Toxicity of wood smoke particles in human lung epithelial cells: the role of PAHs, soot and zinc

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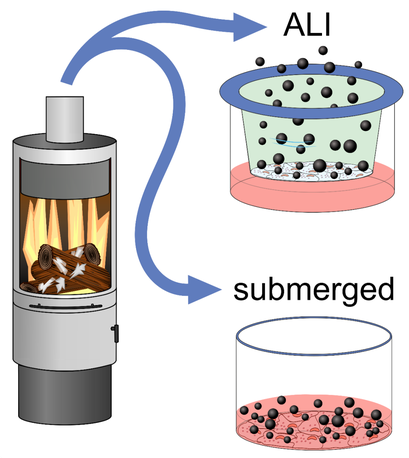
Background

The health impact of emissions from domestic burning of biomass and coal is estimated to contribute to over 4 million premature deaths per year worldwide [1]. Wood is the main fuel source for biomass combustion and the shift towards renewable energy sources will further increase emissions from wood combustion even in developed countries [2].

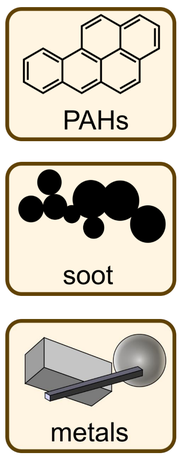
However, little is known about the constituents of wood smoke and biological mechanisms responsible for adverse health effects.

Methods



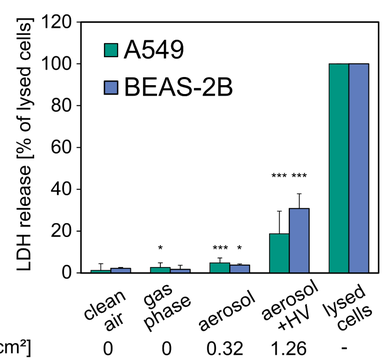
We exposed human A549 and BEAS-2B lung epithelial cells to freshly generated wood smoke at the air-liquid-interface (ALI) using a continuous flow exposure system [3].

Toxicity at ALI was compared to submerged exposure with collected wood smoke particles (WSP). To address critical constituents of WSP, we exposed A549 cells under submerged conditions individually to representative substances for PAHs, soot and metals: Benzo[a]pyrene (B[a]P), Carbon black (CB14) and ZnO Nanoparticles (ZnO) [2].

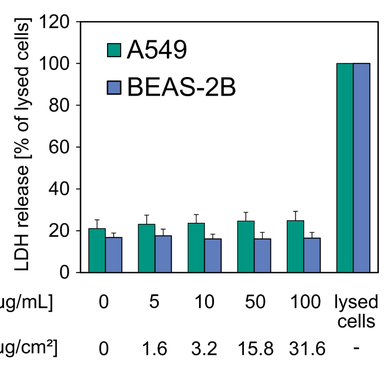


Toxicity of wood smoke at the air-liquid interface compared to submerged exposure

A



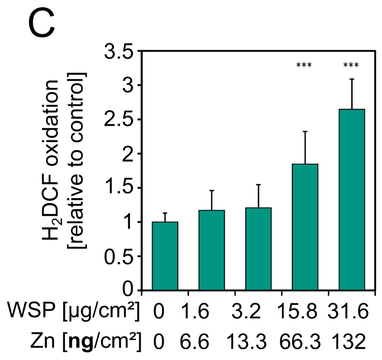
B



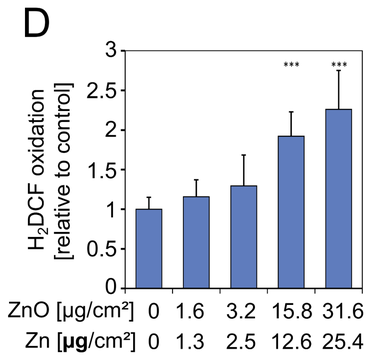
At the ALI, in both cell lines, the 1:10 diluted wood smoke did not induce cell death after 4h exposure. However, enhancing the particle dose by use of an electrostatic field (+HV), led to an increase of toxicity (**A**). Under submerged conditions, even particle doses greatly exceeding the toxic dose at the ALI provoked no signs of acute toxicity after 24 h exposure (**B**).

