

Ultrafine particles in emissions found to cause heart disease

Unregulated pollutants play important role in the development of conditions such as atherosclerosis

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Air pollution has been suggested as a risk factor for developing heart disease for several years, but a new UCLA study has found ultrafine particles from vehicle emissions that may lead to heart attack or stroke.

These unregulated ultrafine particles, potentially the most toxic air pollutant particles, are 10 times smaller than the fine-sized particle pollutants regulated by current vehicle emission standards.

In the first animal study of the impact of ultrafine particles, UCLA scientists, in collaboration with researchers from several other universities, determined that atherosclerosis was exacerbated by ultrafine particles more than by fine particles.

Atherosclerosis is an inflammatory process that involves a hardening of the arteries due to plaque buildup, and can lead to heart attack or stroke.

“Mice exposed to ultrafine particles developed a greater amount of lipid plaques than those exposed to larger pollutants, and this was the first time that ultrafine particles have been shown to promote fat plaque buildup in arteries,” said Jesus Araujo, an assistant professor of medicine and director of environmental cardiology at the David Geffen School of Medicine.

In the study, mice exposed to ultrafine particles from downtown Los Angeles freeway emissions showed 25 percent more atherosclerotic plaque development than mice exposed to fine sized particles, and 55 percent more plaque development than animals breathing filtered air.

Air pollutants inactivate the protective qualities of high density lipoprotein cholesterol, which is known as “good cholesterol” because it cleans up the artery walls by removing excess or damaged molecules, preventing swelling.

“With good genes and a good lifestyle, (the good cholesterol) handles the job well, but if the pressure is excessive and the (cholesterol) is not able to dump its waste, the trash-removing truck collapses and its function is compromised,” said Mohamad Navab, a professor of medicine. “Stimulatory molecules in the lungs such as pollutants contribute to excessive reactive oxygen species, leading to inflammation.”

Ultrafine particles are particularly harmful because they generate many more oxidant radicals – highly reactive molecules that cause injury to cells – than fine particles do. The radicals lead to inflammation, which in turn contributes to clogged arteries.

“When you have ongoing inflammation from other risk factors and you add additional oxidant stress stimuli on top of that, the inflammation is enhanced and you get a multiplicative effect, creating chronic disease processes that are dependent on inflammation,” said Andre Nel, chief of nanomedicine at UCLA.

The bigger fine-sized particles have previously been linked with increased heart disease, with an increase of 24 percent in the incidence of heart attack and stroke for each unit of increase of fine particle pollutants, Araujo said.

The experimental data from the new animal study, which shows that ultrafine particles are more toxic pollutants, could indicate that ultrafine particles may be associated with heart disease in humans, Araujo said.

The particles have only been tested in mice as of now, and although humans are more complex, this study has significant implications, Nel said

“Animal studies project for us one pathway humans might follow,” he said. “(Humans) start out with many more risk factors and propensities, and the more risk factors you add on, the more advanced the disease is likely to become. Air pollution is now one of those risk factors that we need to add on.”

While ultrafine particles have now been shown to be related to increased heart disease, currently there is no way to regulate their emission because they are too small to be captured on the filters available. New chemical technology is needed to measure the extent of their harmful effects, Nel said.

Previous studies on the impact of fine particles pollutants have shown that cardiovascular damage can occur in five to six months, but the current study demonstrated that damage may occur in as little as five weeks.

“We explored the effect in a shorter interval, and thought there was a good chance to see the same effect because we were studying more toxic particles,” Araujo said. “Going from five months to five weeks with more toxic particles was enough to show an increase in plaque buildup.”

Air pollution is now gaining attention as a risk factor for atherosclerosis in addition to the more established risk factors such as high cholesterol, obesity, smoking, inactivity and high blood pressure.

“Knowledge of disease is based on the knowledge of these risk factors, and air pollution may explain a percentage of the disease that is not explained by other factors,” Araujo said.

Air Pollutant Particles

The transmission electron microscopy images of fine (left) and ultrafine (right) particles reveal the differences in size and structure of the two dangerous particles found in air pollution, particularly those caused by vehicle emissions. The fine particle, at 400 nm, is ten times the size of the ultrafine particle and comes from soil, crustal elements and sea salt in the environment. The ultrafine particles have their source in vehicle emissions and the condensation of vapors and are not yet regulated by the Environment Protection Agency because they have only recently been studied.

