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**Re: The Problem With Diesel**

The title of the editorial by Rushton (1) is misleading because considerable improvements in diesel engine and emissions technologies over the last decades have resulted in remarkable differences in the quality and quantity of current exposures, especially regarding polycyclic aromatic compounds (PAHs). The Kotin et al. article (2) cited by Rushton reported that solvent extracts of diesel emissions can cause tumors in mouse skin painting assays. This has been neither relevant for lung cancer nor for bladder cancer in human beings. Heinrich et al. and Mauderly et al. have shown in several rat studies that PAH-free particles can produce results similar to diesel particles containing PAHs (3,4). In addition, those health effects are caused by particle overload in the rat lung and not by organic compounds (5).

A recent review by Hesterberg et al. revealed the relevant decrease of chemical substances in diesel exhaust from contemporary engines by concrete measurement data for PAHs (6).

Stringent emission standards and air quality regulations in the European Union (EU) do not only apply to new engines, as indicated in the editorial (1). Retrofitting of catalysts or diesel particulate traps is feasible and not only available for older diesel engines but for off-road equipment as well (http://www.arb.ca.gov/msprog/decsinstall/decsinstall.htm). (Ultra-)low sulphur fuel quality has been introduced by government directives and is widely available, leading to lower particulate emissions (7) (www.epa.gov/oms/fuels/dieselfuels/index.htm). Financial and tax incentives are offered by certain EU governments to...
car owners to accelerate improved emission standards, and the introduction of Low Emission Zones and other air quality sanctions are incentives supportive of fleet renewal.

Improved fuel quality, proper diesel emission technology, and appropriate exhaust aftertreatment can eliminate the deleterious health effects of diesel exhaust, as McDonald et al. have shown (8). In addition, Lucking et al. demonstrated the preventive effects of a particle trap with regard to adverse cardiovascular effects in men (9). These studies used direct diesel emissions in contrast to the studies in ambient air as done by McCreanor et al., in which specific values for diesel emissions and diesel car traffic volume were missing (10).

It follows that the quantitative estimates presented by Rushton (1) on the burden of occupational cancer due to diesel engine emissions are misleading. In addition, these estimates suffer from unsolved methodological shortcomings (11). Thus, the interpretation by Rushton that the Diesel Exhaust in Miners Study (12,13) demonstrates excess lung cancer risks at concentrations as low as 1–2 μg/m³ is difficult to follow (14).

The editorial by Rushton (1) is entitled “The Problem with Diesel,” but it should be more precise in specifying that the results apply only to older diesel engine exhaust and technology and clearly convey the limitations of the applied burden of disease approach.

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References

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A few months ago, we finalized our reanalysis of the German potash miners cohort (1). This analysis did not show any notable association between exposure to diesel motor exhaust (DME) and lung cancer risk. It demonstrated, however, that previous employment in other mining, especially uranium mining, increased the risk of lung cancer.

We read with great interest the large cohort study and the nested case-control study on DME and lung cancer in the Journal (2,3). According to the authors, the a priori defined analyses of the cohort data by Cox proportional hazard modeling did not reveal a clear association between DME and lung cancer. The authors later calculated a considerable risk due to DME after adding birth year as a linear term to the model, but the effect of this adjustment is neither shown nor discussed. It should, however, be noted that the year of first exposure to DME would not be equivalent to the year of hire, especially for older birth cohorts.

For example, in our cohort, the average time gap between hire and first exposure to DME was 13.7 years for lung cancer cases and 6.3 years for all other individuals. The cumulative exposure to DME as a function of year of birth yielded a concave function. Therefore, we question whether the method proposed by Hein et al. (4) under the specific conditions of the DEMS studies really leads to an adjustment for birth-cohort effects.

Looking at the odds ratios for different exposure metrics from the studies, it is noticeable that the high values for the upper quartiles primarily arise from adjusting for several confounding factors like smoking. As shown in Table 1 for the potash miners, the distribution of controls really leads into the exposure categories is very similar to that of the lung cancer cases. Usually occupational cohorts have the advantage of being homogeneous with regard to confounding factors; hence, adjusting for them will change odds ratios only marginally (5).