

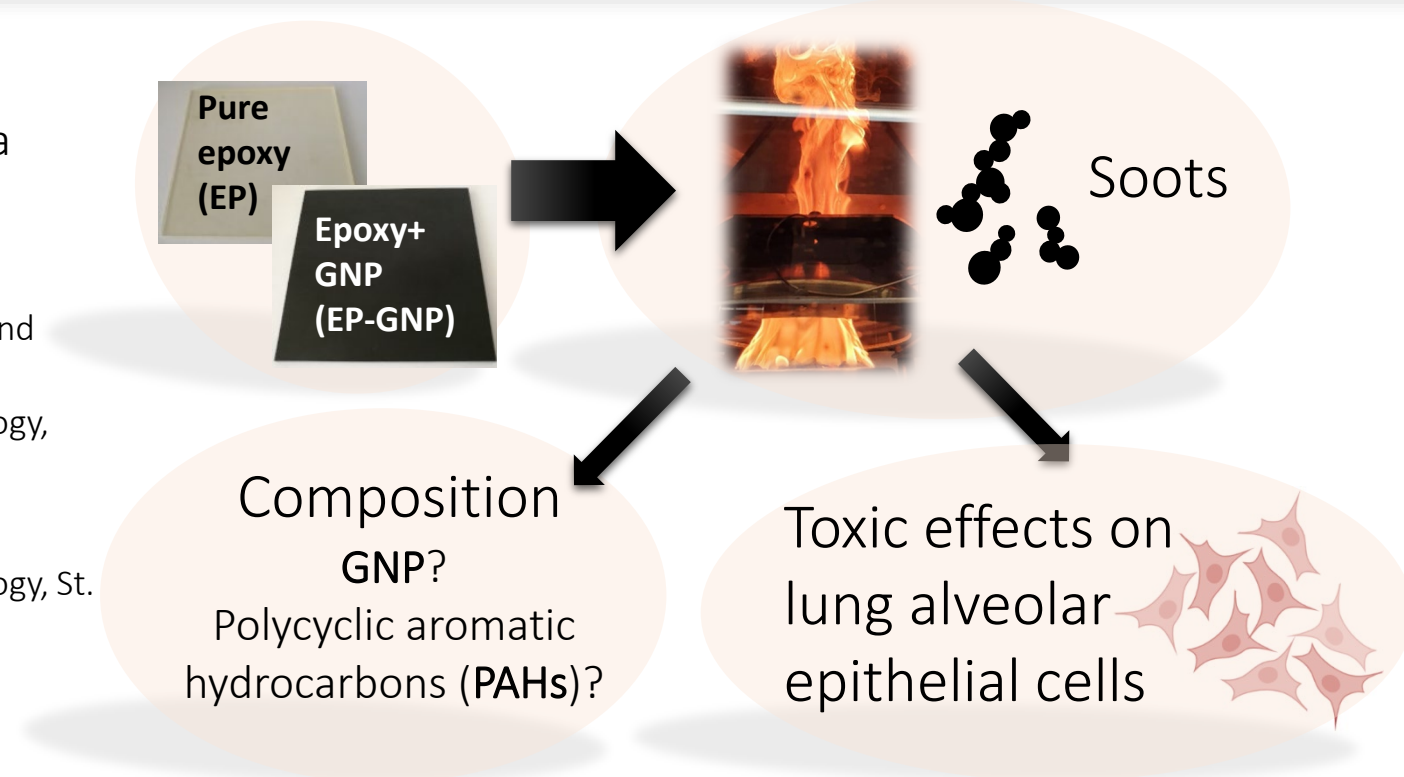
In vitro toxicity of airborne emissions from combustion of graphene nanoplatelet (GNP)-enabled epoxy nanocomposites

