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**From Particulates to NO₂ as health concern triggers from Diesel engine emissions.
A link with emission after-treatment strategies**

Strategies for the reduction of Diesel engine emissions mainly rely on oxidation catalysis, PM filtration and fuel sulphur content reduction. To evaluate the potential health benefit of these strategies, we have assessed the lung and systemic antioxidant profiles through the measurement of Catalase (CAT), Glutathione peroxidase (GPx), Superoxide dismutase (SOD) and Glutathione (GSH) in lung, heart, liver, kidneys of rats exposed by inhalation to diluted Diesel engine emissions on the one hand and in organotypic cultures of rat lung slices exposed to the same aerosols in vitro.

In vivo in the rat, systemic oxidant stress has been evidenced in lung, liver and kidney tissues, as well as increased serum TNF α concentrations reflecting systemic inflammation. These in vivo data are in total agreement and validate the observations made in vitro in the rat lung slices model.

In the absence of emission after-treatment system, oxidative damage was increased when reducing sulphur content in the fuel in a concentration related way.

For a given fuel, oxidative damage was more marked for engine medium rpm and medium load than for medium rpm high load.

The use of Diesel oxidation catalyst proved to induce the highest oxidative damage on both in vitro and in vivo biological systems. The oxidative damage after oxidation catalyst appears to be modulated by the presence of a particle filter on the exhaust line.

The extent of oxidative damage was closely correlated to both the absolute NO₂ and the NO₂/NO_x ratio in emitted aerosols. Experiments with pure NO₂ on in vitro lung slices allowed reproducing similar injuries with iso-NO₂ concentrations as in Diesel engine emissions.

To chemically assess the pro-oxidant potential of the emissions, we use CPH as a spin probe to interact with potential ROS generated during the contact of combustion emitted aerosols with aqueous media. CP* was then assayed by electron spin resonance ESR. We demonstrate a very good correlation between the NO₂/NO_x ratio elevation and the increased occurrence of ROS emitted from Diesel engines. As for oxidant damage, NO₂ by itself could reproduce a significant amount of ROS when applied to the system.

We suggest that these generated ROS from combustion aerosol contact are a major candidate being responsible for the observed tissular and systemic oxidative stresses. Further studies will be conducted to identify and assay separately the major ROS occurrence according to the experimental setup.

As a conclusion, From Euro1 where particulates were the major toxic trigger, through Euro3, to the prospective of Euro5 where oxidant potential becomes the major and more deleterious toxic trigger, we can parallel the evolution of required after-treatment devices known to deliver increased amounts and concentrations of NO₂ at the tailpipe. From this evolution we can forecast new health concerns which are recently reported in the literature from epidemiologic studies.

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