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CHEMICAL EFFECTS

Electrocatalytic CO2 Reduction to Chemicals and Fuels: From Single-Atom to Dual-Atom

2025-07-17

In recent years, single-atom catalysts (SACs) have emerged as a prominent research focus in electrochemical CO2 reduction reactions (CO2RR), owing to their exceptional atomic utilization efficiency and superior catalytic performance. Nevertheless, their practical implementation may be constrained by inherently low metal loading and the presence of linear relationships imposed by their structurally simplistic active sites. Atomic-level engineering of active sites represents a transformative strategy to overcome the intrinsic limitations of SACs. Building upon the foundation of SACs, dual-atom catalysts (DACs) exhibit enhanced metal loading capacity and more sophisticated active site configurations, leading to superior catalytic performance and broader opportunities in electrocatalytic applications. This review first explains the possible reaction pathways for product generation via CO2RR, including the underlying mechanisms, key intermediates, and strategies to optimize these pathways. Subsequently, a comprehensive overview of synthetic strategies and precise atomic-level characterization of atomic-scale catalysts is presented. SACs and DACs are systematically categorized according to their active site architectures and electronic configurations. The significant advantages of DACs over SACs in increasing the metal atom loading, promoting the adsorption and activation of CO2 molecules, regulating intermediates, and promoting C-C coupling are compared. Finally, the prevailing challenges and future development prospects of DACs are summarized.

Authors: Fangjun Wang, Shiyi Chen, Xiang Xu, Xiaohan Chen, Jiang Wu, Shubo Chen, Wenguo Xiang, Lunbo Duan

Full Source: Small (Weinheim an der Bergstrasse, Germany) 2025 Jul 17:e2505474. doi: 10.1002/smll.202505474.

Differential impacts of nickel toxicity: NiO and NiSO4 on skin health and barrier function

2025-07-17

Nickel is recognized as a potent skin sensitizer and a common cause of contact dermatitis. Nickel and its compounds are often associated with particulate matter in industrial settings. This study aimed to evaluate the effects of nickel oxide particulate matter (NiOPM) using in vitro skin models, and to compare the effects of NiSO4 topical application on

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healthy versus atopic dermatitis-sensitized skin. Key endpoints included histological analysis, cell viability, cytokine release, proliferation index, and protein expression. The results revealed that the reconstructed epidermal tissue representing healthy skin was properly stratified. After 24 h of exposure to NiOPM (0.4-4.6 mg/cm2), histological analysis and viability data (>50 %) indicated a lack of cytotoxicity related to irritation. However, ion beam analysis, immunofluorescence, cell proliferation (Ki67 marker), and inflammatory signaling (IL-1α, IL-8) suggest that prolonged exposure may be associated with increased epidermal permeability and oxidative stress, identifying NiOPM as a possible long-term sensitizer. In addition, comparative treatments of NiOPM vs. NiSO4 on models of healthy epidermis and with atopic epidermis, exposed for up to 72 h, demonstrate the damaging effect of NiSO4 as early as the first 24 h. Also, the results suggest differential effects on proliferative cell presence and loricrin expression. These findings indicate that elucidating the sensitization pathways of nickel is complex. The physicochemical characteristics of Ni compounds are closely related to exposure time, skin permeation capacity, and cellular damage.

Authors: Denisse Esther Mallaupoma Camarena, Mariana Corrêa Giannella, Julia de Toledo Bagatin, Silvia Romano de Assis, Tao Chen, Melanie Jane Bailey, Catia Costa, Ella Schneider, Johanna von Gerichten, Silvia Berlanga de Moraes Barros, Natalie Belsey, Silvya Stuchi Maria-Engler

Full Source: Ecotoxicology and environmental safety 2025 Jul 17:302:118626. doi: 10.1016/j.ecoenv.2025.118626.

