

The Problem With Diesel

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Diesel engine exhaust (DEE) is a complex mixture of substances characterized by polycyclic aromatic hydrocarbons (PAH) surrounding an elemental carbon core. Diesel engines have a wide range of industrial applications including on-road equipment (most heavy and medium duty trucks and buses use diesel engines) and off-road applications in the mining, rail, construction, distribution, and farming industries and in the military, including the use of diesel-powered heavy equipment, locomotives, forklift trucks, ships, tractors, and generators. In a recent review of the literature, the highest levels of elemental carbon were reported for enclosed underground work sites in mining and construction with intermediate levels for above-ground semi-enclosed work areas for workshop mechanics, dock workers, and fire station workers, and the lowest levels being reported for enclosed areas separated from the source such as drivers, train crew, parking attendants, vehicle testers, and utility service workers (1).

A large body of epidemiological work has shown consistent associations between particulate matter in ambient air and several health outcomes including chronic bronchitis, ischemic heart disease, stroke, and respiratory infections and exacerbation of asthma. The effect of DEE from traffic on people with preexisting disease has also been shown, for example, in the reduction of lung function resulting from a 2-hour walk by people with mild or moderate asthma down London's busiest shopping street (Oxford Street), where only diesel-powered buses and taxis are permitted access, compared with a similar walk round Hyde Park (2).

The possibility that DEE might cause cancer in humans has been raised since 1955 when it was demonstrated that the particulate fraction of DEE contained PAHs such as benzo(a)pyrene known by then to cause tumors in experimental animals (3). The gas phase includes carbon monoxide and nitrogen oxides, but it is the particulate phase of the exhaust that appears to be implicated as a lung carcinogen. An effect of DEE on bladder cancer is also plausible because metabolites of PAH present in DEE are concentrated in the urine and may interact with the urothelium of the bladder (4).

In this issue of the Journal, two related articles (5,6) report results from studies of DEE in miners based on quantitative estimates of respirable elemental carbon (REC) and focusing on lung cancer. In the cohort study (5), inclusion of co-exposures such as silica did not change the findings substantially. In the case-control study (6), adjustment was carried out for several potential confounders including smoking. A dose-response relationship was found with increasing exposure to REC in both the cohort and case-control studies but is particularly clear in the latter, where an interaction between smoking and DEE was also found. These studies in miners make an important contribution to the body of evidence

about DEE and are timely given the forthcoming International Agency for Research on Cancer (IARC) monograph meeting this year at which the current IARC categorization of DEE as a group 2A (probable human) carcinogen (7) will be reconsidered.

The issue of causality is fundamental when estimating and ranking burden of disease attributable to various exposures. A recent study estimating the burden of occupational cancer in Great Britain chose to include all IARC group 1 (definite) and 2A carcinogens (8). DEE emerged as one of the most important problems. For bladder cancer related to exposure to DEE, the estimated total attributable fraction for bladder cancer was 1.00% (95% confidence interval [CI] = 0.17 to 2.03), with an estimated 47 (95% CI = 8 to 94) deaths in 2005 and 106 (95% CI = 18 to 214) cancer registrations in 2004; for lung cancer, the attributable fraction was 1.84% (95% CI = 0.00% to 3.37%), with 605 (95% CI = 272 to 1107) deaths and 695 (95% CI = 313 to 1269) cancer registrations. DEE was the sixth most important occupational carcinogen, contributing 8.1% of the deaths and 5.9% of the cancer registrations. The majority occurred in land transport as expected but also in the construction industry where nearly 500 000 workers were estimated to have been exposed to DEE over the 40-year risk exposure period before the year of estimation.

