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CHEMICAL EFFECTS

Are eco-friendly "green" tires also chemically green? Comparing metals, rubbers and selected organic compounds in green and conventional tires

2024-06-28

Tires are a major source of synthetic and natural rubber particles, metals and organic compounds, in which several compounds are linked to negative environmental impact. Recent advances in material technology, coupled with focus on sustainability, have introduced a new range of tires, sold as "green, sustainable, and eco-friendly". Although these "green" tires may have lower impact on the environment on a global scale, there is no current knowledge about the chemical composition of "green" tires, and whether they are more eco-friendly when considering the release of tire wear particles or tire-associated chemicals. Here we have investigated the chemical composition of nine "green" vehicle tires, one "green" bike tire and seven "conventional" vehicle tires. No significant difference was found between "green" and "conventional" tires tested in this study. For N-(1,3-dimethylbutyl)-N'-phenyl-p-phenylenediamine (6PPD), the average concentration in "green" tires were higher (16 ± 7.8 μg/mg) compared to "conventional" tires (8.7 ± 4.5 μg/mg). The relationship between metals, selected organic compounds and rubbers demonstrated large variation across brands, and lower variability between tires grouped according to their seasonal use. This study indicates that more work is needed to understand how the shift towards sustainable tires might change the chemical composition of tires.

Authors: Elisabeth S Rødland, Gilberto Binda, Davide Spanu, Stefano Carnati, Laura Röhler Bjerke, Luca Nizzetto

Full Source: Journal of hazardous materials 2024 Jun 28:476:135042. doi: 10.1016/j.jhazmat.2024.135042.

Chemical characteristics and formation mechanism of secondary inorganic aerosols: The decisive role of aerosol acidity and meteorological conditions

2024-06-28

In recent years, there has been a growing concern about air pollution and its impact on the air quality and human health, especially for fine particulate matter (PM2.5) and its associated secondary aerosols in urban areas. This study conducted a year-long field campaign to collect PM2.5 samples day and night in an urban area of central Taiwan. Higher PM2.5

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mass concentrations were observed in winter (27.7 ± 9.7 μg/m3), followed by autumn (22.5 ± 8.3 μg/m3), spring (19.2 ± 6.4 μg/m3), and summer (11.0 ± 3.1 μg/m3). The dominant formation mechanism of secondary inorganic aerosols was heterogeneous reactions of NO3- at night and homogeneous reactions of SO42- during the day. Additionally, significant correlations were observed between aerosol liquid water content (ALWC) and NO3- during nighttime, indicating the importance of aqueous-phase NO3- formation. The role of aerosol acidity was explored and a unique alkaline condition was found in spring and summer, which showed lower PM2.5 concentrations than the neutralized condition. Under the neutralized condition, higher PM2.5 concentrations were commonly found when combining the ammonium-rich regime with molar ratios of [NO3-]/[SO42-] exceeding 1.6, suggesting the importance of reducing both NH3 and NOx. Furthermore, the results showed that reducing NH3 should be prioritized under high temperature conditions, while reducing NOx became important under low temperature conditions. Clustering of backward trajectories showed that long-range transport could enhance the formation of secondary aerosols, but local emissions emerged as the main factor driving high PM2.5 concentrations. This study provides insights for policymakers to improve air quality, suggesting that different mitigation strategies should be formulated based on meteorological variables and that using clean energy for vehicles and electricity generation is important to alleviate air pollution.

Authors: Yu-Chieh Ting, Chuan-Hsiu Huang, Yu-Hsiang Cheng, Ta-Chih Hsiao, Webber Wei-Po Lai, Zih-Jhe Ciou

Full Source: Environmental pollution (Barking, Essex : 1987) 2024 Jun 28:124472. doi: 10.1016/j.envpol.2024.124472.

ENVIRONMENTAL RESEARCH

Folliculogenesis and steroidogenesis alterations after chronic exposure to a human-relevant mixture of environmental toxicants spare the ovarian reserve in the rabbit model

2024-06-28

Background: Industrial progress has led to the omnipresence of chemicals in the environment of the general population, including reproductive-aged and pregnant women. The reproductive function of females is a well-known target of endocrine-disrupting chemicals. This function holds biological processes that are decisive for the fertility of women themselves

Background: Industrial progress has led to the omnipresence of chemicals in the environment of the general population, including reproductive-aged and pregnant women.

