RSNA Press Release

Exposure to Air Pollution May Cause Heart Damage

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OAK BROOK, Ill. — Researchers using cardiac MRI have found that long-term exposure to air pollution is associated with early signs of heart damage, according to a study that was published today in *Radiology*, a journal of the Radiological Society of North America (RSNA). The research indicates that fine particulate matter in the air may contribute to diffuse myocardial fibrosis, a form of scarring in the heart muscle that can precede heart failure.

Cardiovascular disease is the leading cause of death worldwide. There is a large body of evidence linking poor air quality with cardiovascular disease. However, the underlying changes in the heart resulting from air pollution exposure are unclear.

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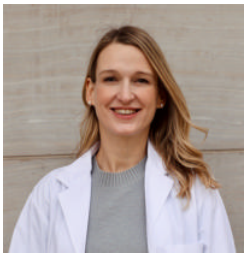

Kate Hanneman, M.D., M.P.H.

"We know that if you're exposed to air pollution, you're at higher risk of cardiac disease, including higher risk of having a heart attack," said the study's senior author Kate Hanneman, M.D., M.P.H., from the Department of Medical Imaging at the Temerty Faculty of Medicine, University of Toronto and University Health Network in Toronto. "We wanted to understand what drives this increased risk at the tissue level."

Dr. Hanneman and colleagues used cardiac MRI, a noninvasive imaging technique, to quantify myocardial fibrosis and assess its association with long-term exposure to particles known as $PM_{2.5}$. At 2.5 micrometers in diameter or less, $PM_{2.5}$ particles are small enough to enter the bloodstream through the lungs. Common sources include vehicle exhaust, industrial emissions and wildfire smoke.

The researchers wanted to evaluate the effects of air pollution on both healthy people and those with heart disease, so the study group included 201 healthy controls and 493 patients with dilated cardiomyopathy, a disease that makes it more difficult for the heart to pump blood.

Higher long-term exposure to fine particulate air pollution was linked with higher levels of myocardial fibrosis in both the patients with cardiomyopathy and the controls, suggesting that myocardial fibrosis may be an underlying mechanism by which air pollution leads to cardiovascular complications. The largest effects were seen in women, smokers and patients with hypertension.

The study adds to growing evidence that air pollution is a cardiovascular risk factor, contributing to residual risk not accounted for by conventional clinical predictors such as smoking or hypertension.

"Even modest increases in air pollution levels appear to have measurable effects on the heart," Dr. Hanneman said. "Our study suggests that air quality may play a significant role in changes to heart structure, potentially setting the stage for future cardiovascular disease."

Knowing a patient's long-term air pollution exposure history could help refine heart disease risk assessment and address the health inequities that air pollution contributes to both in level of exposure and effect. For instance, Dr. Hanneman said, if an individual works outside in an area with poor air quality, healthcare providers could incorporate that exposure history into heart disease risk assessment.

The air pollution exposure levels of the patients in the study were below many of the global air quality guidelines, reinforcing that there are no safe exposure limits.

"Public health measures are needed to further reduce long-term air pollution exposure," Dr. Hanneman said. "There have been improvements in air quality over the past decade, both in Canada and the United States, but we still have a long way to go."

In addition to illuminating the links between air pollution and myocardial fibrosis, the study highlights the important role that radiologists will play in research and clinical developments going forward.

"Medical imaging can be used as a tool to understand environmental effects on a patient's health," Dr. Hanneman said. "As radiologists, we have a tremendous opportunity to use imaging to identify and quantify some of the health effects of environmental exposures in various organ systems."

"Association between Long-term Exposure to Ambient Air Pollution and Myocardial Fibrosis Assessed with Cardiac MRI." Collaborating with Dr. Hanneman were Jacques du Plessis, M.D., Chloe DesRoche, M.D., M.Sc., Scott Delaney, Sc.D., J.D., M.P.H., Rachel C. Nethery, Ph.D., Rachel Hong, B.Sc., Paaladinesh Thavendiranathan, M.D., S.M., Heather Ross, M.D., M.H.Sc., and Felipe Castillo, M.D.