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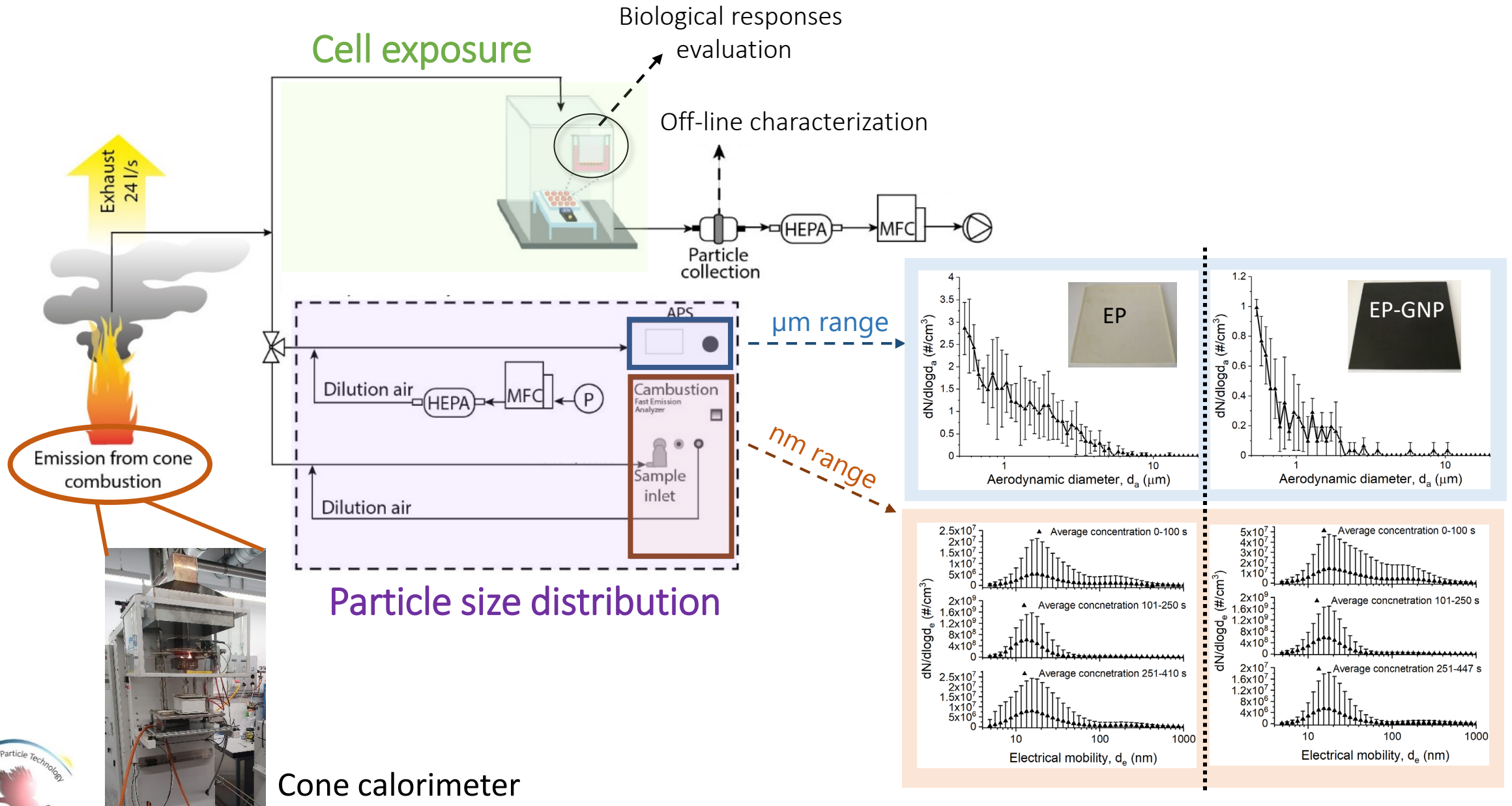
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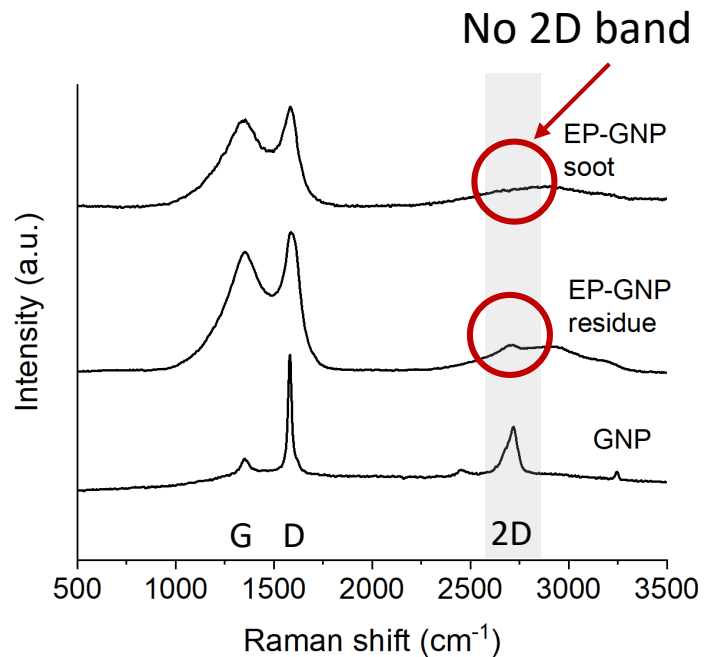
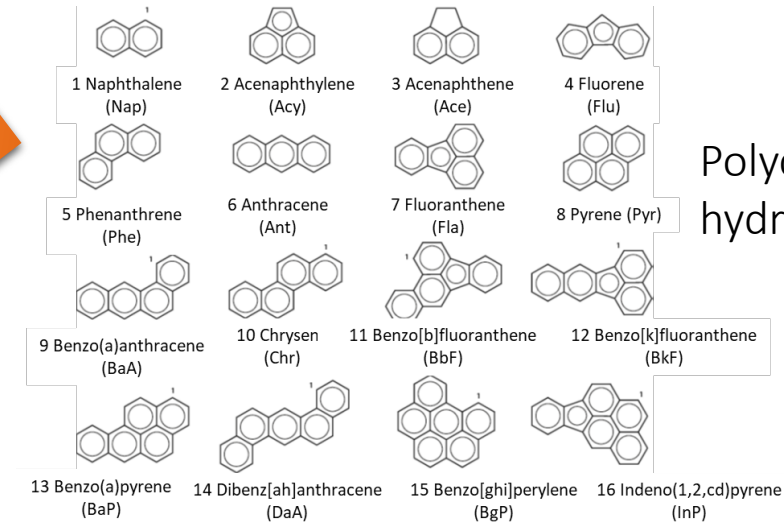
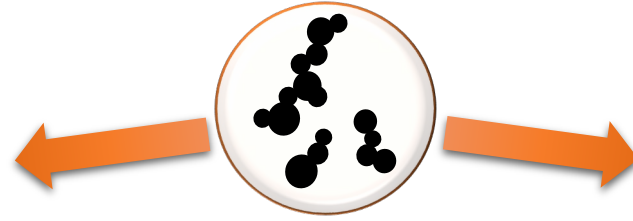


