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Background

- Particulate matter (PM) air pollution has long been recognized as a threat to human health, causing both short and long term adverse effects.
- The aim of this work, which is part of the MAPEC_LIFE (Monitoring Air Pollution Effects on Children for supporting public health policy) study, was to evaluate the **toxicity of polycyclic aromatic hydrocarbons (PAHs) and nitrated-PAHs in ultra-fine air particulates (PM_{0.5})** collected in four different areas in Perugia (Italy) during winter 2014 and spring 2015.

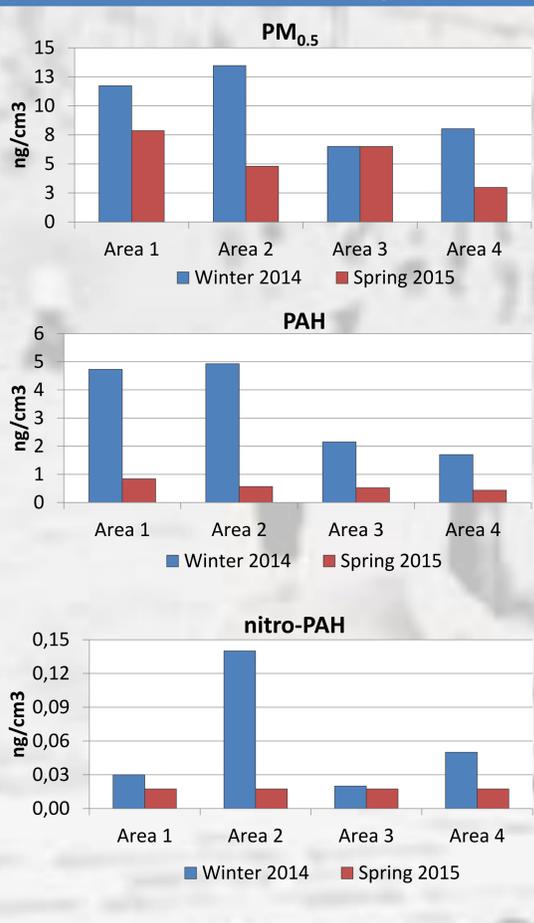
Methods & Results

- **PM_{0.5}** was collected on glass fibre membranes using a high-volume air sampler (Fig.1). After 72 hours, the membranes were subjected to chemical analysis to measure the concentration of **PAHs** and **nitro-PAHs**.
- Chemical analysis showed that the concentration of PM_{0.5}, PAHs and nitro-PAHs levels were higher in winter samples than in the spring ones (Fig.2).



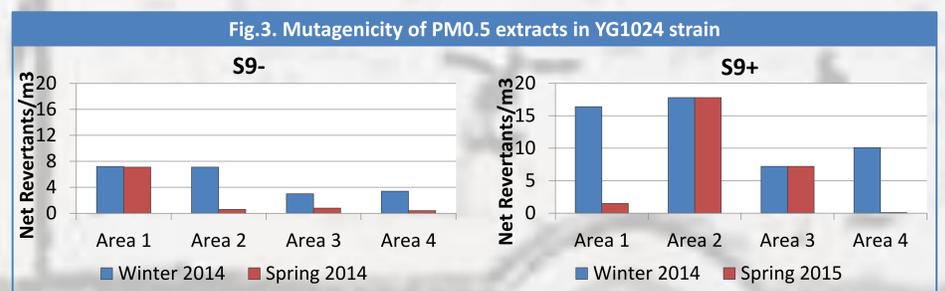
Fig.1. High-volume air sampler

Fig.2. Concentration and chemical composition of PM_{0.5} collected in Perugia.

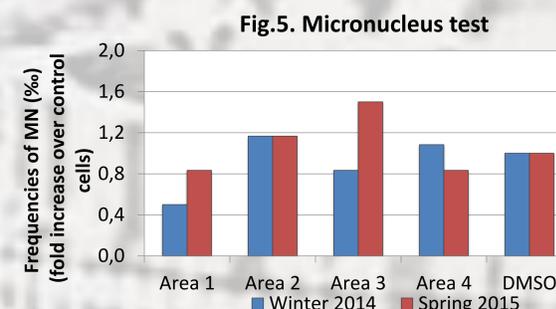
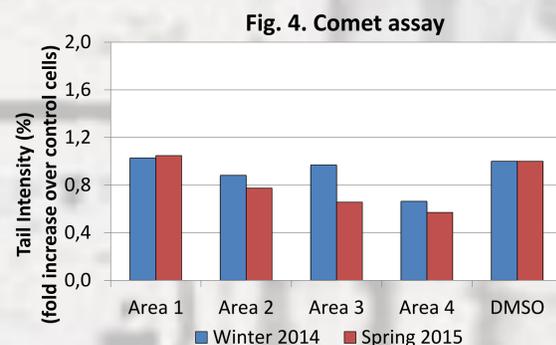


Methods & Results (cont.)

- PM_{0.5} organic extracts (50 m³) were analysed for their ability to cause **mutagenicity** on four different *S. typhimurium* strains (**Ames test**).
- Mutagenic effects were observed only in the YG1024 strain, with an increased activity when the S9 fraction was used, thus indicating the presence of **promutagens** (Fig.3)



- The DNA-damaging effects of PM_{0.5} were evaluated in a lung adenocarcinoma (A549) cell line by means of the comet assay and the cytokinesis-block micronucleus test.
- No genotoxicity was observed in A549 cells following exposure to 50 m³ of PM_{0.5} extracts collected in winter and spring (Fig. 4 and 5)



Discussion

- Our results indicated that the level of air pollution was higher in winter than in spring, as expected;
- PM_{0.5} extracts showed moderated mutagenic effects in the Ames test;
- The absence of genotoxicity, as evaluated in the comet and micronucleus assay, may be due to the lack of sensitivity of A549 lung adenocarcinoma cells;
- Further analysis will be performed using human primary lung cells.

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