# $PM_{2.5}$ from biomass combustion induces stress related and antioxidant proteins in THP-1 macrophages

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The emission of particulate matter (PM) from biomass combustion can negatively affect human health. Macrophages serve as an essential line of defense against PM that penetrates deep into the lungs. To investigate the effect of PM from biomass combustion, cell cultures of PMA-differentiated THP-1 macrophages were exposed to $PM_{2.5}$ obtained from combustion of wood chips (mainly softwood). Major components of this PM are potassium sulfate, calcium minerals and amorphous silicon-/aluminum-rich substances.

$PM_{2.5}$ was able to activate the stress-dependent kinases p38 and JNK. Activation of p38 kinase led to the nuclear translocation of the transcription factor Nrf-2, which was prevented by the p38 inhibitor LN950. Induction of Nrf-2 is known to result in an increase of the antioxidant capacity of cells and the synthesis of stress response proteins such as heme oxygenase-1 (HO-1) [1]. Accordingly, we demonstrated that $PM_{2.5}$ also induced the synthesis of HO-1 by a p38 dependent mechanism. The exposure of THP-1 cells to $PM_{2.5}$ only resulted in a slight increase of intracellular oxygen radicals (ROS). This moderate increase was able to activate JNK in a negative feedback mechanism because the antioxidant buthylhydroxyanisole reversed the observed induction of JNK.

Our results show that $PM_{2.5}$ emitted during softwood combustion only havs a slight potential to stimulate the formation of aggressive ROS in cells because cells may induce a cytoprotective mechanism by increasing the redox potential via the p38/Nrf-2 pathway. Therefore, it can be concluded that short-term exposure to $PM_{2.5}$ from this type of biomass combustion does not cause serious health effects.

[1] Gorrini *et al.* (2013) *Nat.Rev.Drug Discov* **12**, 931-47.