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For patient-friendly information on cardiac MRI, visit *RadiologyInfo.org*.

Images (JPG, TIF):

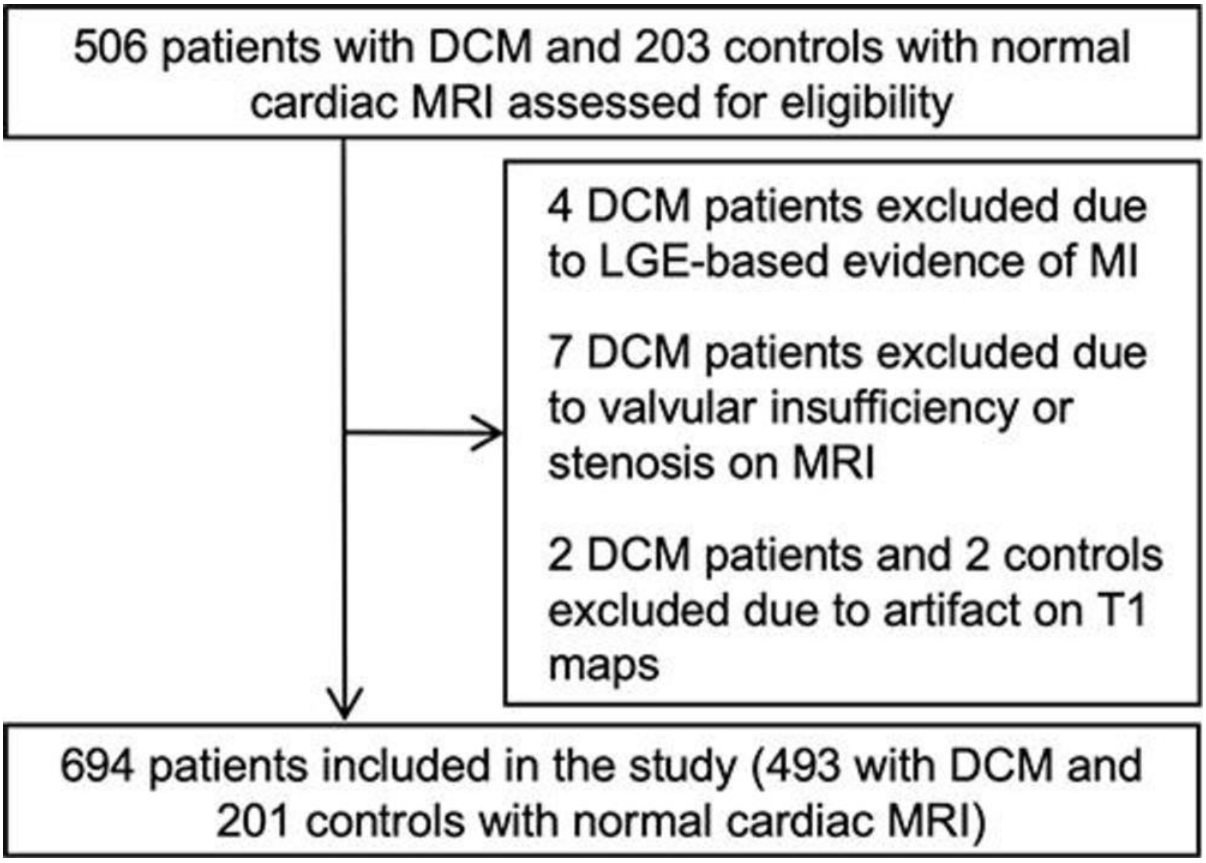

Figure 1. Flowchart details patient selection. An initial 506 eligible patients with dilated cardiomyopathy (DCM) and 203 controls with normal cardiac MRI findings were identified. Fifteen patients were excluded, and a total of 694 patients were included in the final study sample—493 with DCM and 201 controls. LGE = late gadolinium enhancement, MI = myocardial infarction
High-res (TIF) version