Unravelling the potential mechanisms of nano- and microplastic toxicity to the male reproductive system: a systematic review

2025-07-17

The ever-increasing presence of microplastic and nanoplastic (MPs/NPs) particles in the natural environment, organisms, and a wide variety of health products, cosmetics, pharmaceuticals, and foods consumed by humans is a global concern. In recent years, research efforts have shifted towards identifying human exposure and risks associated with MPs/NPs, as well as unravelling the mechanisms underlying their toxicity. This systematic review examined the literature regarding the effects of MPs/NPs on the male reproductive system, focusing on the testis, epididymis, and their associated barriers. Research, conducted primarily on rodents, demonstrated that MPs/NPs of various chemical compositions can bioaccumulate in the testis and epididymis, identifying these organs as key targets of plastic particle toxicity. Several studies using

rodent models reported alterations in the blood-testis barrier, a crucial structure necessary for proper spermatogenesis. Additionally, multiple studies observed increased apoptosis of germ cells, malformations of spermatozoa, and decreased sperm motility, which is typically acquired during epididymal transit. Exposure to MPs/NPs disrupted Sertoli and Leydig cell function, leading to hormone imbalance. This is likely due to a combination of oxidative stress, inflammation, and disruption of the blood-testis barrier. These effects appear to be influenced by a combination of particle characteristics, including size, shape, chemical composition, surface properties, and exposure route. Larger MPs often cause greater structural damage, while smaller NPs more readily penetrate tissues and trigger molecular disruptions. Understanding how these particles alter male reproductive functions is essential for evaluating their full impact on fertility.

Authors: Paloma da Cunha de Medeiros, Aline Gabrielle Gomes da Silva, Ana Beatriz Silva Angelo, Maria Joana Nogueira de Moura, Unnikrishnan Kannan, Mary Gregory, Julie Dufresne, Cibele Dos Santos Borges, Daniel G Cyr

Full Source: Reproductive toxicology (Elmsford, N.Y.) 2025 Jul 17:109002. doi: 10.1016/j.reprotox.2025.109002.

ENVIRONMENTAL RESEARCH

Association between high polygenic risk scores and long-term exposure to air pollution in asthma development: a hospital-based case-control study

2025-07-18

Background: Air pollution is widely associated with allergic diseases, including asthma. Although previous studies have suggested an epidemiological link between air pollution and asthma, the combined effects of air pollutants and polygenic risk scores (PRSs) on asthma risk remain incompletely understood. This study aimed to examine the impact of air pollutants and PRS on asthma risk among patients in a Taiwan medical institution.
Methods: This retrospective matched case-control study utilized data from the Taiwan Precision Medicine Initiative (TPMI) project to compare asthma patients with a non-asthmatic control group. Participants were stratified into quartiles based on their asthma PRS, while air pollutant exposure was assessed by both duration and concentration. Conducted at Taichung Veterans General Hospital, the study followed participants from January 1, 2000, to December 31, 2021. Logistic regression was used to analyze the relationships between air pollution exposure, genetic risk, and asthma incidence.
Results: A total of 9,756 participants were included (3,252 asthma patients and 6,504 controls). Individuals in the highest PRS quartile demonstrated a significantly increased asthma risk (odds ratio = 1.532, 95% CI = 1.333-1.762, p < 0.0001). Long-term exposure to low levels of PM2.5, PM10, NO2, Mn, and O3 further elevated asthma risk, with the association becoming more pronounced under conditions of high air pollution.
Conclusion: Long-term exposure to low concentrations of air pollutants significantly increases asthma risk, especially among individuals with high genetic susceptibility. These findings emphasize the importance of personalized health management for individuals with elevated PRS.
Trial registration: Not applicable.

Authors: I-Chieh Chen, Yi-Ming Chen, Yun-Wen Chen, Tzu-Hung Hsiao, Hui-Wen Yang, Kuo-Tung Tang, Ching-Heng Lin, Yu-Wen Chu

Full Source: Environmental health : a global access science source 2025 Jul 18;24(1):49. doi: 10.1186/s12940-025-01206-2.

