdMa-induced toxicity were identified, including exposure dose, post-exposure duration time, aspect ratio, surface area, reactive oxygen species generation, and metal ion release. The time-dose-property/response modeling paradigm presented here provides a practical and robust approach for predicting in vivo genotoxicity and inflammation and supports the comprehensive risk assessment of morphologically diverse AdMa.

Authors: Michalina Miszczak, Kabiruddin Khan, Pernille Høgh Danielsen, Keld Alstrup Jensen, Ulla Vogel, Roland Grafström, Agnieszka Gajewicz-Skretna

Full Source: Journal of nanobiotechnology 2025 Jun 6;23(1):420. doi: 10.1186/s12951-025-03510-y.

Human health risks from textile chemicals: a critical review of recent evidence (2019-2025)

2025-06-07

Global textile production, driven by consumer demand, raises significant concerns about exposure to chemicals in clothing and related products. This review synthesizes evidence (2019-2025) on hazardous substances in textiles, including dyes, plasticizers, per- and polyfluoroalkyl substances

CHEMWATCH
Bulletin Board
Technical
JUN. 13, 2025

(PFAS), and metals, and identifies and categorizes their associated human health risks. Emerging evidence highlights microfibers as critical vectors for chemical exposure via inhalation and dermal routes, necessitating updated risk assessments. Focusing on dermal absorption as the primary exposure route, risks to vulnerable populations (e.g., infants, pregnant women) and gaps in regulatory frameworks are highlighted. The current analysis reveals that chronic exposure to chemical mixtures in textiles remains poorly understood, with current safety assessments often neglecting synergistic effects. Key findings include elevated risks from phthalates in infant clothing, PFAS in water-repellent fabrics, and carcinogenic aromatic amines (AAs) from azo dyes. We underscore the urgency of harmonized global regulations, advanced biomonitoring, and sustainable alternatives (e.g., enzymatic dyes, biodegradable finishes). Public awareness initiatives and stricter enforcement of certifications like OEKO-TEX® or GOTS are critical to mitigating risks. Interdisciplinary collaboration among textile technologists, toxicologists, and public health experts is essential to develop safer textile alternatives and integrate health-centric approaches into sustainability agendas, safeguarding consumers, workers, and ecosystems.

Authors: Joaquim Rovira, Marília Cristina Oliveira Souza, Martí Nadal, José L Domingo

Full Source: Journal of environmental science and health. Part A, Toxic/hazardous substances & environmental engineering 2025 Jun 7:1-13. doi: 10.1080/10934529.2025.2514406.

Evaluating the relationship between environmental chemicals and obesity: Evidence from a machine learning perspective

2025-06-06

Environmental chemicals are increasingly recognized as important contributors to obesity, yet the number of studies evaluating this relationship remains insufficient. This study aimed to investigate these associations using interpretable machine learning techniques. Data from 1183 participants in the 2011-2012 National Health and Nutrition Examination Survey were analyzed. Several machine learning models, including Support Vector Machines, Random Forest, k-Nearest Neighbors, Naive Bayes, AdaBoost, and XGBoost, were employed to predict generalized and abdominal obesity using environmental chemical exposures and demographic information. The XGBoost model was further explored for its ability to interpret variable contributions, utilizing SHapley Additive exPlanations (SHAP) to identify key predictors. Logistic

regression models revealed that 4-OH-PHEN, 2-OH-NAP, and 2-OH-PHEN were positively associated with generalized obesity, whereas UMo and 3-OH-FLUO exhibited negative associations. Similarly, 4-OH-PHEN demonstrated a positive association with abdominal obesity, whereas 3-OH-FLUO, USr, and BPb were negatively associated. To further examine these relationships, dose-response associations between environmental chemicals and obesity were analyzed using restricted cubic spline plots. A nonlinear relationship was identified between UMo and obesity (P-nonlinear=0.016). Mediation analysis revealed that blood lipids partially mediated the relationship between certain environmental chemicals and obesity. This study underscores the importance of interpretable machine learning in understanding the complex associations between environmental chemicals and obesity. It identified specific chemicals associated with generalized and abdominal obesity and shed light on the mediating role of blood lipids. These findings contribute to the growing body of evidence on the role of environmental exposures in obesity and provide potential pathways for future research and interventions.