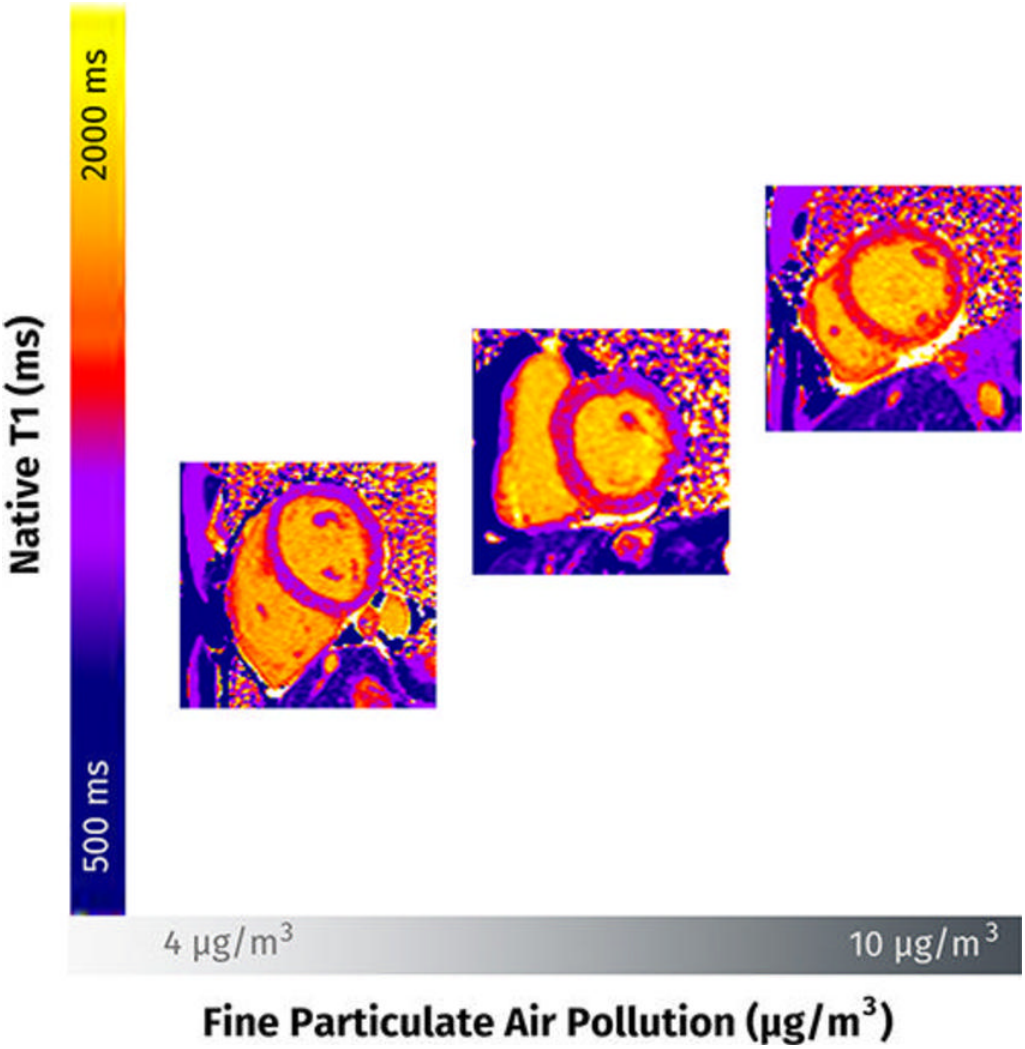

Figure 2. Images from cardiac MRI native T1 mapping show that higher long-term exposure to fine particulate air