Contribution of PAHs, soot and metals to wood smoke particle toxicity

C

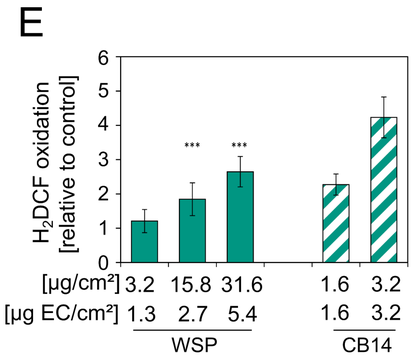


D

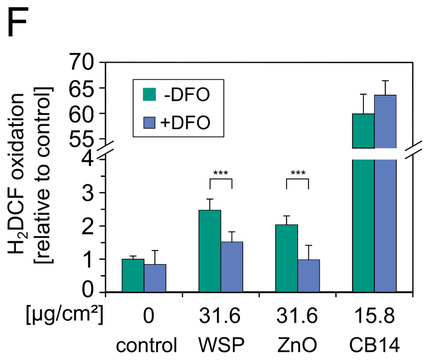


Formation of reactive oxygen species (ROS) in A549 cells after 3h submerged exposure. WSP induced cellular ROS (**C**). ZnO increased ROS formation only at high concentrations, which are well beyond the levels of Zn present in the tested WSP samples (**D**).

E

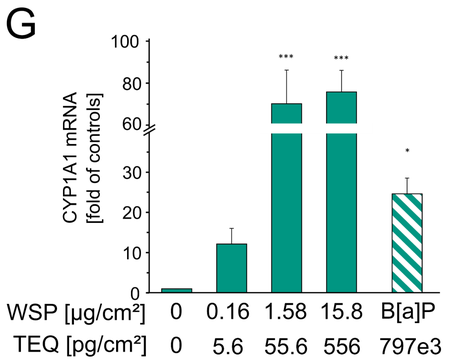


F



In A549 cells CB14 potently induced H_2DCF oxidation, in comparable magnitude to WSP when EC content is used as dose metric (**E**). The use of a metal chelator (DFO) decreased ROS formation by WSP, indicating a contribution of other metals than Zn (**F**).

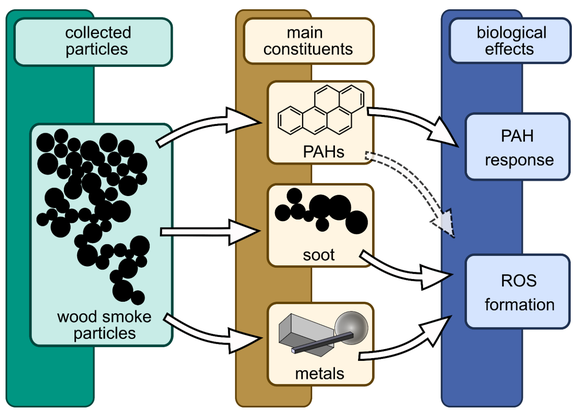
G



PAHs adsorbed to WSPs were much more potent in activating PAH target gene expression in A549 cells than B[a]P individually applied in suspension (4h submerged exposure) (**G**).

Conclusion

- WSP lead to cell death at the air-liquid-interface, but not under submerged conditions
- Mechanistic investigations employing submerged cell culture methods indicate a critical role of soot, metals and especially PAHs for hazardous effects of WSP
- The toxicological responses to wood smoke at the air-liquid-interface should be evaluated in more detail



Downloads

Poster, Abstract & Publications

References

[1] World Health Organization. "Indoor air pollution and health." Fact sheet N292 (2014).
[2] Dilger et al. "Toxicity of wood smoke particles in human A549 lung epithelial cells: the role of PAHs, soot and zinc." Archives of toxicology 90.12 (2016): 3029-3044.
[3] Mülhopt, Dilger et al. "Toxicity testing of combustion aerosols at the air–liquid interface with a self-contained and easy-to-use exposure system." Journal of Aerosol Science 96 (2016): 38-55.