and for the health of future generations. However, insufficient research has evaluated the risk of combined mixtures on this function. This study aimed to assess the direct impacts of a realistic exposure to eight combined environmental toxicants on the critical process of folliculogenesis.
Methods: Female rabbits were exposed daily and orally to either a mixture of eight environmental toxicants (F group) or the solvent mixture (NE group, control) from 2 to 19 weeks of age. The doses were computed from previous toxicokinetic data to reproduce steady-state serum concentrations in rabbits in the range of those encountered in pregnant women. Ovarian function was evaluated through macroscopic and histological analysis of the ovaries, serum hormonal assays and analysis of the expression of steroidogenic enzymes. Cellular dynamics in the ovary were further investigated with Ki67 staining and TUNEL assays.
Results: F rabbits grew similarly as NE rabbits but exhibited higher total and high-density lipoprotein (HDL) cholesterol levels in adulthood. They also presented a significantly elevated serum testosterone concentrations, while estradiol, progesterone, AMH and DHEA levels remained unaffected. The measurement of gonadotropins, androstenedione, pregnenolone and estrone levels yielded values below the limit of quantification. Among the 7 steroidogenic enzymes tested, an isolated higher expression of Cyp19a1 was measured in F rabbits ovaries. Those ovaries presented a significantly greater density/number of antral and atretic follicles and larger antral follicles without any changes in cellular proliferation or DNA fragmentation. No difference was found regarding the count of other follicle stages notably the primordial stage, the corpora lutea or AMH serum levels.
Conclusion: Folliculogenesis and steroidogenesis seem to be subtly altered by exposure to a human-like mixture of environmental toxicants. The antral follicle growth appears promoted by the mixture of chemicals both in their number and size, potentially explaining the increase in atretic antral follicles. Reassuringly, the ovarian reserve estimated through primordial follicles number/density and AMH is spared from any alteration. The consequences of these changes on fertility and progeny health have yet to be investigated.

Authors: Sara El Fouikar, Nathalie Van Acker, Virginie Héliès, François-Xavier Frenois, Frank Giton, Véronique Gayrard, Yannick Dauwe, Laila Mselli-Lakhal, Delphine Rousseau-Ralliard, Natalie Fournier, Roger Léandri, Nicolas Gatimel
Full Source: Journal of ovarian research 2024 Jun 28;17(1):134. doi: 10.1186/s13048-024-01457-6.

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Silica nanoparticles triggered epithelial ferroptosis via miR-21-5p/GCLM signaling to contribute to fibrogenesis in the lungs

2024-06-27

The toxicity of silica nanoparticles (SiNPs) to lung is known. We previously demonstrated that exposure to SiNPs promoted pulmonary impairments, but the precise pathogenesis remains elucidated. Ferroptosis has now been identified as a unique form of oxidative cell death, but whether it participated in SiNPs-induced lung injury remains unclear. In this work, we established a rat model with sub-chronic inhalation exposure of SiNPs via intratracheal instillation, and conducted histopathological examination, iron detection, and ferroptosis-related lipid peroxidation and protein assays. Moreover, we evaluated the effect of SiNPs on epithelial ferroptosis, possible mechanisms using in vitro-cultured human bronchial epithelial cells (16HBE) cells, and also assessed the ensuing impact on fibroblast activation for fibrogenesis. Consequently, fibrotic lesions occurred in the rat lungs, concomitantly by enhanced lipid peroxidation, iron overload, and ferroptosis. Consistently, the in vitro data showed SiNPs triggered oxidative stress and caused the accumulation of lipid peroxides, resulting in ferroptosis. Importantly, the mechanistic investigation revealed miR-21-5p as a key player in the epithelial ferroptotic process induced by SiNPs via targeting GCLM for GSH depletion. Of note, ferrostatin-1 could greatly suppress ferroptosis and alleviate epithelial injury and ensuing fibroblast activation by SiNPs. In conclusion, our findings first revealed SiNPs triggered epithelial ferroptosis through miR-21-5p/GCLM signaling and thereby promoted fibroblast activation for fibrotic lesions, and highlighted the therapeutic potential of inhibiting ferroptosis against lung impairments upon SiNPs exposure.

The toxicity of silica nanoparticles (SiNPs) to lung is known.

Authors: Songqing Lv, Yan Li, Xueyan Li, Lingnan Zhu, Yurou Zhu, Caixia Guo, Yanbo Li
Full Source: Chemico-biological interactions 2024 Jun 27:111121. doi: 10.1016/j.cbi.2024.111121.