As the authors point out in the mining studies published in this issue, the levels of exposure they have found are high compared with other studies. The median elemental carbon values in the Oxford Street study were 7.5 $\mu\text{g}/\text{m}^3$ (range 3.9–16) compared with 1.3 $\mu\text{g}/\text{m}^3$ (range 0.4–6.7) in Hyde Park. The Oxford Street values are comparable to the lower exposures found in the mining study. Background rates of between 1 and 2 $\mu\text{g}/\text{m}^3$, similar to those in Hyde Park, have been found in other studies of urban environments. The article by Pronk et al. (1) indicates that the exposures to DEE have generally been declining, although it is difficult to quantify the rate of decline.

Occupational and environmental exposures are expected to continue to decrease in the future in line with increasingly stringent emissions standards for diesel engines (see, eg, <http://www.dieselnet.com/standards/eu/ld.php#stds> for the European Union), although standards generally apply only to new engines and not those already in use at the time of the standard implementation; however, as old equipment is replaced over time, all engines will eventually conform to the emissions standards.

The continuous analyses and particularly the lagged categorical analyses in the case-control study by Silverman et al. (6) indicate a sharp rise in risk at lower levels and show that background levels of 1–2 $\mu\text{g}/\text{m}^3$ are still likely to carry a small excess risk; substantial proportions of the population exposed at these low levels of

exposure would thus continue to contribute to the burden of cancer from DEE.

These results indicate that stringent occupational and particularly environmental standards for DEE should be set and compliance ensured to have an impact on health outcomes. In the occupational situation, in addition to lower emission and more efficient engines, reduction in DEE can be achieved through: 1) engineering controls such as improved ventilation and regular maintenance of vehicles; 2) improving worker practices such as limiting the number of vehicles, particularly in closed spaces, and turning off engines when not in use; and 3) as a last resort, the use of appropriate respiratory protective equipment. Reduction in the general environment presents more of a challenge, although some of the occupational control measures are also relevant. However, the necessity for such reduction is becoming increasingly apparent and is essential if the health of large numbers of people is not to be compromised.

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Notes

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Physical Activity as a Standard Cancer Treatment

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Adequate physical activity is a cornerstone of physical health and mental well-being. Many cancer survivors who have a good prognosis are at higher risk of dying from other causes, such as cardiovascular diseases, and could possibly reap the same benefits of exercise as individuals without a cancer diagnosis. Furthermore, the physical and mental stresses of a cancer diagnosis, including side effects of treatments, present the cancer survivor with additional challenges. An increasing body of literature, including randomized trials, demonstrates the benefits of physical activity on physiological parameters, quality of life, functional status, fatigue, and depression for at least some cancers. Despite acknowledging potential risks of some specific physical activities at certain intensities for some subgroups of cancer survivors, many organizations, such as the American Cancer Society (1), the World Cancer Research Fund and the American Institute for Cancer Research (2), the American College of Sports Medicine (3), and the US Department of Health and Human Services (4), now advocate physical activity for cancer survivors. Details regarding the type, duration, and intensity of exercise will have to be tailored to the individual patient, depending on various factors such as age, physical health, specific cancer, and treatments; however, the vast majority of patients will likely benefit to some degree from physical activity.

In this issue of the Journal, Ballard-Barbash et al. (5) extensively review the potential effects of physical activity on cancer-specific and all-cause mortality. They also summarized the effect of physical activity interventions on potential intermediate biomarkers of cancer progression, such as insulin. Unlike previous reviews, which focused on functional health and quality of life, this review addresses the more provocative question of whether physical activity can actually improve cancer-specific survival. Many may accept general health benefits of physical activity for cancer survivors, but the implication of a direct anticancer effect could engender some skepticism. Given the limited success that the most potent and cleverly designed drugs have had on cancer to date, why should something as seemingly simple as walking have potent anticancer activity? We are far from having definitive answers to this question, but as reviewed by Ballard-Barbash et al. (5), perhaps, we are getting closer to an answer.

Whereas most cancer therapies to date focus on killing the tumor, physical activity may offer two complementary roles for standard cancer therapy. First, cancer-specific mortality, although generally attributed to the destructive behavior of the tumor, is also dependent on the general health of the patient. Overall health status is inherently integrated and substantially influenced by factors such