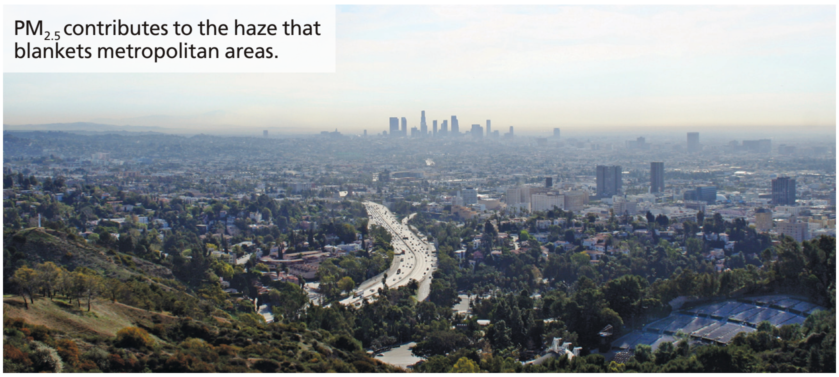
Particulate Soup

Identifying the Most Toxic Constituents of $PM_{2.5}$

Chronic exposure to air pollution has long been linked with cardiopulmonary-related mortality, and ambient fine particulate matter ($PM_{2.5}$) is often considered a primary cause of this association. Once inhaled, these particles can cause inflammation and oxidative stress. This in turn may result in systemic effects, including the buildup of plaque deposits that can lead to heart attacks and strokes. A new study comparing air pollution exposure with health data gathered over a 5-year span now takes a closer look at which individual constituents of $PM_{2.5}$ may be most likely responsible for associations between ambient air quality and mortality [*EHP* **118:363–369; Ostro et al.**].



$PM_{2.5}$ contributes to the haze that blankets metropolitan areas.

Ambient $PM_{2.5}$ contains solid and liquid particles from many sources, particularly from fossil fuel combustion; among other constituents, it contains elemental and organic carbon, sulfates, nitrates, iron, potassium, silicon, and zinc. The National Research Council has highlighted the importance of routinely collecting toxicity data on particle constituents to help refine air quality standards, target control strategies, and enhance the accuracy of health impact assessments.

In the current study, the authors used data from the California Teachers Study for 45,000 active and former female public school professionals. The teachers lived within 8 or 30 km of monitors that collected data on $PM_{2.5}$ between June 2002 and July 2007. The large amount of individual-level data provided by participants allowed researchers to control for risk factors that could possibly confound the analysis. Smoking rates and indoor occupational exposures, for example, were very low in this study cohort, making it easier to identify an independent effect of outdoor air pollution. Air pollution measurements were generally taken twice a week, and information from health questionnaires also helped determine individual exposure.

Of 8 constituents studied, organic carbon and sulfates were found to be most strongly associated with all-cause, cardiopulmonary, ischemic heart disease, and pulmonary mortality. Even modest concentrations of these 2 constituents were associated with mortality from all 4 causes. According to the authors, the study provides new information to help focus and streamline regulatory efforts on a variety of sources of $PM_{2.5}$, including gasoline and diesel fuel, and other combustion activities. They write that the reduction of ambient $PM_{2.5}$, particularly from fuel combustion, may offer significant public health benefits.

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Endocrine Damper?

Flame Retardants Linked to Male Hormone, Sperm Count Changes

Additive flame retardants such as tris(1,3-dichloro-2-propyl) phosphate (TDCPP) and triphenyl phosphate (TPP), which are not chemically bonded to the products they are intended to protect, may escape into indoor environments such as homes, offices, and car interiors. A new study shows that men living in homes with higher amounts of TCDPP and TPP in their house dust had reduced sperm counts and altered levels of hormones related to fertility and thyroid function [*EHP* **118:318–323; Meeker and Stapleton**]. Because the research to date suggests both compounds are ubiquitous in U.S. homes, the study points to a pressing need for further investigation into the sources and levels of day-to-day exposure to the compounds as well as their potential health effects.

TDCPP has long been the main flame retardant used in automotive foam cushioning, while TPP has been used for decades in a wide variety of applications, including furniture foam. Since polybrominated diphenyl ether (PBDE) flame retardants were banned in Europe and discontinued in the United States in 2004, the use of alternative flame retardants such as TDCPP and TPP has been on the rise. Indoor dust is known to be an important source of exposure to PBDEs (which also are additive compounds), and the authors suspect this could also be true for other flame retardants.

In the current study, TDCPP was found in 96% and TPP in 98% of the house dust samples. As has been reported for other flame retardants found in house dust, the concentrations of the flame retardants in the samples varied markedly, with ranges of <107–56,090 ng/g for TDCPP and <173–1,798,100 ng/g for TPP. The concentrations of TDCPP in the men's homes were comparable to those of PBDEs, whereas the levels of TPP were considerably higher.

Because the study participants were part of a larger project involving men recruited from a Boston infertility clinic, the authors had access to information about the men's reproductive and thyroid hormone levels as well as their semen quality. They estimated associations for an interquartile range (IQR) increase in the level of each chemical measured in the dust samples, adjusting for potential confounders such as age and body mass. IQR analyses reflect the difference between the concentrations at the highest and lowest ends of the middle 50% of exposures.

This analysis revealed that each IQR TPP increase in the homes was associated with a 19% decrease in sperm concentrations and a 10% increase in prolactin levels. Increased prolactin can be a marker of decreased dopamine activity and also may be associated with erectile dysfunction. The authors also found that each IQR increase in TDCPP in the homes was associated with a 17% increase in prolactin and a 3% decline in free levels of the thyroid hormone thyroxine.

The findings mirror the limited toxicology data available on the study's end points. They are also consistent with findings on other organophosphate compounds such as the urinary metabolite of the insecticide chlorpyrifos [*EHP* 112:1665–1670; Meeker et al.]. The authors hope to follow up by exploring human exposure pathways for these flame retardant chemicals and by reassessing these relationships with markers of endocrine function among a greater number of men from the larger ongoing study.

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