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AIR POLLUTION EXPOSURE MAY TRIGGER MI

BOSTON—While chronic risk factors for myocardial infarction (MI), such as obesity and cigarette use, are well known, acute precipitants are less well understood. A recent survey reveals that exposure to particulate pollution may trigger MI within hours or a day.[1]

Increases in particulate air pollution have been associated with increased hospital admission rates for cardiovascular disease. Remarked Annette Peters, PhD, of GSF-National Research Center for Environment and Health in Neuherberg, Germany, “We wanted to know how long the induction time was between exposures and the onset of symptoms.” Dr. Peters and colleagues interviewed 772 patients (489 male; mean age, 61.6 years) who had had MIs in the greater Boston area between January 1995 and April 1996. The investigators compared MI timing with hourly air

measurements of concentrations of particles less than 2.5 μm in diameter (PM_{2.5}), black carbon, and gaseous pollutants.

EARLY AND LATE RESPONSES

“Even short exposures may be a risk factor,” said Dr. Peters. “We saw that there was slightly increased risk for MI at one hour, and a significant increase in risk for MI one to two hours after exposure” to particulate pollution. Odds ratios (ORs) for acute MI were transiently but significantly elevated following exposure to fine particulates, and they rose as particulate concentrations rose. For patients in the highest quintile of particulate exposure (PM_{2.5} concentrations, averaged over two hours, between 17.1 and 74.8 $\mu\text{g}/\text{m}^3$ in the 180 to 60 minutes immediately before the MI), the OR was 1.44.

Additionally, Dr. Peters and colleagues observed a delayed effect: “Elevated concentrations 24 to 48 hours before were also predictive for MI.” Risk of MI was significantly elevated between 24 and 48 hours after an exposure (OR following PM_{2.5} concentrations, averaged over this 24-hour period, of 16.3 to 52.2 $\mu\text{g}/\text{m}^3$ was 1.32). This delayed effect showed a statistically significant positive trend, increasing with higher PM_{2.5} levels. “We combined information on exposures 180 to 60 minutes before and 48 to 24 hours before [MI], and found that the risks associated with each exposure period were independent,” Dr. Peters said.

“We observed these effects [from particulate concentrations] below accepted standards,” noted Dr. Peters. “There seems to be no threshold: Effects seem to be linear even at low concentrations.” The risk analyses are consistent with observations of increased hospital admissions either on the same day or with a one-day lag relative to elevated 24-hour average particle concentrations. “The effect of [larger particles (PM₁₀ – PM_{2.5})] was weaker, so the fine particles, below 2.5 microns, are thought to be involved,” said Dr. Peters. “We also analyzed gases. For ozone, there was a positive

association with MI within hours, but this [effect] was weaker; we found the same for carbon monoxide.”

POLLUTION’S TWO-PRONGED ATTACK

Dr. Peters identified two mechanisms by which exposure to particulate pollution may trigger MI. “There might be a direct activation of the autonomic nervous system by particulate pollution. This might be the faster mechanism,” she hypothesized. Accordingly, heart rate is accelerated with air pollution exposure, the authors noted. Additionally, “Particles might trigger alveolar macrophages to produce cytokines, thereby increasing coagulability of the blood to trigger clot formation. This might be the delayed mechanism.” The authors noted that ventricular fibrillation, increased blood viscosity, and elevated levels of C-reactive protein are also associated with exposure to high particulate concentrations. These factors could conceivably combine to disturb an atherosclerotic plaque and trigger formation of an occlusive thrombus to cause MI, they proposed.

Given that even short exposures to elevated PM2.5 can trigger MI, “it’s probably important to avoid exposure in conjunction with other triggers, such as exercise,” Dr. Peters suggested.

To those already at risk for MI, she advised, “On high-pollution summer days, air-conditioning does reduce particulate concentration indoors.”

—Mimi Zucker, PhD

**Getting High Raises Risk of
MI**

Boston—Another transient trigger of MI may be much easier to avoid: inhaled cannabis smoke. Although marijuana has long been perceived as “safe” relative to other illicit drugs, those who began using marijuana in their youth during the late 1960s and 1970s are now in an age-group plagued by cardiovascular risk factors. A survey conducted by Murray A. Mittleman, MD, DrPH, and colleagues shows that marijuana may not be as benign as previously thought.[1] Two cardiovascular effects are observed within an hour of smoking marijuana: increased heart rate and decreased oxygen saturation.

In an interview with **RESPIRATORY REVIEWS**, Dr. Mittleman, an Assistant Professor of Medicine at Beth Israel-Deaconess Medical Center in Boston, cited “a series of studies ... evaluating a dose-response relationship for cannabinoids on heart rate.” In their study, the researchers noted, “Overall, there is a net increase in myocardial oxygen demand with a decrease in oxygen supply, which is due in part to an increase in carboxyhemoglobin.” According to Dr. Mittleman, the increased carboxyhemoglobin seen with marijuana use is probably “a generic effect of inhaling plant combustion products,” as with cigarette smoking and other smoke exposures. However, cannabis in particular may pose an additional threat: it raises supine blood pressure and resting heart rate by 20% to 100%.

The researchers also cited a study in which the effects of smoking cannabis cigarettes on angina were compared to those of smoking “placebo” marijuana cigarettes that lacked tetrahydrocannabinol (THC), the psychoactive ingredient of marijuana. Those who inhaled THC showed a lowered threshold for angina. What is the mechanism for cannabis’ cardiovascular effects? “There is a hypothesis that the increase in heart rate is mediated by withdrawal from parasympathetic effects, but that’s fairly simplistic,” said Dr. Mittleman. “There are cannabinoid receptors in vascular tissues and in the heart, but it’s not clear whether these are responsible. The interesting thing is that the [cannabinoid] receptor that’s been found [in vessels] is very different [from] the one [from the central nervous system] that’s been cloned already.”

How big a risk does marijuana use pose? “In

relation to chronic risk factors, such as cigarette smoking, it's probably less important—use in habitual marijuana smokers is usually infrequent in relation to cigarette smoking,” said Dr. Mittleman. “However, those with a higher baseline risk (because of cigarette smoking or obesity, for example), and especially those with established heart disease, may be exposing themselves to a risk they should pay attention to.”

—Mimi Zucker, PhD

Reference

1. Mittleman MA, Lewis RA, Maclure M, et al. Triggering myocardial infarction by marijuana. *Circulation*. 2001;103:2805-2809.

Reference

1. Peters A, Dockery DW, Muller JE, Mittleman MA. Increased particulate air pollution and the triggering of myocardial infarction. *Circulation*. 2001;103:2810-2815.

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