Bern University of Applied Sciences

adolphe merkle institute
excellence in pure and applied nanoscience

UNI FR
UNIVERSITÉ DE FRIBOURG
UNIVERSITÄT FREIBURG

Hazard assessment of gasoline direct injection engine exhaust directly exposed onto the surface of a 3D human lung model

Christoph Bisig[1], Pierre Comte[2], Andreas Mayer[3], Jan Czerwinski[2], Alke Petri-Fink[1], Barbara Rothen-Rutishauser[1]

[1]Adolphe Merkle Institute (AMI), University of Fribourg, Switzerland; [2]Bern University for Applied Sciences (UASB), Switzerland; [3]Technik Thermischer Maschinen (TTM), Switzerland

Introduction

Gasoline direct injection (GDI) engines are increasingly used, due to their greater power, better fuel efficiency, and lower CO_2-emissions, though with the drawback of emitting more (nano)particles than the older multipoint port fuel injection (MPI) [1]. The demand of implementing gasoline particle filters (GPF) on GDI vehicles is therefore growing. Studies on possible toxicity from gasoline exhaust are scarce, and most research so far has been performed with MPI engines (e.g. [2]). We therefore investigated the effects of whole diluted exhaust from a new GDI vehicle exposed to a sophisticated 3D human lung tissue model. In addition, changes of exhaust composition upon installation of two different GPFs and their effects on lung cell responses were compared.

Fig. 1 Exposure setup. [A] An exposure box (blue) is directly connected to the exhaustpipe of a passenger car. The exposure box contains two chambers ([a] filtered air and exhaust). [B] Scheme of the human lung model composed of three different human cells. The cells are at the air-liquid interface, air (or exhaust) on top, and medium on the bottom [3].

Results

Particles, gases, metals in the lungs can cause reactive oxygen species (ROS).
Polyaromatic hydrocarbons (PAH) can bind to the aryl hydrocarbon receptor (AhR).

ROS → Oxidative stress.
HMOX1: Induced after oxidative stress.
SOD1/2: First line against ROS.
GSR: Recovery of glutathione (one of the main detoxification molecule).

can lead to → Pro-inflammation.
TNF: involved in acute, systemic inflammation
IL8: recruits other immune cells

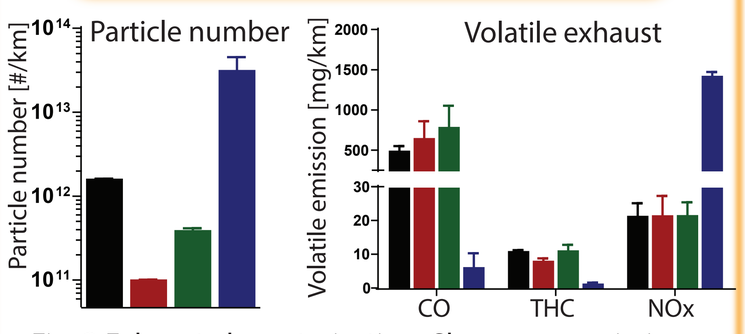
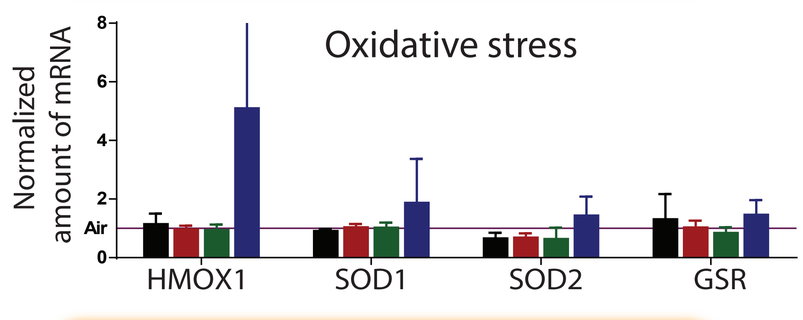


Fig. 2 Exhaust characterisation. Shown are emissions from the extra high part of the WLTC only.
→ Both GPFs reduced particle number
→ GDI emitted low amounts of THC and NOx
→ Diesel emitted high amounts of PN and NOx



PAH → AryI hydrocarbon receptor (AhR) activation.
NQO1: Detoxification of xenobiotics
IDO1: Maintains stable condition in immune cells (homeostasis)
NFE2L2: Regulates various other genes
CYP1A2: Detoxification of xenobiotics (e.g. PAHs)

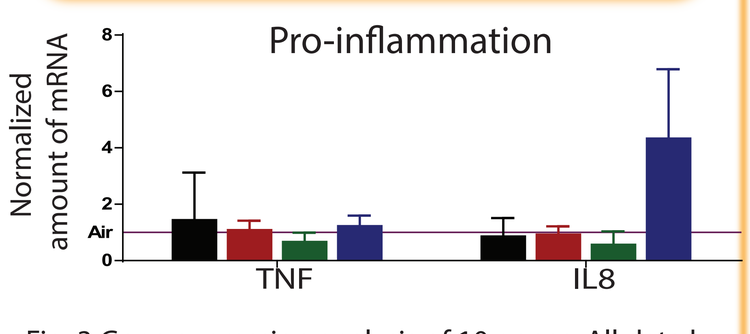


Fig. 3 Gene expression analysis of 10 genes. All data has been normalised to filtered air control (Air line=1). n=4-8.

