Acute coronary syndromes such as unstable angina or myocardial infarction often develop unexpectedly and severely interrupt a patient’s life. Two main pathological processes, atherosclerosis and thrombosis, lead to acute coronary syndromes such as unstable angina and myocardial infarction. The typical atherosclerotic lesion is a fibrolipid plaque composed of a pool of lipids covered with a connective tissue cap. Although the plaque narrows the coronary arteries, acute coronary syndromes only occur when a plaque erodes, fissures, or ruptures and a thrombus is formed that partially or totally occludes the arteries and impedes blood flow. The sudden onset of the disease has led to research examining triggers of acute coronary syndromes. Among the factors that contribute to acute coronary syndromes in vulnerable patients are physical exertion, extreme anger, sexual activity, and drug abuse. These factors alter the shear stress at the arterial walls and increase the risk of thrombosis. Triggering of myocardial infarction caused by these stressors occurs within 1 or 2 hours of exposure, and the risk subsides rapidly after this time window. This further emphasizes the transient nature of the risks associated with these triggers and distinguishes these factors from those risk factors of thrombosis. The study by Pope and colleagues is unique in that it is based on a large ongoing registry with angiographically characterized patients, distinguishing it from studies based on hospital discharge diagnoses. The observed association was attributable to the subgroup of subjects whose angiographic examination had identified at least 1 severe stenosis in the coronary arteries (defined as at least 1 coronary artery with ≥70% maximal stenosis as determined at angiography). Recent understanding of the underlying pathology suggests that a severely occluded vessel is not necessarily the location of plaque rupture, but detection of at least 1 diseased vessel is indicative of the severity of the underlying atherosclerosis. The study by Pope and colleagues strongly suggests that the triggering of acute coronary syndromes by fine ambient particles may occur predominantly in vulnerable groups of the population.

Ambient particulate air pollution has been linked to exacerbation of cardiovascular disease morbidity and mortality. Among the ambient air pollutants, fine particles, which are defined as particles with an aerodynamic diameter smaller than 2.5 mm, are suspected to be closely linked to exacerbation of cardiovascular disease. Fine ambient particles penetrate into the lung, and a fraction of them are deposited in the alveoli. Fine particles are heterogeneous with respect to their size and chemical composition. Their size varies between 3 orders of magnitude, ranging between 2.5 mm and a few nanometers. In the alveoli, the larger fine particles may activate macrophages and induce a systemic response geared to clean the alveoli from extraneous matter. In addition, adsorbed substances and particles smaller than 200 nm may cross the air–liquid interface and enter the blood stream. There is evidence that exposure to particles below 200 nm (such as diesel particles) alter endothelial function within hours and induce a prothrombotic state. Therefore, it seems plausible to extend the concept of triggering factors to environmental agents such as fine particles, as suggested by the study of Pope and colleagues and by others.

The study conducted by Pope and colleagues has several limitations. First, it did not determine the time of symptom onset, nor were data on hourly fine-particle concentrations available to further narrow the time window of the relevant fine-particle exposure to trigger a myocardial infarction. Acute coronary syndromes were more frequently observed in association with elevated concentrations of fine particles on the same and on the previous day, indicating that exacerbation of disease was quite immediate—within hours, when one considers that the exact timing of the onset of symptoms was unknown to the investigators. Second, no data on chemical or biological markers were available to further validate the mechanism by which fine particles trigger myocardial infarctions.
physical characteristics were available to further identify the particle properties responsible for the triggering of the myocardial infarctions. In particular, fine particles from the Utah area have been demonstrated to contain relatively high concentrations of transition metals implicated to catalyze the formation of oxygen radicals and their progeny, increasing the oxidative stress burden. Oxidative stress has been implicated as contributing to impaired fibrinolytic function in association with cigarette smoke as well as diesel particles. Third, the results obtained for largely nonfatal acute coronary syndromes may not be transferable to cases of sudden cardiac death. Substantial evidence has been found, however, that fine particles may also trigger arrhythmia. and therefore, fine particles also may increase the likelihood of a “vulnerable myocardium” prone to develop arrhythmia when ischemia occurs.

The study Pope and colleagues is an important hallmark, linking ambient fine particles to exacerbation of underlying coronary artery disease. It is important for risk assessment quantifying the public health impact of fine-particle exposure, because triggering of nonfatal myocardial infarction may advance disability of otherwise asymptomatic subjects for several years, leading to substantial health care and societal costs. The study also calls for further investigations within the cohort of the Intermountain Collaborative Heart Study because it would be scientifically intriguing to further characterize the vulnerable subgroup of those patients presenting with acute coronary syndromes on high–air pollution days. In particular, fine particles may serve as a model exposure to characterize genetically determined susceptibility for developing a prothrombotic state in response to oxidative stress. Why are fine particles a fine model substance? Because all of us breathe them, 24 hours a day.

Disclosures
None.

References

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Annette Peters

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