PHARMACEUTICAL/TOXICOLOGY

Repeated Silica exposures lead to Silicosis severity via PINK1/PARKIN mediated mitochondrial dysfunction in mice model

2024-06-27

Background and objectives: Silicosis, one of the occupational health illnesses is caused by inhalation of crystalline silica. Deposition of extracellular matrix and fibroblast proliferation in lungs are linked to silicosis development. Mitochondrial dysfunction plays critical role in some diseases, but how these processes progress and regulated in silicosis, remains limited. Detailed study of silica induced pulmonary fibrosis in mouse model, its progression and severity may be helpful in designing future therapeutic strategies.

Methods: In present study, mice model of silicosis has been developed after repeated silica exposures which may closely resemble clinical symptoms of silicosis in human. In addition to efficiently mimicking the acute/chronic transformation processes of silicosis, this is practical and efficient in terms of time and output, which avoids mechanical injury to the upper respiratory tract due to surgical interventions. Sonicated sterile silica suspension (120 mg/kg) was administered through intranasal route thrice a week at regular intervals (21, 28 and 35 days).

Results: Presence of minute to larger silicotic nodules in H&E-stained lung sections were observed in all silica induced model groups. Enhanced ECM deposition was noted in MT stained lung sections of silica exposure groups as compared to control which were confirmed by significantly higher MMP9 expression levels and hydroxyproline content in silica 35 days group. Increase in Reactive oxygen species (ROS), inflammatory cell recruitment mainly, neutrophils and macrophage were observed in all three silica exposure groups. Transmission electron microscopic analysis has confirmed presence of many aberrant shaped mitochondria (swollen, round shape) in 35 days model where autophagosomes were minimum. Western blot analysis of mitophagy and autophagy markers such as Pink1, Parkin, Cytochrome c, SQSTM1/p62, the ratio of light chain LC3B II/LC3B I was found higher in 21 and 28 days which were significantly reduced in 35 days silica model.

Conclusions: Higher MMP9 activity and MMP9 /TIMP1 ratio demonstrate excessive extracellular matrix damage and deposition in 35 days model. Significantly reduced expressions of autophagy and mitophagy markers

Background and objectives: Silicosis, one of the occupational health illnesses is caused by inhalation of crystalline silica.

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have also confirmed progression in fibrosis severity and its association with repeated silica exposures in 35 days model group.

Authors: Sneha Kumari, Payal Singh, Rashmi Singh
Full Source: Cellular signalling 2024 Jun 27:121:111272. doi: 10.1016/j.cellsig.2024.111272.

Longitudinal associations of air pollution and green space with cardiometabolic risk factor clustering among children in the Netherlands

2024-06-27

Background: This study examines longitudinal associations of air pollution and green space with cardiometabolic risk among children in the Netherlands.

Methods: Three Dutch prospective cohorts with a total of 13,822 participants aged 5 to 17 years were included: (1) the Amsterdam Born Children and their Development (ABCD) study from Amsterdam (n = 2,547), (2) the Generation R study from Rotterdam (n = 5,431), and (3) the Lifelines study from northern Netherlands (n = 5,844). Air pollution (PM2.5, PM10, NO2, and elemental carbon (EC)) and green space exposures (density in multiple Euclidean buffer sizes) from 2006 to 2017 at home address level were used. Cardiometabolic risk factor clustering was assessed by a MetScore, which was derived from a confirmatory factor analysis of six cardiometabolic risk factors to assess the overall risk. Linear regression models with change in Metscore as the dependent variable, adjusted for multiple confounders, were conducted for each cohort separately. Meta-analyses were used to pool cohort-specific estimates.

Results: Exposure to higher levels of NO2 and EC was significantly associated with increases in MetScore in Lifelines (per SD higher exposure: βNO2 = 0.006, 95 % CI = 0.001 to 0.010; βEC = 0.008, 95 % CI = 0.002 to 0.014). In the other two cohort studies, these associations were in the same direction but these were not significant. Higher green space density in 500-meter buffer zones around participants' residential addresses was not significantly associated with decreases of MetScore in all three cohorts. Higher green space density in 2000-meter buffer zones was significantly associated with decreases of MetScore in ABCD and Lifelines (per SD higher green space density: βABCD = -0.008, 95 % CI = -0.013 to -0.003; βLifelines = -0.002, 95 % CI = -0.003 to -0.00003). The pooled estimates were βNO2 = 0.003 (95 % CI = -0.001 to 0.006) for NO2, βEC = 0.003 (95 % CI = -0.001, 0.007) for EC, and β500m buffer = -0.0014 (95 % CI = -0.0026 to -0.0001) for green space.