GDI (both filtered and with GPF)
→ No oxidative stress
→ No pro-inflammation
→ No increase in AhR activation

Diesel (unfiltered)
→ Oxidative stress
→ Pro-inflammation
→ Increased metabolisation (NQO1 upregulation)

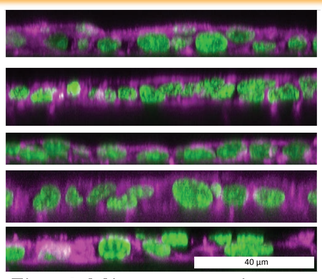


Fig. 4 Microscopy images. XZ cross-sections. Nucleus (green), F-Actin cytoskeleton (magenta).
→ Confluent monolayer for all conditions

Materials and Methods

Vehicle setup
- Gasoline direct injection (GDI) passenger car (Euro5)
  Unfiltered (original)
  uncoated GPF
  coated GPF
- Diesel passenger car (Euro2)
  unfiltered (original)
- Worldwide Harmonized Light Vehicles Test Cycles (WLTC)

Exposure setup
- 6 hours (app 10 WLTC) exposure to filtered air or 10x diluted exhaust

3D human lung epithelial tissue model is composed of
- Bronchial epithelial cells (16hbe, red)
- Macrophages (from human monocytes, thiel)
- Dendritic cells (from human monocytes, green)
- The cells are grown at the air liquid interface (air on top, medium on bottom), (Fig.1b) and [3].

Endpoints (shown here)
- Gene expression analysis (Fig. 3). All data has been normalized to filtered air and GAPDH, a standard gene whose expression is independent of a treatment.
- Microscopy images (Fig. 4). DAPI stained the nucleus and Phalloidin Rhodamine F-actin cytoskeleton.

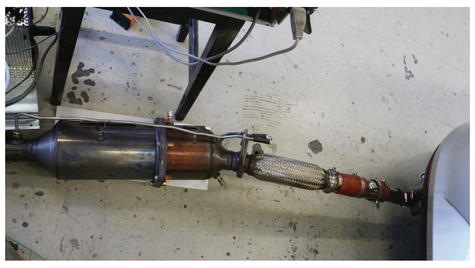


Fig. 5 Picture of tailpipe. A possible addition of the particle filter. 1:10 dilution of exhaust.

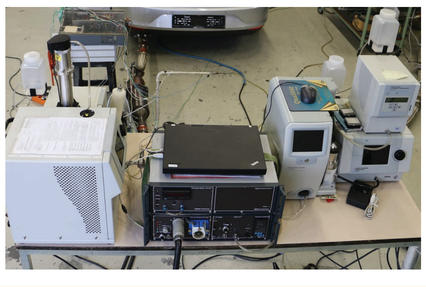


Fig. 6 Exhaust analysis directly at tailpipe (particles, shown in this picture) or in the CVS tunnel (CO, THC, NOx)

Conclusion

- Coated and uncoated GPFs reduced PN significantly in comparison to unfiltered GDI exhaust, while the volatile emissions did not differ in the three GDI exhausts.
- Diesel exhaust contained significant higher PN and NOx emissions compared to GDI exhaust, and effects in the 3D lung cell model such as oxidative stress, pro-inflammation, and metabolic activation were measured.
- The GDI vehicle used in the current study did not induce adverse effects in an acute exposure scenario for unfiltered as well as filtered conditions. We have, however, shown recently that depending on the vehicle unfiltered GDI exhaust can induce oxidative stress and metabolic activation [4], therefore the conclusions cannot be generalized for all GDI vehicles.
- In addition, long-term exposures are recommended for future studies to reveal chronic effects.

[1] Zhang, S. and McMahon, W.; SAE International Journal of Fuels and Lubricants 5(2), 637-646 (2012).
[2] Reed, M.D. et al.; Inhalation toxicology 20(13), 1125-1143 (2008).
[3] Bisig, C.J. et al.; CHIMIA International Journal for Chemistry 69(1), 68-68(1) (2015).
[4] Bisig, C.J. et al. Emission Control Science and Technology 1.3 (2015): 237-246.

This work was funded by the VERT Association, Swiss federal office for the environment, Swiss federal office for energy, Adolphe Merkle Foundation, Schweizer Erdölvereinigung, thank you very much.

Christoph Bisig
Adolphe Merkle Institute
christoph.bisig@unifr.ch
christoph.bisig@bfh.ch