

**EFFECTS OF WASTE INCINERATOR FLY ASH ON
PROINFLAMMATORY RESPONSES OF PULMONARY
MACROPHAGES AND EPITHELIAL CELLS**

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Anthropogenic emissions including fly ash from combustion processes contribute to particulate air pollution which is under suspicion to threaten public health. Fly ash from a municipal waste incinerator was selected as a model for potential constituents of atmospheric particulate matter PM₁₀ or PM_{2.5} to identify parameters relevant for the induction of toxic processes in the target cells of the lung.

For *in vitro* studies rat alveolar macrophages (NR8383) and human bronchial epithelial cells (BEAS-2B) were incubated with various concentrations of incinerator fly ash (IFA) up to 200 µg/ml resuspended in medium and compared to its soluble and insoluble fractions as well as to quartz as positive control. The supernatant medium was analyzed for TNF-α, IL-6 and IL-8 as markers for proinflammatory responses and the viability of the cell was assayed by the MTT test. The effects were also studied in LPS activated macrophages and TNF-α stimulated epithelial cells.

IFA and its insoluble fraction were shown to induce cytotoxicity and cytokine release in a dose-dependent manner. Quartz caused similar effects compared to IFA in NR8383 but was less effective in BEAS-2B. The insoluble fraction was nearly ineffective. The LPS induced TNF-α release from macrophages was amplified by IFA and its insoluble fraction as well as the TNF-α induced IL-6 release from BEAS-2B cells. The TNF-α induced IL-8 release from BEAS-2B was not affected.

These results suggest that *in vitro* exposure to IFA adversely effects cell viability, induces the release of proinflammatory cytokines and modulates cell functions. The insoluble fraction of IFA was much more effective than the soluble fraction, however, the causing agent of this model fly ash has yet to be detected.