Associations between life course exposure to ambient air pollution with cognition and later-life brain structure: a population-based study of the 1946 British Birth Cohort

2025-07-16

Background: Previous research has linked higher exposure to air pollution to increased cognitive impairment at older ages. We aimed to extend the existing evidence in this area by incorporating exposures across the life course in addition to measures of cognition and brain structural imaging in participants at midlife to older age.
Methods: For this population-based study, we used data from the Medical Research Council National Survey of Health and Development (NSHD; also known as the 1946 British Birth Cohort) and a neuroimaging substudy of the NSHD known as Insight 46. Participants were recruited after birth in a single week during March, 1946. Our objectives were to assess whether exposure to air pollutants in midlife (age 45-64 years) was associated with poorer processing speed and poorer verbal memory between the ages of 43 years and 69 years, and whether exposures were associated with poorer cognitive state and brain structure outcomes at age 69-71 years. Air pollution exposure data were available for nitrogen dioxide (NO2; ages 45-64 years); particulate matter with diameter less than 10 µm (PM10; ages 55-64 years); and nitrogen oxides (NOx) and particulate matter with diameters less than 2·5 µm (PM2·5) and between 2·5 µm and less than

10 µm (PMcoarse) and particulate matter absorbance (PM2·5abs) as a measure of black carbon absorption (ages 60-64 years), with adjustments for early-life exposures to black smoke and sulphur dioxide. Verbal memory was tested with a 15-item recall task and processing speed with a visual search task at ages 43, 53, 60-64, and 69 years. The Addenbrooke's Cognitive Examination III (ACE-III), a measure of cognitive state, was conducted at age 69 years. Whole-brain, ventricular, hippocampal, and white matter hyperintensity volumes were assessed by MRI at age 69-71 years. Generalised linear models and generalised mixed linear models were used to explore associations between pollution exposure, cognitive measures, and brain structural outcomes, adjusted for sociodemographic factors including smoking status and neighbourhood deprivation. Findings: Between the ages of 43 years and 69 years, we included 1534 NSHD participants in the verbal memory and processing speed analysis. Of 2148 participants who underwent testing during the wave of follow-up in 2015-16, at age 69 years, 1761 were included in the ACE-III analysis. Of the 502 NSHD participants recruited into the Insight 46 substudy, 453 were included in the analysis. Higher exposure to NO2 and PM10 was associated with slower processing speed between the ages of 43 years and 69 years (NO2 β -8·121 [95% CI -10·338 to -5·905 per IQR increase in exposure]; PM10 β -4·518 [-6·680 to -2·357]). Higher exposure to all tested pollutants was associated with lower ACE-III score at age 69 years (eg, NO2 β -0·589 [-0·921 to -0·257]). Higher exposure to NOx was associated with smaller hippocampal volume (β -0·088 [-0·172 to -0·004]) and higher exposure to NO2 and PM10 was associated with larger ventricular volume (NO2 β 2·259 [0·457 to 4·061]; PM10 β 1·841 [0·013 to 3·669]) at age 69-71 years. Interpretation: Acknowledging the probable effects of exposure early in life, higher exposure to nitrogen dioxide, nitrogen oxides, and coarse particulate matter in midlife to older age was associated with poorer cognition, processing speed, and brain structural outcomes, strengthening evidence for the adverse effects of air pollution on brain function in older age.

Funding: The National Institute for Health and Care Research, the Medical Research Council (MRC), Alzheimer's Research UK, the Alzheimer's Association, MRC Dementias Platform UK, and Brain Research UK.

Authors: Thomas Canning, Jorge Arias-de la Torre, Helen L Fisher, John Gulliver, Anna L Hansell, Rebecca Hardy, Stephani L Hatch, Ian S Mudway, Amy Ronaldson, Molly Cartlidge, Sarah-Naomi James, Sarah E Keuss, Jonathan M Schott, Marcus Richards, Ioannis Bakolis

Full Source: The lancet. Healthy longevity 2025 Jul 16:100724. doi: 10.1016/j.lanhl.2025.100724.