pollution is associated with higher extent of myocardial fibrosis.
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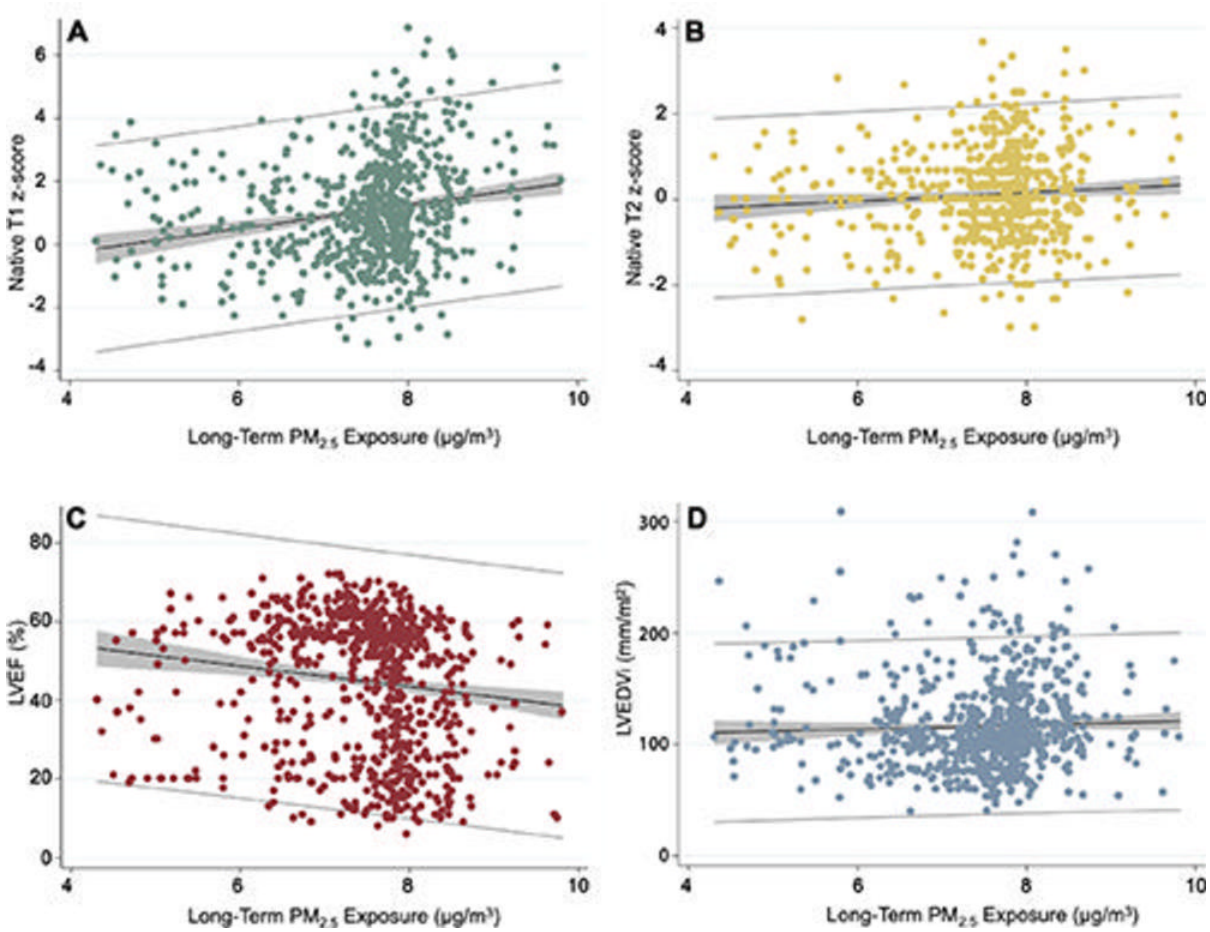