Combustion experiment

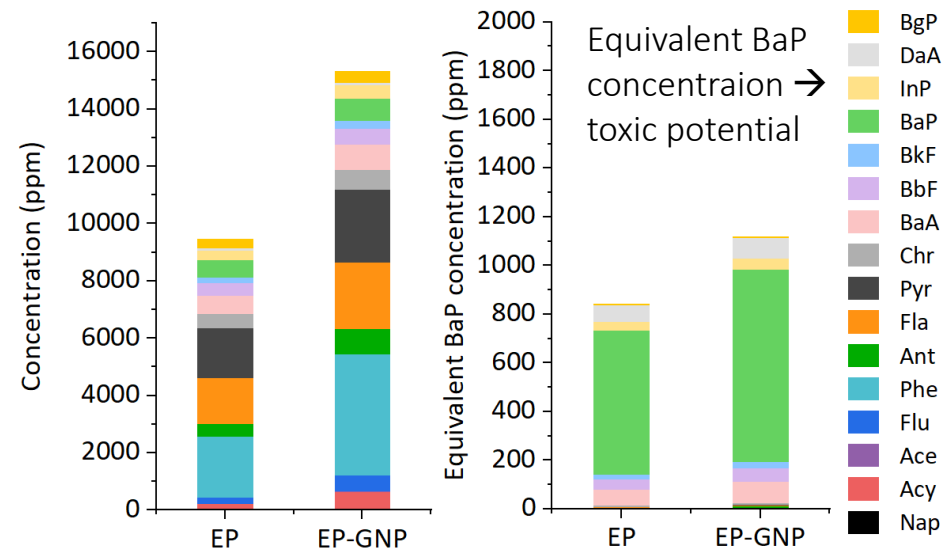


Soot characterization

Release of GNP into the airborne fraction?



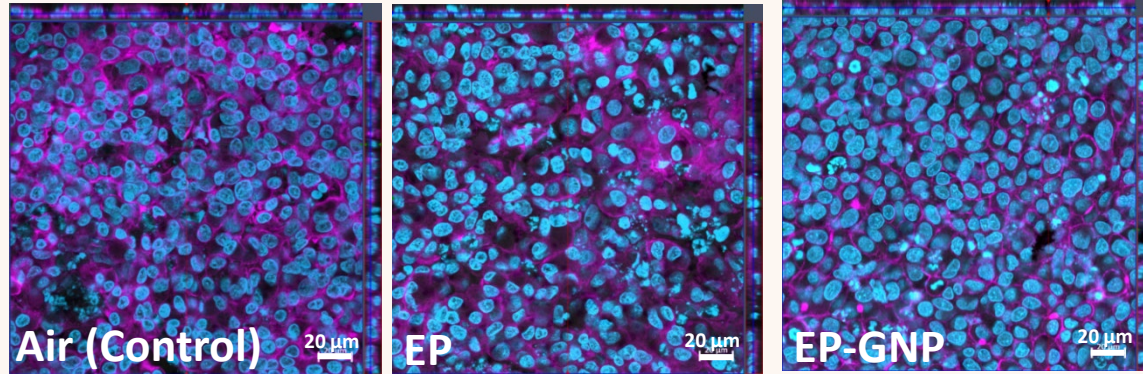
Absence of GNP in soots



Elevated PAH concentrations in EP-GNP soots

Biological responses on lung alveolar epithelial cells (A549)