Background: This study examines longitudinal associations of air pollution and green space with cardiometabolic risk among children in the Netherlands.
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Conclusions: More green space exposure at residence was associated with decreased cardiometabolic risk in children. Exposure to more NO2 and EC was also associated with increased cardiometabolic risk.
Authors: Mingwei Liu, Ilonca Vaartjes, Gerard Hoek, Vincent W V Jaddoe, Susana Santos, Anton Schreuder, Tanja G M Vrijkotte, Diederick E Grobbee, Erik J Timmermans
Full Source: Environment international 2024 Jun 27:190:108852. doi: 10.1016/j.envint.2024.108852.

Adverse impact of phthalate and polycyclic aromatic hydrocarbon mixtures on birth outcomes: a metabolome Exposome-Wide Association Study

2024-06-28
It has been well-investigating that individual phthalates (PAEs) or polycyclic aromatic hydrocarbons (PAHs) affect public health. However, there is still a gap that the mixture of PAEs and PAHs impacts birth outcomes. Through innovative methods for mixtures in epidemiology, we used a metabolome Exposome-Wide Association Study (mExWAS) to evaluate and explain the association between exposure to PAEs and PAHs mixtures and birth outcomes. Exposure to a higher level of PAEs and PAHs mixture was associated with lower birth weight (maximum cumulative effect: -143.5 g) rather than gestational age. Mono(2-ethlyhexyl) phthalate (MEHP) (posterior inclusion probability, PIP =0.51), 9-hydroxyphenanthrene (9-OHPHE) (PIP =0.53), and 1-hydroxypyrene (1-OHPYR) (PIP =0.28) were identified as the most important compounds in the mixture. In mExWAS, we successfully annotated four overlapping metabolites associated with both MEHP/9-OHPHE/1-OHPYR and birth weight, including arginine, stearamide, Arg-Gln, and valine. Moreover, several lipid-related metabolism pathways, including fatty acid biosynthesis and degradation, alpha-linolenic acid, and linoleic acid metabolism, were disturbed. In summary, these findings may provide new insights into the underlying mechanisms by which PAE and PAHs affect fetal growth.

It has been well-investigating that individual phthalates (PAEs) or polycyclic aromatic hydrocarbons (PAHs) affect public health.

Authors: Yiwei Fang, Wenjun Yin, Chao He, Qiuzi Shen, Ying Xu, Chunyan Liu, Yuanzhong Zhou, Guotao Liu, Yun Zhao, Huiping Zhang, Kai Zhao
Full Source: Environmental pollution (Barking, Essex : 1987) 2024 Jun 28:124460. doi: 10.1016/j.envpol.2024.124460.

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OCCUPATIONAL

Occupational Exposure Limits for Reproductive Toxicants - A Comparative Analysis

2024-06-26
We investigated the level of protection of reproductive and developmental toxicity offered through occupational exposure limits (OELs) and Derived No-Effect Levels for workers' inhalation exposure (wDNELs). We compared coverage of substances that have a harmonised classification as reproductive toxicant 1A or 1B (Repr.1A/B), numerical values and scientific basis of 12 lists of OELs and wDNELs from REACH Registrants' and the Committee for Risk Assessment. Across the 14 sources of OELs and wDNELs, 53% of the Repr1A/B-substances had at least one exposure limit (counting groups of metals as one entry). Registrants' wDNELs covered the largest share, 40%. The numerical values could be highly variable for the same substance across the lists. How often reproductive toxicity is identified as the critical effect varies between the examined lists, both due to different assessments of the same substance and different substance coverage. Reviewing the margin of safety to reproductive toxicity cited in the documents, we found that 15% of safety margins were lower to reproductive toxicity than the critical effect. To conclude, neither the REACH nor work environment legislation supply wDNELs or OELs for a substantial share of known reproductive toxicants. EU OELs cover among the fewest substances in the range, and in many cases national OELs or wDNELs are set at more conservative levels.

We investigated the level of protection of reproductive and developmental toxicity offered through occupational exposure limits (OELs) and Derived No-Effect Levels for workers' inhalation exposure (wDNELs).

Authors: Linda Schenk, Meng-Rung Ho, Piia Taxell, Pasi Huuskonen, Mimmi Leite, Inese Martinsone, Karl-Christian Nordby, Linda Paegle, Loreta Strumylaite
Full Source: Reproductive toxicology (Elmsford, N.Y.) 2024 Jun 26:108649. doi: 10.1016/j.reprotox.2024.108649.