Figure 3. Scatterplots show the relationship between long-term exposure to fine particulate matter with 2.5-µm or smaller aerodynamic diameter ($PM_{2.5}$) and cardiac MRI parameters in all patients, including **(A)** native T1 z score, **(B)** native T2 z score, **(C)** left ventricular ejection fraction (LVEF), and **(D)** left ventricular end-diastolic volume indexed to body surface area (LVEDVi). Linear regression lines are shown in black, 95% CIs in gray shading, and 95% prediction intervals in blue. Each 1-µg/m3 increase in 1-year mean ambient $PM_{2.5}$ exposure was associated with a 0.38 higher native T1 z score (β coefficient: 0.38; 95% CI: 0.24, 0.51; P < .001).="">
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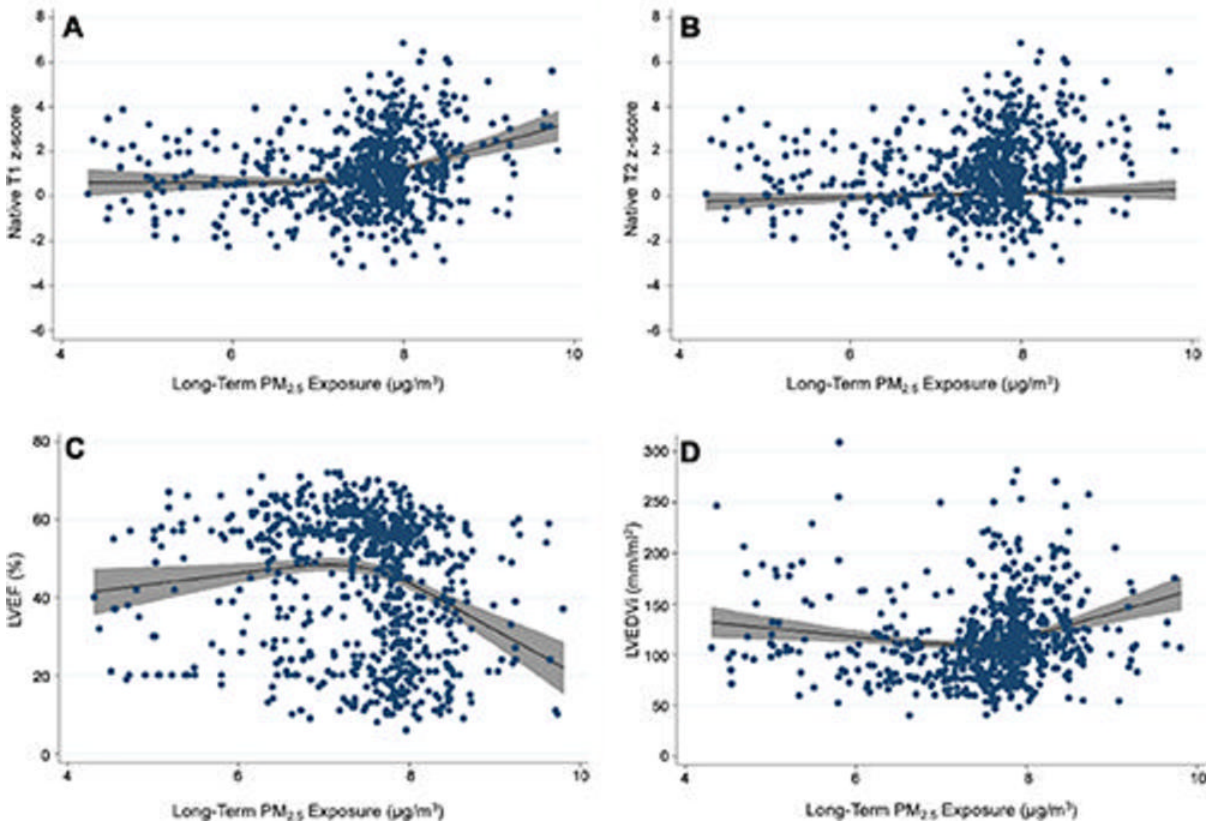

