# P019- CYTOTOXICITY AND MUTAGENICITY OF PARTICULATE MATTER FROM DOMESTIC ACTIVITIES

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Considerable amounts of particulate matter (PM) are produced indoors while people are cooking, cleaning, ironing or heating (Schiavon et al. 2015). Since people spend most of their time indoors, which promote the exposure to indoor air pollutants from a short distance, indoor PM is extremely important due to its possible side effects on health (Zhang et al. 2017). The organic constituents, in particular, polycyclic aromatic hydrocarbons (PAHs) and their derivatives, have been linked to carcinogenic and mutagenic effects (Kamal et al. 2015). The aim of this study was to evaluate the cytotoxic and mutagenic potential of particulate matter below 10 μm (PM10) obtained in the emissions from cooking and ironing activities.

Field measurements were conducted to collect PM10 samples released from frying horse mackerel, stuffing chicken, grilling and frying boneless pork strips, and from ironing at different conditions. The cytotoxicity of the PM10 total organic extracts was assessed using the WST-8 and the LDH assays. The A549 cell line, representative of the alveolar type II pneumocytes of the human lung, was used. The mutagenicity of the PM10-bound polycyclic aromatic hydrocarbons was screened through the Ames test, using S. typhimurium TA98 and TA100 strains with and without metabolic activation by the S9 fraction (rat liver microsomal fractions).

PM10 organic extracts from cooking and ironing showed a significant decrease ($p < 0.05$) of the metabolic activity of A549 cells. The highest significant decrease was observed for the PM10 organic extract from grilling boneless pork strips (decrease to 73 ± 5%) and from steam ironing-normal ventilation (decrease to 59 ± 3%), for cooking and ironing samples, respectively, at the highest concentration (150 µg mL-1). No significant differences were observed in the release of the cytoplasmic enzyme LDH into the culture supernatant, both for cooking and ironing PM10 organic extracts.

The results from the Ames test revealed that all the PAH samples presented ratios below 2 (twofold principle of mutagenicity confirmation) for the TA100 strain with and without metabolic activation. For the TA98 strain without metabolic activation, ratios above 2 were achieved for all the PAH cooking samples. However, when the S9 fraction was introduced into this strain, the mutagenic effect disappeared, suggesting that these samples lost their mutagenicity after being metabolised.

Altogether, our results show that PM10 organic extracts from cooking and ironing activities affect the metabolic activity of A549 cells but not their cell membrane integrity. Total PAHs from cooking samples presented mutagenic response only with strain TA98 in the absence of the S9 mixture, suggesting that the direct frameshift mutagenic activity was more prominent than base pair substitutions.

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