No change in Cell morphology

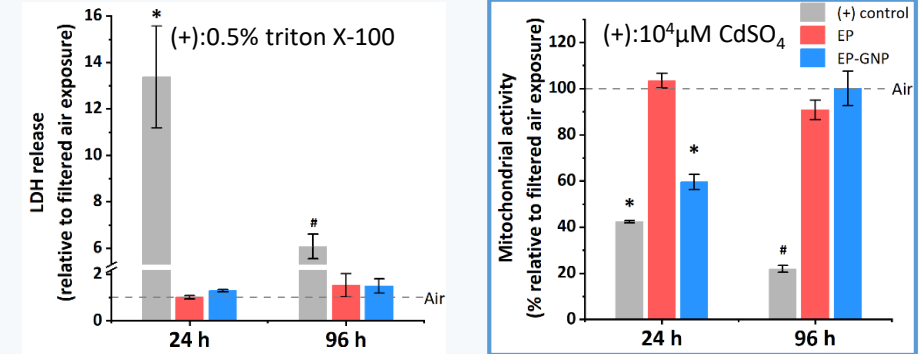


24 h post-exposure

Blue-DAPI

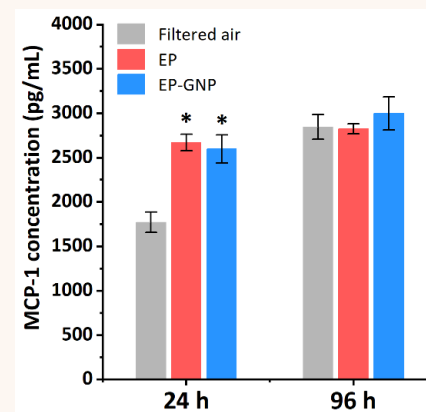
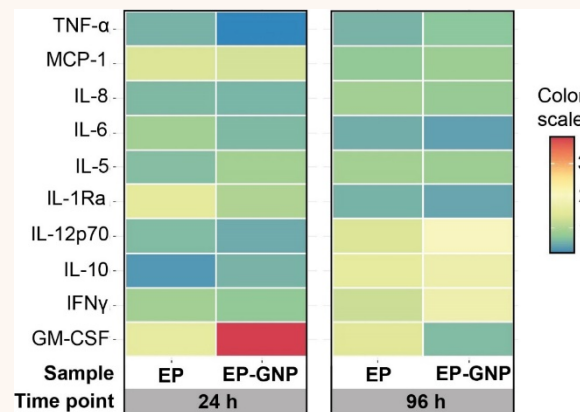
Magenta: Alexa488

Cell viability (LDH and MTS)



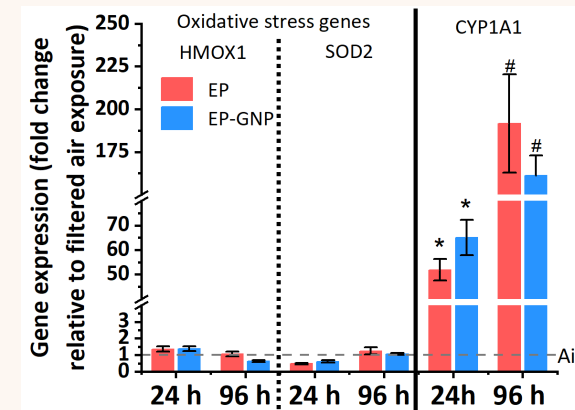
EP-GNP reduced mitochondrial activity.

Pro-(inflammatory) responses – 10 Cytokines



Both EP and EP-GNP induced release of MCP-1.

Oxidative stress and aryl hydrocarbon receptor (AhR)



Oxidative stress-related genes

- Neither EP nor EP-GNP affected expression of HMOX1 and SOD2.

AhR (detoxification of xenobiotics)

- Both EP and EP-GNP caused upregulation of CYP1A1 genes.

Conclusions

- No GNP was detected in the airborne fraction emitted from the combustion of EP-GNP, while GNP was found in the residual ash.
- GNP as nanofiller enhanced PAH concentration of the particles released from the combustion of the epoxy composite.
- Aerosol emission from EP-GNP combustion induced a reduction in mitochondrial activity of alveolar epithelial cells at 24 h after exposure, while EP did not.

Outlook

- Implementation of FTIR to the combustion platform – online measurement of toxic gases ex. SO_2 and NO_x

