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Study links air pollution to clogged arteries

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Got high cholesterol? You might want to stay away from air pollution. That's the message of a new UCLA study linking diesel exhaust to atherosclerosis, or hardening of the arteries, which significantly increases one's risk for heart attack and stroke.

Published in the July 26 edition of the online journal *Genome Biology*, the findings are the first to explain how fine particles in air pollution conspire with artery-clogging fats to switch on the genes that cause blood vessel inflammation and lead to cardiovascular disease.

"When you add one plus one, it normally totals two," said principal investigator Dr. André Nel, chief of nanomedicine at the David Geffen School of Medicine at UCLA and a researcher at UCLA's California NanoSystems Institute. "But we found that adding diesel particles to cholesterol fats equals three. Their combination creates a dangerous synergy that wreaks cardiovascular havoc far beyond what's caused by the diesel or cholesterol alone."

The researchers set up a scenario to investigate the interaction between diesel exhaust particles and the fatty acids found in low-density lipoprotein (LDL) cholesterol — the "bad" type of cholesterol that leads to artery blockage.

In particular, the team was interested in how oxidation — cell and tissue damage resulting from exposure to molecules known as free radicals — contributes to inflammation and artery disease. Free radicals enter the body through small particles present in polluted air and are also

byproducts of normal processes, such as the metabolic conversion of food into energy.

“Diesel particles are coated in chemicals containing free radicals, and the fatty acids in LDL cholesterol generate free radicals during metabolism in the cells,” said first author Ke Wei Gong, a UCLA cardiology researcher. “We wanted to measure what happens when these two sources of oxidation come into contact.”

The scientists combined the pollutants and oxidized fats and cultured them with cells from the inner lining of human blood vessels. A few hours later, the team extracted DNA from the cells for genetic analysis.

“We saw that the diesel particles and oxidized fats had worked in tandem to activate the genes that promote cellular inflammation — a major risk factor for atherosclerosis,” said Dr. Jesus Araujo, UCLA assistant professor of medicine and director of environmental cardiology at the Geffen School of Medicine.

“The interaction left a genetic footprint that reveals how interaction between the particles and cholesterol accelerates the narrowing and blockage of the blood vessels,” Araujo noted.

To duplicate these findings in living cells, the UCLA team exposed mice with high cholesterol to the diesel particles and saw activation of some of the same gene groups in the animals’ tissue.

“Exactly how air pollutants cause cardiovascular injury is poorly understood,” Nel said. “But we do know that these particles are coated with chemicals that damage tissue and cause inflammation of the nose and lungs. Vascular inflammation in turn leads to cholesterol deposits and clogged arteries, which can give rise to blood clots that trigger heart attack or stroke.”

The researchers' next step will be to convert the genes' responses to the pollutant-cholesterol combination into a biomarker that will enable physicians to easily evaluate air pollution's effect on health, especially cardiovascular disease.

"Once a biomarker is developed, we'd simply need to test a blood sample in order to measure a person's exposure to particulate matter and determine whether it has reached levels that require medical intervention," Araujo said.

The American Cancer Society has reported a 6 percent increase in heart- and lung-related deaths for every 10 micrograms per cubic meter rise in particulates.

"Our results emphasize the importance of controlling air pollution as another tool for preventing cardiovascular disease," Gong said.

Source: University of California - Los Angeles