Long-term exposure to $PM_{2.5}$ from biomass combustion in vitro induces stress proteins and reduced metabolism in human bronchial epithelial cells (BEAS-2B)

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The combustion of biomass results in the emission of particulate matter (PM), which can negatively affect the air quality. The defense system of the human body can deal with short-term exposure of PM, however long-term exposure seems to overextend the defense system and increases the risk of cardiovascular and respiratory diseases as well as lung cancer [1].

In the present study, we have exposed human Beas-2B lung epithelial cells over a period of 60 days to $PM_{2.5}$ in a concentration of 100μg/ml to characterize the resulting cellular effects. PMs were obtained from fly ash during the burning of wood chips. Compared to the untreated control cells, the cell number significantly decreased upon exposure to $PM_{2.5}$ until complete loss was visible. We observed that the AMP-kinase was activated by $PM_{2.5}$ indicating a decreased energy status. Correspondently, the eukaryotic elongation factor 2 (eEF-2) was phosphorylated - a sign that translation of proteins may be affected. Moreover, the inducible stress kinase p38 was activated. As a further indication for the stress response of the cells translocation of the anti- oxidative transcription factor Nrf-2 was observed combined with an expression of the target stress response protein hemeoxygenase- 1 (HO-1) and an increased GSH- content. Based on these results it can be assumed that lung cells firstly try to combat PM induced damage, which finally results in cell loss probably due to autophagy which has to be confirmed by further experiments.

Our results indicate that long-term exposure to PM may cause lasting damage to lung epithelial cells, which can possibly impair the barrier function and the regeneration potential of the epithelial tissue.

[1] Thurston *et al*. (2002) JAMA **287**, 1132-1141.