Authors: Huan Liu, Huiwen Gu, Jie Li, Yifei Fang, Sheng Yang, Geyu Liang
Full Source: Ecotoxicology and environmental safety 2025 Jun 6:300:118457. doi: 10.1016/j.ecoenv.2025.118457.

ENVIRONMENTAL RESEARCH

Air pollution exposure and respiratory and cardiovascular emergency visits: A time-stratified case-crossover analysis in Taiwan

2025-06-07

Taiwan has undergone rapid urbanization and industrial expansion in recent years, which have significantly contributed to elevated levels of air pollution. This study examines the association between multiple air pollutants and emergency department (ED) visits for respiratory and cardiovascular diseases in selected counties and cities of Taiwan from 2015 to 2019. Using a time-stratified case-crossover approach and conditional logistic regression models adjusted for potential confounders, we analysed over 2,268,048 ED visits to assess the effects of pollutants like PM2.5, PM10, NO2, O3, and SO2 on disease-specific ED visits among residents living nearby coal-fired power plant. We also assessed effects modification by age (20-29 years, 30-59 years, and ≥ 60) and sex groups (male and female). Findings reveal that exposure to PM2.5 and PM10 at lag 5 was associated with increased asthma events, with odds ratios (ORs) of 1.13 (95 % CI: 1.03-1.23) for PM2.5 and 1.16 (95 % CI: 1.05-1.28) for PM10. Additionally, every 15 ppb increase in O3 was associated with increased asthma events at lag 0 (OR = 1.04, 95 % CI: 1.02-1.06). This study further demonstrates that a 1 ppb increase in NO2 was associated with increased ischemic heart disease (IHD) events at lag 1 (OR = 1.16, 95 % CI: 1.00-1.35), stroke at lag 0 (OR = 1.05, 95 % CI: 1.03-1.07), and ischemic stroke (IS) at lag 3 (OR = 1.27, 95 % CI: 1.05-1.49), respectively. These findings underscore the importance of continuous air quality monitoring to enhance the protection of sensitive populations.

Authors: Kaleem Khan, Pin-Zhen Huang, Wen-Chi Pan, Jung-Wei Chang
Full Source: Ecotoxicology and environmental safety 2025 Jun 7:300:118479. doi: 10.1016/j.ecoenv.2025.118479.

PHARMACEUTICAL/TOXICOLOGY

A comprehensive examination of the impact of environmental pollution on lung cancer: A review

2025-06-05

Background: Lung cancer is a leading cause of death worldwide, with environmental factors playing critical roles in its development and progression. Respirable and food-borne contaminants are major contributors to lung cancer onset, influencing various physiological pathways that lead to lung injury and tumor formation.
Aim of review: This review aims to examine the effects of common environmental pollutants on lung cancer development, highlighting the role of specific contaminants, such as PM2.5 (particulate matter with aerodynamic diameter less than 2.5 μm) and nitrogen oxides, and warning people to pay more attention to environmental pollutants.
Key scientific concepts of review: Environmental pollutants play a significant role in increasing the susceptibility to lung cancer by triggering various biological mechanisms that lead to lung injury and tumorigenesis. Excessive PM2.5 exposure contributes to the overall burden of lung cancer via Wnt/β-catenin, Reactive oxygen species-DNA methyltransferases (ROS-DNMT), phosphatidylinositol 3-kinase (PI3K)/protein kinase B (PKB/Akt), Janus kinase/signal transducers and activators of transcription (JAK/STAT) signalling pathways. The primary mechanisms by which NO contributes to the occurrence and development of pulmonary neoplasm revolve around the production and regulation of ROS. Occupational exposure and ecosystem pollution to hazardous substances, including microplastics, pesticides, asbestos, cadmium, and nickel, are the well-established risk factors for the development of lung cancer via DNA damage,

Copyright Chemwatch 2025 ©

oxidative stress, and inflammation pathways. This review emphasizes the importance of effective prevention strategies for lung cancer by reducing environmental pollution levels.
Authors: Hongyan Zhang, Hongyi Wei, Sijia Han, Lufeng Zheng, Xiaodong Chen, Ziwei Li, Lanbo Wang
Full Source: Journal of advanced research 2025 Jun 5:S2090-1232(25)00392-3. doi: 10.1016/j.jare.2025.06.006.

