Diesel Exhaust Inhalation Impairs Vascular Functions

In a new experimental study, European researchers have shown that exposure to increased levels of combustion-derived air pollution for as little as 1 hour can impair vascular functions in humans. These data provide a plausible biological link to explain the association between air pollution and the acute heart attack.

A large number of experimental and epidemiological studies have demonstrated the association between long- and short-term exposure to air pollution and increased risk of cardiovascular mortality related to heart diseases, arrhythmias, and heart attack. These associations are strongest for fine particulate air pollutants (PM$_{2.5}$), of which the combustion-derived particulates of diesel exhaust are an important component.

Despite the strength of the epidemiological evidence, the important constituents and biological mechanisms responsible for the cardiovascular effects of air pollution are largely unknown. In order to shed light on this topic, a group of European researchers have assessed the effect of diluted diesel exhaust inhalation on cardiovascular functions in humans.

To this end, the scientists conducted an experimental study involving 30 healthy male non-smokers between 20 and 38 years old. All the subjects had normal lung function and reported no symptoms of respiratory tract infection for at least 6 weeks before or during the study.

Each subject was exposed for 1 hour in a specially built diesel exposure chamber according to a standard protocol. The diesel exhaust was generated from an idling Volvo diesel engine. More than 90% of the exhaust was shunted away, and the remaining part was diluted with air and fed into the exposure chamber at a steady-state concentration. The exposures were standardized by keeping the particulate concentration at 300 µg/m$^3$ and were associated with constant concentrations of NO$_2$, NO, CO, total hydrocarbons, formaldehyde, and suspended particles. The authors argue that these conditions can be assumed as relevant for the assessment of short-term health effects in humans since particulate matter concentrations can regularly reach levels of 300 µg/m$^3$ in heavy traffic, occupational settings, and the world’s largest cities. Note that the exposure to 300 µg/m$^3$ for 1 hour increases a person’s average exposure during a 24-hour period by only 12 µg/m$^3$.

The vascular function analyses (vasomotor and fibrinolytic functions) were performed in the subjects several hours after diesel exposure.

The results of this study show for the first time that inhalation of diesel exhaust, a common urban air pollutant, can damage vascular function in humans. At levels encountered in an urban environment, inhalation of dilute diesel exhaust has been shown to damage two important and complementary aspects of vascular function: the regulation of vascular tone and fibrinolysis. The authors argue that these important findings provide a plausible mechanism that links air pollution to the pathogenesis of atherothrombosis and acute heart attack. Furthermore, they discuss that this may be the result of reduced NO bioavailability in the vasculature and postulate that this effect is mediated by oxidative stress induced by the particulate fraction of diesel exhaust.

1 This study was performed with the approval of the local research ethics committee, in accordance with the Declaration of Helsinki, and the written, informed consent of all volunteers.


Contact: nick.mills@ed.ac.uk
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Additional Information: A recent project (LIFE04 ENV/D/000054) co-funded by the EU LIFE programme aims to demonstrate an affordable ultrafine particle measuring device for pan-European implementation. Prototypes will be installed at high traffic concentration sites in Dresden and Augsburg (Germany), Stockholm and Prague. For more information see project web site

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