Erratum to: Biological Effects in Lung Cells In Vitro of Exhaust Aerosols from a Gasoline Passenger Car With and Without Particle Filter

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This article contains an error in Figure 2B and 3 as well as in Table 1. The corrected figures, text and Table 1 appear below.

Oxidative stress and anti-oxidative response
Interestingly, according to the gene *SOD2* and *GSR* (Figure 2B), filtered exhaust overall appears to have resulted in a lower response to oxidative stress than unfiltered exhaust: *SOD2*-expression was increased 1.5 ± 0.5-fold by unfiltered exhaust but suppressed to 0.7 ± 0.1-fold activity by filtered exhaust. The according values for *GSR* are: 3.9 ± 2.8 (no GPF) and 1.3 ± 0.2 (GPF). *HMOX1* expression was induced 3.1 ± 1.3-fold by unfiltered exhaust and 0.9 ± 0.2-fold by filtered exhaust.

Pro-inflammation
In contrary to the observed oxidative stress, no pro-inflammation was measured. Both filtered and unfiltered exhaust did not increase the assessed genes, *TNFα* and *IL8*. While filtered exhaust resulted in a baseline expression (0.95 ± 0.38 and 0.73 ± 0.21- fold for *TNFα* and *IL8*), unfiltered exhaust decreased the expression of both genes (to 0.5 ± 0.2-fold and 0.7 ± 0.1) (Figure 3).

1 Conclusions

The biological tests showed that the removal of the particulate fraction decreased significantly the AhR- activation in human lung cells *in vitro*, also the Ames test suggested a lower genotoxicity for filtered gasoline exhaust. However, the GPF exhaust increased GSH oxidation in the lung cell cultures.

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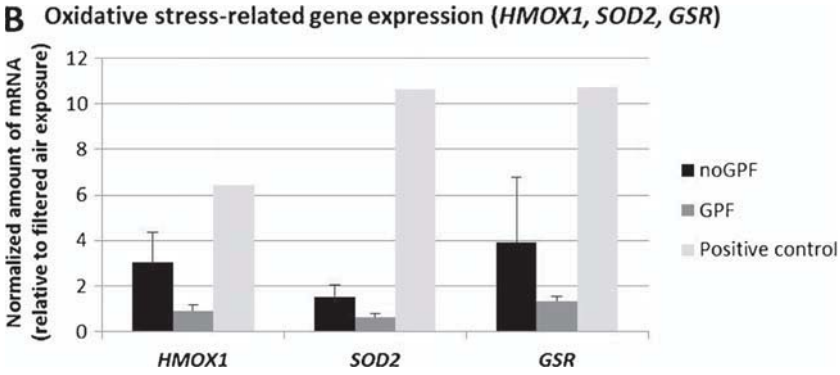

Fig. 2 **B** Oxidative stress. Transcriptional response to oxidative stress of three oxidative stress- responsive genes indicates increased oxidative stress upon exposure to unfiltered exhaust for *SOD2* and *GSR*. Error bars indicate SEMs. Experimental repetitions are for no GPF ($n = 3$) and GPF ($n = 4$). The respective controls are shown as white bars.

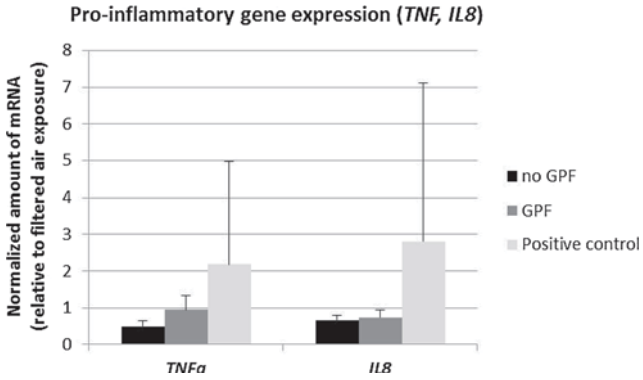

Fig. 3 Exhaust-related changes in the transcriptional activity of two pro-inflammatory genes, i.e. *TNF*α and *IL8*. No upregulation of pro-inflammatory genes was observed. Error bars indicate SEMs. Experimental repetitions are for no GPF ($n = 3$) and GPF ($n = 4$). *$p > 0.05$ no GPF vs. GPF. The respective controls are shown as white bars.

Table 1 Summary of the effects induced by non-filtered as well as filtered gasoline exhaust emissions in lung cells *in vitro*. The term oxidative stress is related to the gene expression analysis while anti-oxidative stress is related to level of the anti-oxidative protein GSH. The table indicates differences in response compared to filtered air exposures.

	Without GPF (i.e. particulate fraction and volatile compounds)	With GPF (i.e. volatile compounds)
Cytotoxicity	No effect	No effect
Cell morphology	No effect	No effect
Oxidative stress	increase	No effect
Anti-oxidative stress	No effect	(increase)
Pro-inflammation	No effect	No effect
AhR activation	increase	No effect
Genotoxicity (Ames test)	increase	No effect

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