Exposure to fine particulate matter ($PM_{2.5}$) has been known for years for its adverse effects on human health[1]. Depending on $PM_{2.5}$ origin, chemical composition and adverse health effects can vary[2]. Short term mechanisms like oxidative stress, inflammation and metabolization can be induced by $PM_{2.5}$ exposure[3], but chronic mechanisms like epithelial to mesenchymal transition (EMT) are rarely evaluated. The aim of this work is to chemically characterize $PM_{2.5}$ from distinct origins and to study the toxicological potency of $PM_{2.5}$ on pulmonary cells in order to establish the link between specific particle constituents with specific cell responses. This study could explain in depth mechanisms involved in pathologies like asthma, COPD or lung cancer metastasis.

$PM_{2.5}$ were collected at four sites influenced by urban, industrial, road traffic, and port and maritime traffic emissions. $PM_{2.5}$ from all four sites were extensively chemically characterized. Results highlight clear content differences in the four influences.

A549 and BEAS-2B lung cells were then exposed to organic and water-soluble fractions of $PM_{2.5}$ from the four influences. *In vitro* cytotoxicity and viability were studied using lactate dehydrogenase and CellTiter-Glo® (Promega) assays, by exposing cells up to 100 µg/cm² for 24, 48 and 72 hours. No significant cytotoxic effect was observed but cells viability was altered.

Oxidative stress and antioxidant response were evaluated with GSH/GSSG-Glo™ (Promega) assay and RT-qPCR of antioxidant enzymes. Pro-inflammatory cytokines were studied, notably IL-1β and activation of the inflammasome by Caspase-Glo® 1 Inflammasome Assay (Promega). Cytochromes P450 were analyzed by RT-qPCR and their activities was quantified by P450-Glo™ (Promega) assay. Depending on $PM_{2.5}$ origin, oxidative stress, inflammation and metabolization responses vary. Industrial and road traffic influences induced the most important responses.

EMT was evaluated on A549 cells after 7 days of exposure with cells morphology, as well as protein quantification of epithelial and mesenchymal markers. Results show highest EMT responses under road traffic influence.

[1] Loomis, D. et al. The Lancet. Oncology 14, 1262–1263 (2013).
[2] Thomson, E. M. et al. Particle and Fibre Toxicology 12, 24 (2015)
[3] Leikauf, G. D., et al. Experimental & Molecular Medicine 52, 329-337 (2020)