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Harmonizing cannabis and environmental policy

2025-07-19

Policy fragmentation contributes to the myriad environmental impacts of the cannabis industry and impedes solutions. Examination of how this plays out in the context of the US provides real-world examples, further complicated by the lack of a robust overarching federal policy framework. A key determinant of environmental problems is whether cannabis cultivation is conducted indoors (greenhouses or windowless plant factories) or outdoors in open fields. For example, emissions of greenhouse gases are 100-times greater for indoor cultivation, and water use is higher when accounting for water associated with power production. Other distinct spheres of impact involve land use, waste production, air pollution, ecosystem degradation, and adverse indoor environmental quality conditions faced by workers. Some consequences are differentially burdensome for marginalized populations, suggesting an overlay of environmental justice considerations. Relevant but uncoordinated regulatory entities include those focused formally on cannabis regulation, law enforcement, energy supply and demand, building codes, air quality, climate change, water supply and quality, waste management, fish and wildlife, occupational safety, public health, and urban planning. While criminalization has contributed to environmental concerns, both legal and illicit operations are problematic. Cannabis legalization enables improved policy coordination, but is not a panacea or intrinsic solution. The potential for policymaker collaboration and regulatory coordination includes removing barriers to improved practices and, in particular, providing incentives for lower-impact outdoor cultivation. However, politics often stands as an impediment to otherwise pragmatic policy solutions.

Authors: Evan Mills

Full Source: The International journal on drug policy 2025 Jul 19:143:104923. doi: 10.1016/j.drugpo.2025.104923.

PHARMACEUTICAL/TOXICOLOGY

Random peptide mixtures of tryptophan and lysine suppress the aggregation of a cancer-related mutant of the Axin protein

2025-07-10

Aggregation of dysfunctional proteins can lead to a variety of diseases including cancer. We have previously developed chaperone-derived peptides that inhibit aggregation of the cancer-related L106R mutant

of Axin RGS. Here we show that significantly improved inhibition was achieved using random peptide mixtures (RPMs) designed to mimic the chemical characteristics of the chaperone-like peptides. 20-mer RPMs of tryptophan and lysine suppressed aggregation of Axin RGS L106R with up to 50-fold improved activity compared to parent inhibitors. Conversely, peptides derived from the lead hotspot of Axin RGS aggregation that were designed to be specific, were unable to prevent aggregation of the protein. RPMs constitute the most efficient strategy to date to magnify peptide inhibitory activity against Axin RGS L106R aggregation, as they contain multiple active species and conformations that cover a larger inhibitory space and shield multiple hotspots at once. Our results demonstrate that the chemical composition of the peptide, and not the specific sequence, is the key factor for inhibitory activity.
Authors: Tommaso Garfagnini, Zvi Hayouka, Assaf Friedler
Full Source: RSC chemical biology 2025 Jul 10. doi: 10.1039/d5cb00141b.

Per- and polyfluoroalkyl substances (PFAS) toxicity and mitigation of adipogenic dysregulation in 3T3-L1 preadipocytes

2025-07-16
Obesity and metabolic disorders are rising global concerns, with one factor linked to per- and polyfluoroalkyl substances (PFAS), a class of persistent endocrine-disrupting chemicals that affect adipogenic pathways. While PFAS are known to promote terminal lipid accumulation in mature adipocytes, their effects during dynamic adipocyte differentiation, particularly at human-relevant doses and in mixtures, remain poorly understood. This study employed 3T3-L1 preadipocytes to assess PFAS toxicity and mitigation in cell viability, redox balance, mitochondrial integrity, lipid content, and gene expression during differentiation. Repeated, low-dose exposure to 0.1 μM PFAS (individual and mixture) time-dependently reduced cell viability through induced reactive oxygen and nitrogen species by 22-49 % and reduced mitochondrial membrane potential. The 2-4-fold increase in lipid accumulation, especially by PFOA, correlated with Pparγ activation by 47-86 % ($r2 \geq 0.98$). PFAS also triggered pro-inflammatory cytokines (Tnfα, Il-6) and suppressed anti-inflammatory mediators (Tgfβ, Il-10, Il-4), suggesting interactions between adipogenic and inflammatory pathways. Importantly, montmorillonite clays amended with choline or chlorophyll at 0.001 % and 0.01 % dose-dependently mitigated these effects, supporting critical PFAS pathways. By integrating mechanistic toxicology with mitigation interventions, this work establishes PFAS adipogenic toxicity through oxidative, metabolic, and inflammatory pathways, and supports nutrient-amended clays as sorbents to reduce PFAS bioavailability and prevent toxicity pathways during adipocyte development.
Authors: Qinfu Wang, Soonkyu Chung, Meichen Wang
Full Source: Food and chemical toxicology : an international journal published for the British Industrial Biological Research Association 2025 Jul 16:204:115649. doi: 10.1016/j.fct.2025.115649.