Per- and polyfluoroalkyl substances and associated proteomic biomarker patterns of immuno-suppression and cell-proliferation in an adolescent population from Northern Norway: The Fit Futures Study

2025-06-05
Background: Per- and polyfluoroalkyl substances (PFAS) are stable man-made chemicals which are extensively used in the production of common consumer products. High human exposure has been associated with immunotoxicity. This study aimed to explore the relationship between PFAS exposure and proteomic biomarkers in a cross-sectional cohort of an adolescent population in northern Norway.
Methods: This study included 839 adolescents. The serum concentration of 18 PFAS and 92 proteomic biomarkers were measured. Eight PFAS and 75 biomarkers were detected in >70% of the sample population, respectively. This study investigated these eight PFAS and 75 biomarkers. Factor analysis (FA) was used to reduce the dimensionality of the 75 biomarkers into factors/patterns. We applied a multivariate regression modelling by fitting factors as dependent variables with each of the PFAS as independent variables in separate models.
Results: Of the five factors extracted by the FA, Factor 2 loaded 10 biomarkers crucial in the development of innate and adaptive immunity. Factor 3 loaded five biomarkers which are important in mitogenic, cell-proliferation, and inflammation processes. Perfluorooctanesulfonate (PFOS), perfluoroundecanoate (PFUnDA), and perfluoroheptane sulfonate (PFHpS) were inversely associated with Factor 2: PFOS, (β= -0.26 [95% CI -0.42, -0.11]); PFUnDA, (β= -0.20 [95% CI -0.30, -0.10]); and PFHpS, (β= -0.17 [95% CI -0.28, -0.05]); while perfluorooctanoate (PFOA) was positively associated with Factor 3: PFOA, (β= 0.28 [95% CI 0.12, 0.44]).

CHEMWATCH
Bulletin Board
Technical
JUN. 13, 2025

Conclusion: Exposure to PFOS, PFUnDA, and PFHpS were associated with biomarkers related to immunosuppression, while exposure to PFOA was associated with biomarkers related to carcinogenesis.
Authors: Sunday Oluwafemi Oyeyemi, Maria Averina, Sandra Huber, Dolley Charles, Anne-Sofie Furberg, Kassaye Yitbarek Yigzaw, Tom Wilsgaard, Christopher Sivert Nielsen, Guri Grimnes
Full Source: Environmental research 2025 Jun 5:122096. doi: 10.1016/j.envres.2025.122096.

Bladder cancer risk in aluminum production workers: A systematic review

2025-06-05
Objective: To summarize the key findings of occupational exposures during aluminium production in cohort studies on bladder cancer published between 1979 and 2023.
Materials and methods: This systematic review was conducted in accordance with PRISMA guidelines and registered in PROSPERO. Study quality was evaluated using the Newcastle-Ottawa scale.
Results: The literature search identified 24 cohort studies examining the standardized incidence (SIR) and mortality ratios (SMR) of bladder cancer among aluminum production workers. Five of the 13 studies examined SIR (95% CI) and three of the 16 studies that examined SMR (95% CI) reported a statistically significant increased risk of bladder cancer among aluminum production workers. The highest SIR was recorded in the secondary aluminum smelter (2.85; 95% CI: 1.23-5.62), suggesting a significantly elevated risk. In aluminum reduction plants, the SIR was 1.82 (95% CI: 1.59-2.07), while in Söderberg plants, it was 1.69 (95% CI: 1.06-2.57) and 1.4 (95% CI: 1.0-1.9), respectively. For aluminum plants overall, the SIR was 1.30 (95% CI: 1.10-1.50). The results indicate an increased risk, with the highest SMR of 5.90 (95% CI: 1.58-15.1), suggesting a significantly elevated hazard in the secondary aluminum smelter. The other values-3.47 (95% CI: 1.25-9.62) for the aluminum smelter and 2.24 (95% CI: 1.77-2.79) for the aluminum reduction plant-also indicate an increased risk, albeit with varying degrees of statistical certainty.
Conclusion: The International Agency for Research on Cancer has classified aluminum production as a Group 1 carcinogenic activity, providing strong evidence of its association with bladder cancer. The studies primarily included workers from Prebake, Söderberg, and aluminum reduction plants. Some findings indicate a significant yet variable risk across different segments of aluminum production. However, the review did not clearly confirm an increased risk of bladder cancer in specific aluminum-related

