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Multi-level approach for the study of air pollution impact on health

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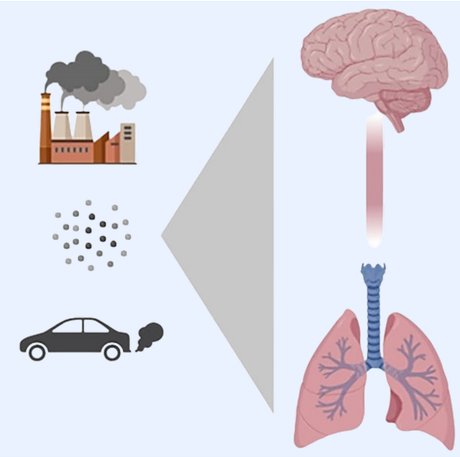
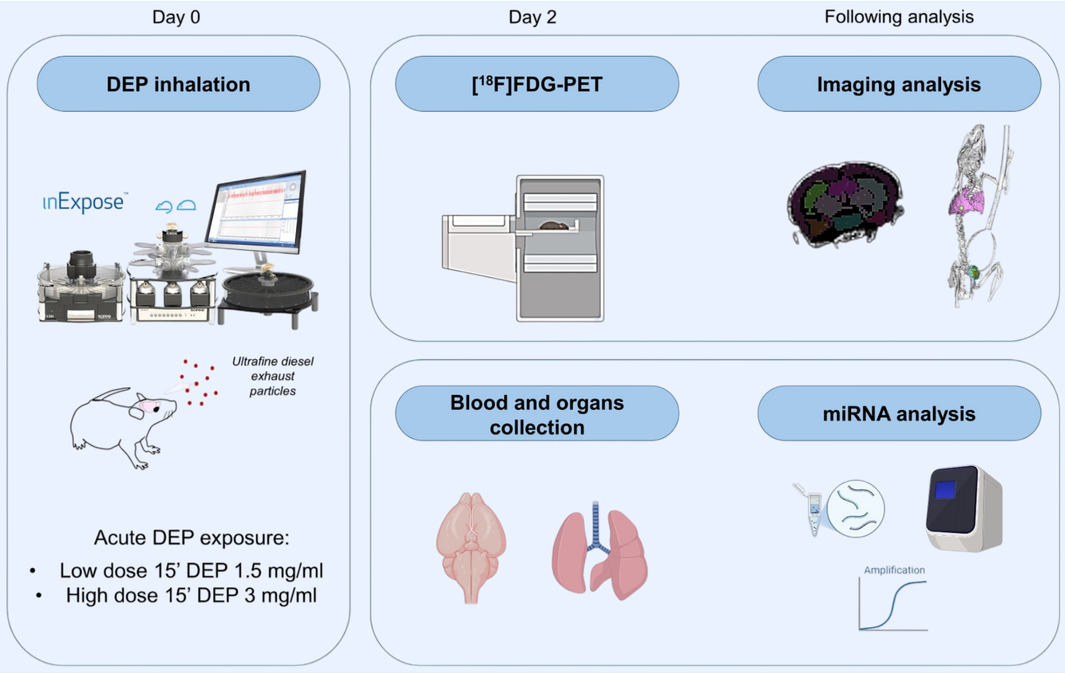
BACKGROUND

Air pollution represents a global public health emergency affecting individual of all ages. Among its constituents, Diesel exhaust particles (DEP) - a major component of traffic-related air pollution - have been identified as key factors for neuroinflammation and oxidative stress. We exposed healthy animals to DEP in acute manner as an animal model to assess air pollution effects in lungs and brain, in particular PM 2.5, using physiological exposure and advanced in vivo imaging as Positron Emission Tomography (PET) and epigenetic analysis.



METHODS



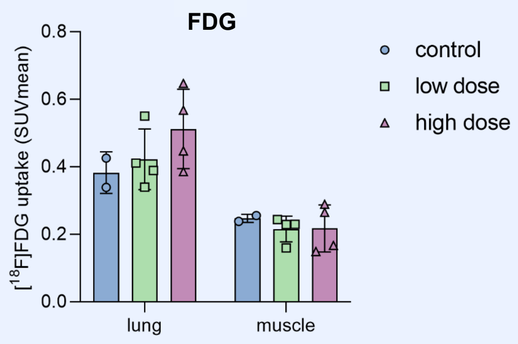
Healthy male C57Bl/6 mice (n=10) divided in:

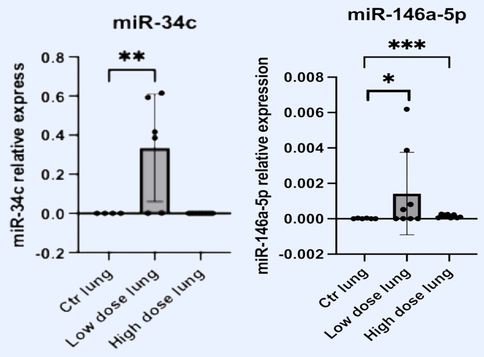
- **Control group** (saline, n=2)
- **Low-dose group** (1,5 mg/ml, n=4)
- **High-dose group** (3 mg/ml, n=4)

At day 0 → mice were exposed to a single, nose-only dose of DEP for 15 minutes
At day 2 → animals underwent whole-body PET/CT imaging with [18F]FDG, followed by sacrifice for blood and organ collection.