Figure 4. Scatterplots obtained with restricted cubic spline transformation (three knots) of 1-year mean fine particulate matter with 2.5-µm or smaller aerodynamic diameter ($PM_{2.5}$) exposure to investigate nonlinear relationships with cardiac MRI parameters in all patients, including **(A)** native T1 z score, **(B)** native T2 z score, **(C)** left ventricular ejection fraction (LVEF), and **(D)** left ventricular end-diastolic volume indexed to body surface area (LVEDVi). Regression lines are in black and 95% CIs in gray shading. Other than for left ventricular ejection fraction, there are no clear nonlinear relationships except for where there are few data points at extreme exposure values.
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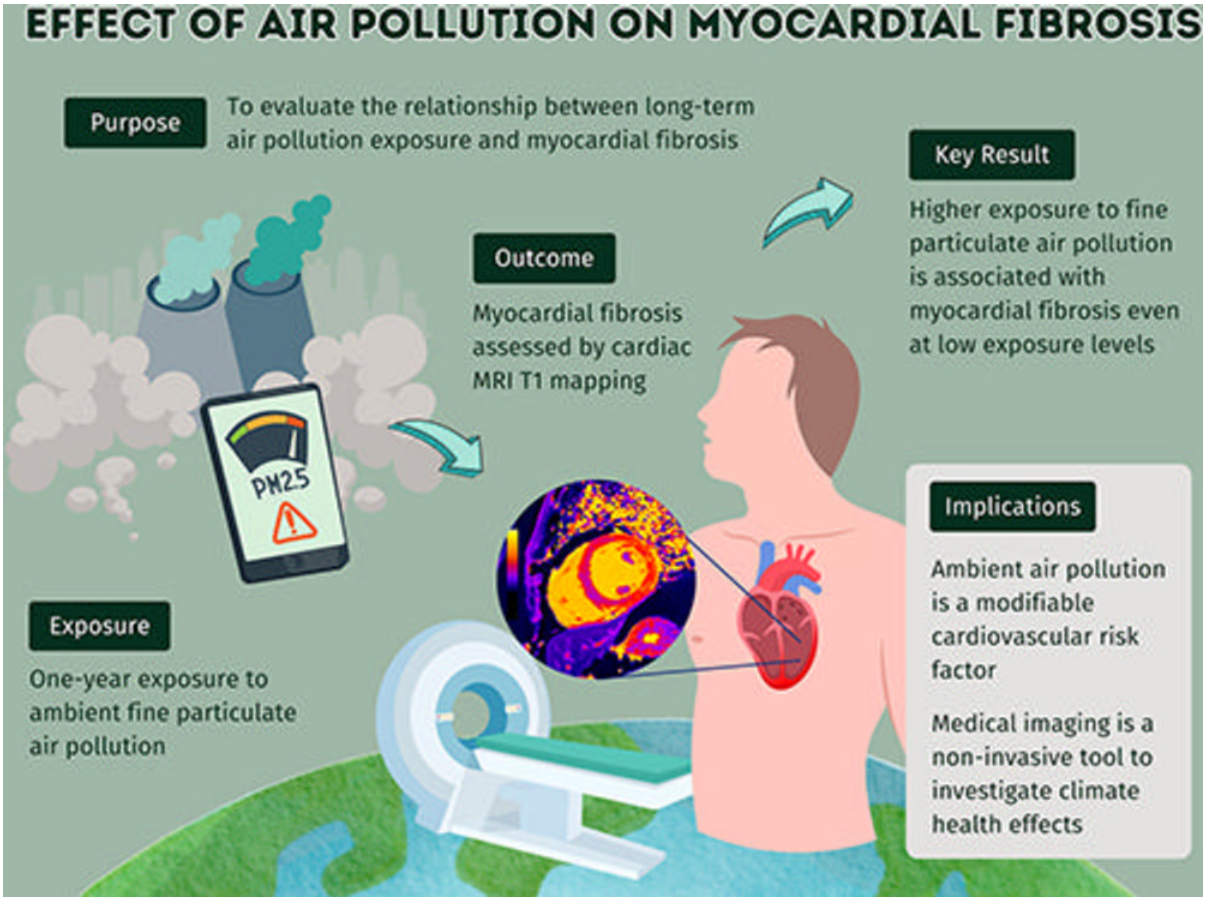

Figure 5. Diagram shows summary of study purpose, exposure, outcome, and key results. Higher long-term exposure to ambient fine particulate air pollution is associated with greater diffuse myocardial fibrosis at cardiac MRI native T1 mapping in patients with dilated cardiomyopathy and controls with normal MRI findings. $PM_{2.5}$ = fine particulate matter with 2.5-μm or smaller aerodynamic diameter.
High-res (TIF) version

Resources:

Editorial
Study abstract