

Particle air pollution clearly causes substantial deaths and illness, but what makes fine particles so toxic—the size, the chemical compound, or both?

Mounting Evidence Indicts Fine-Particle Pollution

Talk about heart-stopping news: Spending time in traffic may triple some people's risk of having a heart attack an hour later. That's what German researchers reported last October in the *New England Journal of Medicine* (*NEJM*), based on responses from 691 heart attack survivors about their activities in the days before they fell ill. The study seemed to support the notion that tiny air pollution particles from tailpipes, along with stress, could help trigger a heart attack. Yet in another recent study in which volunteers in upstate New York breathed in lungfuls of these so-called ultrafines, particles less than 0.1 micrometer (μm) in diameter, the effects were minimal. If ultrafines were the main culprit, "you would have expected to see something more," says Daniel Greenbaum, president of the Health Effects Institute (HEI) in Cambridge, Massachusetts.

The discordant studies illustrate the dilemma posed by fine particle air pollution. The term refers to particles of dust, soot, and smoke consisting of hundreds of chemicals that are defined by their mass and size—2.5 μm in diameter or less, or about one-30th the width of a human hair. They are known collectively as $\text{PM}_{2.5}$. Hundreds of studies have suggested that breathing fine particles spewed by vehicles, factories, and

power plants can trigger heart attacks and worsen respiratory disease in vulnerable people, leading to perhaps 60,000 premature deaths a year in the United States. In response, the U.S. Environmental Protection Agency (EPA) in 1997 added new regula-



At risk. Studies with elderly volunteers have shown that slight changes in outdoor particle levels can change heart rate variability.

tions to existing rules for coarser particles (PM_{10}), issuing the first-ever standards for $\text{PM}_{2.5}$. But the move came only after a bitter fight over whether the science supported the rules and a mandate from Congress for EPA to expand its particle research program.

Now the issue is getting another look as EPA faces a December 2005 deadline for revisiting its $\text{PM}_{2.5}$ standard. EPA scientists, after reviewing piles of new data implicating $\text{PM}_{2.5}$ in health effects, have proposed tightening the 1997 standard to further reduce ambient concentrations of fine particles. Some scientists and industry groups remain skeptical, noting that researchers still haven't pinned down what makes particles dangerous—whether it's mainly size, and that the tiniest particles are most potent; or chemistry, such as metal content; or some combination of the two. Despite 8 years and some \$400 million in research, finding out exactly how fine particles do their dirty work has proved frustratingly elusive, researchers say. "We've gotten glimpses, but we don't yet have enough systematic coverage of the problem," says epidemiologist Jon Samet of Johns Hopkins University in Baltimore, Maryland.

Unmasking a killer

Although the evidence against fine particles, initially circumstantial, has grown stronger, gaps still remain. It began with epidemiologic studies finding that when levels of particulate matter (PM) edged up in various cities, hospital visits and deaths from heart and lung disease rose slightly, too. Two landmark studies

How Dirty Air Hurts the Heart

A decade ago, most cardiologists never suspected that breathing tiny particles of soot and dust could damage their patients' hearts, let alone trigger a heart attack. Today "there's no doubt that air pollution plays a role in cardiovascular disease," says cardiovascular researcher Robert Brook of the University of Michigan, Ann Arbor.

Fine particles seems to affect the heart in two ways: by changing the heart's rhythm and by causing systemic inflammation. Many studies—from animal experiments to tests in which retirement home residents wore heart monitors—have shown that breathing particle pollution can slightly quicken the pulse and make the heart-beat less variable. The mechanism isn't yet known, but one possibility is that airway receptors stimulate nerves in the heart. A less variable heart rate, in turn, makes the heart more prone to arrhythmia

(irregular heartbeat), which can presage cardiac arrest.

People don't usually die from arrhythmias unless they are very ill already, Brook notes. But particles also penetrate the lung's alveoli and cause inflammation and oxidative stress. The lung cells then pump proteins called cytokines into the bloodstream. This apparently sparks other immune responses that promote blood clot formation and the constriction of blood vessels. These effects, in turn, may cause deposits of lipids known as plaques to rupture and block blood flow to the heart. "If these things all come together, somebody who's vulnerable might be pushed over the edge" and have a heart attack, says epidemiologist Annette Peters of the National Research Center for Environment and Health in Neuherberg, Germany.

Over the long term, inflammation from breathing particles may also contribute to atherosclerosis, or hardening of the arteries, in the same way that secondhand tobacco smoke is thought to inflict