occupations. Aluminum exposure may influence the lifetime risk of bladder cancer among these workers.
Authors: E Kasperczyk, M Lesicka, E Reszka
Full Source: Actas urologicas espanolas 2025 Jun 5:501789. doi: 10.1016/j.acuroe.2025.501789.

OCCUPATIONAL

PFOS caused fertility defects and disrupted spermatogenic gene networks in the male medaka with a transgenerational history of ancestral BPA exposure

2025-06-04
Environmental chemical exposures induce heritable and transgenerational effects in reproductive and metabolic systems. How organisms respond when exposed to contemporary environmental chemicals overlaid upon the transgenerational inheritance of their ancestors' exposure profiles is currently unknown. Here, we investigated the effects of a second hit of perfluorooctane sulfonate (PFOS) exposure on male reproductive health in medaka fish (Oryzias latipes) with or without a history of ancestral bisphenol A (BPA) exposure. The PFOS exposure occurred in offspring four generations after ancestors' BPA exposure (10 ug/L) during their embryonic development. Three concentrations of PFOS (0, 0.002, and 0.02 mg/L for 21 days) were tested in two lineages: control lineage with no ancestral history of BPA exposure and BPA lineage whose ancestors were exposed to BPA four generations ago. Our results show that the second hit of PFOS significantly decreases fertility in fish with a pre-existing history of ancestral BPA exposure. RNA sequencing of the testis revealed that PFOS exposure in the BPA lineage caused a significant increase in the number of upregulated genes. In contrast, in the control lineage, it caused downregulation of genes related to cell cycle dysregulation. Differentially expressed genes in the PFOS-exposed BPA lineage fish were related to apoptosis, proteolysis, and cytoskeletal disarrangement. The genes associated with Sertoli cell function and spermatogenesis, mainly associated with locomotion, mitotic cycle, and cell morphogenesis, were significantly dysregulated in the BPA lineage fish. Altogether, the present study found exacerbated fertility defects and significant alterations in the molecular networks associated with Sertoli cell function & spermatogenesis due to the second hit of PFOS in the fish with a history of ancestral BPA exposure, suggesting that a pre-existing history of ancestral environmental chemical exposure can be a contributing factor

CHEMWATCH
Bulletin Board
Technical
JUN. 13, 2025

for adverse health outcomes in the upcoming generations upon exposure to contemporary legacy chemicals.
Authors: Sourav Chakraborty, Seraiah T Coe, Santosh Anand, Mehwish Faheem, Xuegeng Wang, Ramji Kumar Bhandari
Full Source: Environmental research 2025 Jun 4:122056. doi: 10.1016/j.envres.2025.122056.

Characterising neonicotinoid insecticide exposures among the Irish population using human biomonitoring

