dynamics were observed for all samples. These changes include an increase in the percentage of cells at G0/G1 phase accompanied by a decrease in cells at S and G2 phases. This suggests an arrest at G0/G1 phase where cells remain quiescent without replicating DNA.

According to these findings, PM_{10} collected in a university cafeteria induces toxicity in lung cells. Since the cafeteria is a popular space for socializing and having quick meals and snacks, it is important to conduct further research to fully understand the health impacts of these pollutants and take necessary measures to mitigate any negative outcomes.

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P21-48
Toxicity of subway related nanoparticles in human lung cell models

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Air pollution in cities poses a significant health risk and particulate matter is classified as a Group 1 carcinogen. Among the various pollutants, nanoparticles – often defined as particles smaller than 100 nm – are believed to play a critical role in toxicity due to their higher surface-to-volume ratio and reactivity compared to larger particles. Metal-containing nanoparticles can for instance participate in Fenton-like reactions, leading to oxidative stress and DNA damage. Subways are a major source of airborne metal-containing particles in urban areas. Particles from the subway have previously been suggested to be more toxic than particles from tire wear, road dust, or wood combustion. However, research on the specific role of nanoparticles in subway particle toxicity remains limited.

In our study, which is part pf the European nPETS project that focuses on understanding toxicity and health effects of nanoparticles from different transport modes [1], we compared the toxicity of nanoparticles from different subway materials including rails, wheels and third rail. All particles were generated in the laboratory from electrodes of the same material as in the Stockholm subway system with a spark discharger. The respective particles, along with nanoparticles generated from iron electrodes as a reference, were thoroughly characterized regarding elemental composition, size and form. By using a human lung cell model (A549) and monocyte-derived macrophages (dTHP-1), the subway related nanoparticles were tested for cytotoxicity, genotoxicity, oxidative potential as well as inflammation response by different approaches including the alkaline comet assay, real-time quantitative PCR and MSD multiplexing.

The results showed that the nanoparticles mainly consisted of iron and had a primary size of 7–10 nm but formed agglomerates. Only low cytotoxic effects were observed in the doses tested (10–200 μg/mL), but a clear and concentration-dependent increase in DNA damage was noted in both cell lines investigated. Also, increased expression of the pro-inflammatory cytokine *IL-8* was noted upon exposure of dTHP-1 cells, but not in A549. However, no or less clear changes were noted for the other genes tested (*IL-1β, IL-6, TNF-α, HMOX, GADD45*). No changes in release of cytokines (IL-8, IL-6, TNF-α, IL-1β) were observed and none of the nanoparticles tested led to an increase in acellular ROS formation. Overall, we could only find minor differences in the toxicity between these nanoparticles. Taken together, our results indicate relatively low cytotoxicity and inflammatory potential of agglomerated nanoparticles of subway-related materials but a dose-dependent increase in DNA damage was observed.

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P21-49
Airway impedance in Mexican children exposed to artisanal brick kiln fumes

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Background: During artisanal brick production, a large amount of gases such as carbon monoxide, nitrogen oxides, sulfur dioxide and solid particles less than 10 and 2.5 microns are emitted into the atmosphere. Artisanal brick kilns are particularly common in certain marginalized areas in Central Mexico, posing a real health threat for inhabitants near communities, especially for children.

Purpose: To measure airway resistance and reactance in children exposed to artisanal brick kiln fumes and to compare results in the same parameters in non-exposed children (control group).

Methods: We recruited children from 6 to 12 years old, both genders, exposed and not exposed to artisanal brick kiln fumes. Clinical and sociodemographic history as well as frequency of respiratory symptoms were obtained through a validated questionnaire. Airway impedance was evaluated by pulse oscillometry with the Vmax® Encore system oscillometer following quality criteria of the American Thoracic Society (ATS).

Results: 84 non-asthmatic children were included; 25 (29.8%) of them were exposed to artisanal brick kiln fumes (exposed group), while 59 (70.2%) were non-exposed (control group). The exposed group showed a higher frequency of allergic rhinitis (OR=4.6, 95% CI: 1.02–21.3). Also, we observed that resistance at 5 Hz (R5Hz) and 20 Hz (R20Hz) were significantly higher in exposed children (p=0.04). Results from a generalized linear model showed that, after adjusting for tobacco smoke and wood smoke exposure, variable "place of residence" was the factor most associated with oscillometry alteration, since living near an artisanal brick kiln increased between 4 to 15% R5 Hz parameter.

Conclusions: Artisanal brick kiln fumes exposure in children aged 6 to 12 was associated with airway impedance alterations. Proximal and peripheral airway resistance is significantly higher in exposed children. Children exposed to artisanal brick kiln fumes present 4.3 times greater risk for presenting proximal airway obstructive alterations.

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