The recent article by Wong and colleagues reported that particulate matter with aerodynamic diameter <2.5 μm (PM2.5) was associated with increased risk of mortality rate for all, lung, digestive organs for males and females in Hong Kong and breast, female genital, and lymphohematopoietic cancers in females (1). The mechanism proposed to explain these findings was defects in DNA repair function and replication mostly due to heavy metal content of PM2.5 particulates. A more likely mechanism is oxidative damage to DNA by polycyclic aromatic hydrocarbons (PAH) adsorbed by PM2.5 (2). PAHs can enter the blood through the lungs and easily reach many organs. PAHs were found to increase risk of digestive tract cancers (3). As smoking tobacco also involves PM2.5 and PAH, it would be expected that all cancers associated with smoking (4) would also be affected by PM2.5. In fact, that was generally found to be the case in an ecologic study of an index for PM2.5, acid deposition, in the United States (5). In that study, mortality rates for 11 types of cancer (respiratory, digestive tract, bladder, female genital, blood, and skin cancer) as well as all-cancer mortality rate were found significantly correlated with acid deposition in 1985 for the period 1970 to 1994 after adjustment for several other cancer risk-modifying factors. Acid deposition is largely due to emissions from coal-fired power plants and is associated with PM2.5 particulates largely composed of carbon with adsorbed PAHs along with sulfuric acid. Acid deposition was highest in the northeast.

Disclosure of Potential Conflicts of Interest
No potential conflicts of interest were disclosed.

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References
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William B. Grant

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