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OCCUPATIONAL

Non-Pharmacological interventions for psychological stress reactions in disaster nursing rescue workers: a scoping review

2025-07-18
Background: Disasters exert a significant negative impact on the mental health of medical staff. Although many non-pharmacological interventions exist, there currently is a lack of literature that systematically summarizes these interventions. Therefore, this study aims to review existing literature on the use of non-pharmacological interventions to address psychological stress reactions in disaster nursing rescue workers.
Methods: This study was guided by the methodological framework proposed by Arksey and O'Malley. A systematic search was conducted from the inception to January 19, 2025 in the Web of Science, PubMed, Cochrane Library, Embase, CINAHL, APA PsycInfo, CNKI, WanFang Data, VIP, and CBM databases. The protocol for this review has been registered on OSF, with the DOI: https://doi.org/10.17605/OSF.IO/X6YPF .
Results: The initial databases searches yielded 7308 articles. A total of 18 studies were included, of which 11 were randomized controlled trials and 7 were quasi-experimental studies. There are four types of non-pharmacological interventions: (1) Mindfulness and mind-body exercise interventions, (2) Psychological adaptation and emotion regulation interventions, (3) Digital cognitive-behavioral interventions, (4) Exercise and time management interventions. Outcome indicators are symptoms of psychological stress response, including anxiety, depression, terror, stress, burnout, post-traumatic stress disorder (PTSD), fatigue, insomnia, and these studies also assessed wellbeing, resilience, and self-compassion.
Conclusions: Non-pharmacological interventions have been effective in reducing psychological stress responses in disaster nursing rescue workers. In the future, targeted intervention plans should be tailored to the specific mental health issues of rescue workers, and their effectiveness

and cost-efficiency need to be further evaluated through large-sample, multi-center, and diversified research designs.
Clinical trial number: Not applicable.
Authors: Hongxiu Wang, Ying Xiao, Huilu Yao, Yiting Wen, Meiling Deng, Wei Yang, Xiaorong Mao
Full Source: BMC nursing 2025 Jul 18;24(1):941. doi: 10.1186/s12912-025-03559-y.

Analysis of Occupational and Non-Occupational Factors Related to Spinal Pain in Office Workers Who Use Computer: A Cross-Sectional Study From Türkiye

2025-07-19
Background: Identifying work-related musculoskeletal pain and the factors that influence it is the first step in preventing and reducing office worker complaints.
Purpose: This study aimed to analyze occupational and non-occupational risk factors related to spinal pain and to develop a model for predicting the risk of spinal pain in office workers.
Methods: A cross-sectional study design was used. The Cornell Musculoskeletal Discomfort Questionnaire was used to determine the presence of spinal pain. The sample consisted of computer-using office workers from a university (N = 264), of whom 162 (61.3%) were male, 102 (38.7%) were female. They were assessed for occupational (work-related inquiries, Rapid Upper Limb Assessment [RULA] evaluates the risk of workers to upper extremity-related ergonomic risk factors, and Rapid Office Strain Assessment [ROSA] evaluates workplace-related ergonomic risk factors) and non-occupational factors (demographic information). Independent samples t-test was used to compare office workers with and without spinal pain. The model was developed using stepwise multiple regression analysis.
Results: Chair features were linked to pain in the neck, upper and lower back. Computer peripherals were related to the pain in the neck and upper back. Accurate predictors of spinal pain in computer-using office workers were identified as older age, female gender, higher BMI, prolonged weekly computer use time, no passive rest breaks, higher RULA and ROSA scores.
Conclusion: Prevention of spinal pain should be focused on comprehensive health strategies aimed at increasing awareness of work

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posture and workplace ergonomics, especially for older, female, and high BMI computer-using office workers.
Authors: Zehra Nur Gülüstan, Simge Sağlam, Özge Dere, Asalet Aybüke Güp, Özge İpek Dongaz, Banu Bayar
Full Source: Workplace health & safety 2025 Jul 19:21650799251353263. doi: 10.1177/21650799251353263.