Worse pulmonary function in association with cumulative exposure to nanomaterials. Hints of a mediation effect via pulmonary inflammation

2024-06-28
Background: Today, nanomaterials are broadly used in a wide range of industrial applications. Such large utilization and the limited knowledge on to the possible health effects have raised concerns about potential consequences on human health and safety, beyond the environmental burden. Given that inhalation is the main exposure route, workers exposed

to nanomaterials might be at risk of occurrence of respiratory morbidity and/or reduced pulmonary function. However, epidemiological evidence regarding the association between cumulative exposure to nanomaterials and respiratory health is still scarce. This study focused on the association between cumulative exposure to nanomaterials and pulmonary function among 136 workers enrolled in the framework of the European multicentric NanoExplore project.
Results: Our findings suggest that, independently of lifelong tobacco smoking, ethnicity, age, sex, body mass index and physical activity habits, 10-year cumulative exposure to nanomaterials is associated to worse FEV1 and FEF25 - 75%, which might be consistent with the involvement of both large and small airway components and early signs of airflow obstruction. We further explored the hypothesis of a mediating effect via airway inflammation, assessed by interleukin (IL-)10, IL-1β and Tumor Necrosis Factor alpha (TNF-α), all quantified in the Exhaled Breath Condensate of workers. The mediation analysis results suggest that IL-10, TNF-α and their ratio (i.e., anti-pro inflammatory ratio) may fully mediate the negative association between cumulative exposure to nanomaterials and the FEV1/FVC ratio. This pattern was not observed for other pulmonary function parameters.
Conclusions: Safeguarding the respiratory health of workers exposed to nanomaterials should be of primary importance. The observed association between cumulative exposure to nanomaterials and worse pulmonary function parameters underscores the importance of implementing adequate protective measures in the nanocomposite sector. The mitigation of harmful exposures may ensure that workers can continue to contribute productively to their workplaces while preserving their respiratory health over time.
Authors: Giulia Squillacioti, Thomas Charreau, Pascal Wild, Valeria Bellisario, Federica Ghelli, Roberto Bono, Enrico Bergamaschi, Giacomo Garzaro, Irina Guseva Canu
Full Source: Particle and fibre toxicology 2024 Jun 28;21(1):28. doi: 10.1186/s12989-024-00589-3

Asbestos exposure and asbestosis mortality in Italian cement-asbestos cohorts: Dose-response relationship and the role of competing death causes

2024-06-29

Objectives: In Italy, asbestos was used intensively until its ban in 1992, which was extended for asbestos cement factories until 1994. The aim of this study was to evaluate the dose-response between asbestos exposure and asbestosis mortality across a pool of Italian occupational cohorts, taking into account the presence of competing risks.
Methods: Cohorts were followed for vital status and the cause of death was ascertained by a linkage with mortality registers. Cause-specific (CS) Cox-regression models were used to evaluate the dose-exposure relationship between asbestosis mortality and the time-dependent cumulative exposure index (CEI) to asbestos. Fine and Gray regression models were computed to assess the effect of competing risks of death.
Results: The cohort included 12,963 asbestos cement workers. During the follow-up period (1960-2012), of a total of 6961 deaths, we observed 416 deaths attributed to asbestosis, 879 to lung cancer, 400 to primary pleural cancer, 135 to peritoneal cancer, and 1825 to diseases of the circulatory system. The CS model showed a strong association between CEI and asbestosis mortality. Dose-response models estimated an increasing trend in mortality even below a CEI of 25 ff/mL-years. Lung cancer and circulatory diseases were the main competing causes of death.
Conclusions: Asbestos exposure among Italian asbestos-cement workers has led to a very high number of deaths from asbestosis and asbestos-related diseases. The increasing risk trend associated with excess deaths, even at low exposure levels, suggests that the proposed limit values would not have been adequate to prevent disability and mortality from asbestosis.
Authors: Paolo Girardi, Sara Rigoni, Daniela Ferrante, Stefano Silvestri, Alessia Angelini, Francesco Cuccaro, Enrico Oddone, Massimo Vicentini, Francesco Barone-Adesi, Sara Tunesi, Enrica Migliore, Francesca Roncaglia, Orietta Sala, Roberta Pirastu, Elisabetta Chellini, Lucia Miligi, Patrizia Perticaroli, Vittoria Bressan, Enzo Merler, Danila Azzolina, Alessandro Marinaccio, Stefania Massari, Corrado Magnani
Full Source: American journal of industrial medicine 2024 Jun 29. doi: 10.1002/ajim.23629.

Objectives: In Italy, asbestos was used intensively until its ban in 1992, which was extended for asbestos cement factories until 1994.