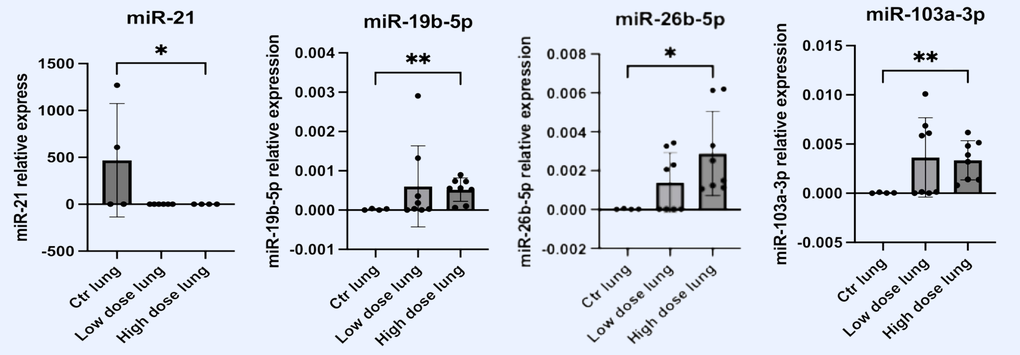
RESULTS

Lung

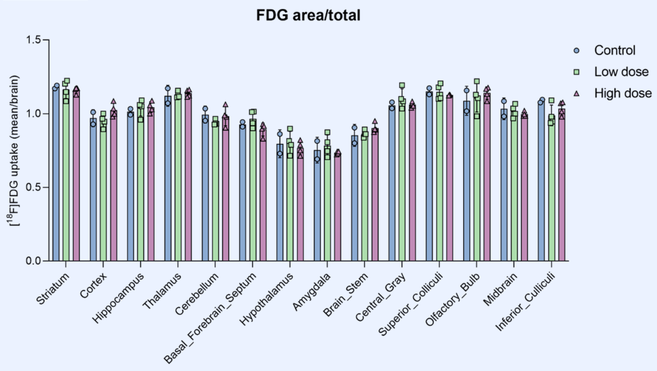




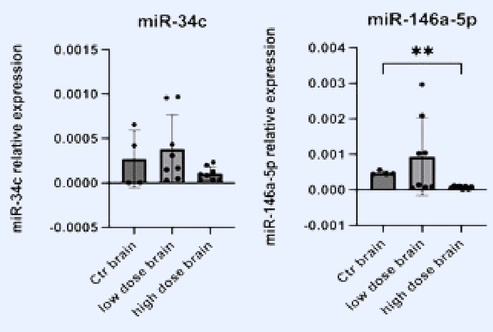
miRNA analysis



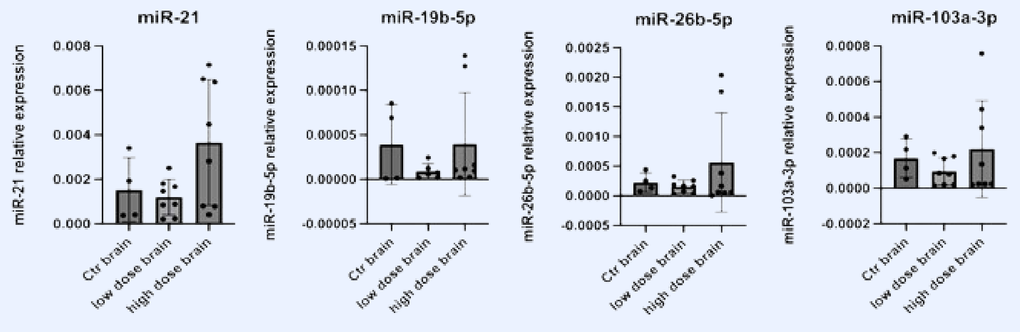
Brain



Low-dose miRNA



High-dose miRNA



Mice exposed to DEP showed an increased FDG uptake in lung compared to the controls that would appear to be dose dependent.

Alteration in microRNA profiling was observed in both brain and lung tissue, highlighting a role of miRNA involved in inflammatory pathways (inflamma-miR) in the response to DEP exposure

CONCLUSIONS

This preliminary study represents a model for multi-level investigations into environmental health risks, exploring the systemic impact of DEP exposure in healthy and disease-relevant mouse models, exploiting a physiological pollutant administration route (inExpose® system) and an in vivo molecular imaging combined with epigenomic analysis for the readout. Future studies will require an expanded cohort to validate these findings. Notably, a potential correlation has emerged between miRNA expression levels and genes associated with oxidative stress and inflammation—an area that warrants further investigation, particularly in light of the two different exposure doses used.

AKNOWLEDGMENTS

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