2025-06-07
Neonicotinoid and neonicotinoid-like insecticides (NNIs) are the most widely used class of insecticides in the world, with previous large-scale human biomonitoring studies of NNIs showing widespread exposure. They have been identified as priority substances requiring further toxicological and human exposure research by numerous initiatives, including the Partnership for the Assessment of Risks from Chemicals (PARC). The study aimed to conduct the first human biomonitoring study investigating exposures to NNIs in Ireland by analysing 227 urine samples from the Irish population. Samples were collected between 2019 and 2020 from 14 farm and 54 non-farm families throughout Ireland and analysed for seven NNIs (acetamiprid, clothianidin, imidacloprid, thiacloprid, thiamethoxam, flupyradifurone, and sulfoxaflor) and nine of their metabolites using online-solid phase extraction coupled to liquid chromatography-tandem mass-spectrometry (online-SPE-LC-MS/MS). The results found that 75 % of samples had quantifiable levels of at least one parent compound or metabolite. N-desmethyl acetamiprid (dme-ACE) and imidacloprid-olefin (IMI-olefin), the main metabolites of acetamiprid and imidacloprid, were the most widely detected analytes and could be quantified in 57 % and 34 % of the urine samples, respectively. Based on reverse dosimetry, the maximum urinary concentration of dme-ACE corresponded to 31.7 % of the acceptable daily intakes (ADI) for acetamiprid, which has been recently reduced five-fold. In comparison, the maximum urinary concentrations of all other NNIs analysed in the study corresponded to less than 3 % of the ADIs of the respective parent NNIs. Though NNI exposure was widespread among this study group, the exposure levels were below current regulatory guidance values.
Authors: Sonja A Wrobel, Darragh M Doherty, Holger M Koch, Heiko U Käfferlein, Daniel Bury, Craig Slattery, Marie A Coggins, Alison Connolly
Full Source: International journal of hygiene and environmental health 2025 Jun 7:268:114610. doi: 10.1016/j.ijheh.2025.114610.

Arsenic exposure, genetic susceptibility, lifestyle, and glucose-insulin homeostasis impairment: Revealing the association and interaction in a repeated-measures prospective study

2025-06-05

Arsenic exposure, genetic factors, and lifestyle are individually associated with glucose-insulin homeostasis, yet no study focuses on their interactions. We aimed to investigate the combined effects and interactions of urinary total arsenic (UTAs), genetic susceptibility, and lifestyle on glucose-insulin homeostasis. We included 6136 and 5408 observations from the Wuhan-Zhuhai cohort for cross-sectional and longitudinal analyses, respectively. At baseline (2011-2012) and two subsequent follow-ups (2014-2015, 2017-2018), we repeatedly measured UTAs, fasting plasma glucose (FPG), fasting plasma insulin (FPI), and homeostasis model-assessed insulin resistance (HOMA-IR) and β-cell function (HOMA-β). Polygenic risk scores (PRSs) and healthy lifestyle score (HLS) were constructed. Linear mixed models including participant-specific random intercept to account for repeated-measures design were used to assess associations and interactions of UTAs, PRSs, and HLS with glucose-insulin homeostasis. UTAs was associated with annual growth rates of FPG, FPI, and HOMA-IR with βs (95% CIs) of 0.020 (0.008, 0.032), 0.007 (0.002, 0.013), and 0.011 (0.004, 0.017), respectively. For each standard deviation increase in trait-specific PRSs, 6-year changes in FPG, FPI, HOMA-IR, and HOMA-β were 0.136 (95% CI: 0.066, 0.207) mmol/L, 0.041 (0.013, 0.070), 0.040 (0.011, 0.068), and -0.063 (-0.098, -0.028) ln-unit, respectively. UTAs and PRS had significant interactions on the longitudinal progressions of elevated FPI and HOMA-IR (all Pinteraction<0.05). Compared with the reference group, participants with the fourth-quartile PRS and high UTAs had 0.029 (0.012, 0.047) and 0.032 (0.012, 0.051) ln-unit increases in annual growth rates of FPI and HOMA-IR, respectively. Moreover, participants with low UTAs, low PRS, and high HLS had the largest longitudinal progressions of decreased FPI (-0.033 [-0.052, -0.013]) and HOMA-IR (-0.038 [-0.060, -0.016]). Arsenic exposure and genetic susceptibility are synergistic risk factors for accelerated deterioration of insulin resistance. Our findings emphasize the importance of reducing

CHEMWATCH
Bulletin Board
Technical
JUN. 13, 2025

arsenic exposure and adopting healthy lifestyle to maintain normal glucose-insulin metabolism regardless of genetic risk.

Authors: Yongfang Zhang, Jiahao Song, Ming Zhang, Gaoyin Xiong, Yanjun Guo, Wei Liu, Linling Yu, Quanhong Liu, Yueru Yang, Shuhui Wan, Qing Liu, Zhiying Huo, Ruiyi Liang, Bin Wang, Weihong Chen

Full Source: Environmental research 2025 Jun 5:122085. doi: 10.1016/j.envres.2025.122085.