MedicalXpress

Jekyll into Hyde: Breathing auto emissions turns HDL cholesterol from 'good' to 'bad'

May 15 2013

Academic researchers have found that breathing motor vehicle emissions triggers a change in high-density lipoprotein (HDL) cholesterol, altering its cardiovascular protective qualities so that it actually contributes to clogged arteries.

In addition to changing HDL from "good" to "bad," the inhalation of emissions activates other components of oxidation, the early cell and tissue damage that causes inflammation, leading to hardening of the arteries, according to the research team, which included scientists from UCLA and other institutions.

The findings of this early study, done in mice, are available in the online edition of the journal *Arteriosclerosis, Thrombosis and Vascular Biology*, a publication of the American Heart Association, and will appear in the journal's June print edition.

Emission particles such as those from vehicles are major pollutants in urban settings. These particles are coated in chemicals that are sensitive to free radicals, which have been known to cause oxidation. The mechanism behind how this leads to atherosclerosis, however, has not been well understood.

In the study, the researchers found that after two weeks of exposure to vehicle emissions, mice showed oxidative damage in the blood and liver—damage that was not reversed after a subsequent week of receiving filtered air. Altered HDL cholesterol may play a key role in

this damaging process, they said.

"This is the first study showing that air pollutants promote the development of dysfunctional, pro-oxidative HDL cholesterol and the activation of an internal oxidation pathway, which may be one of the mechanisms in how air pollution can exacerbate clogged arteries that lead to heart disease and stroke," said senior author Dr. Jesus Araujo, an associate professor of medicine and director of environmental cardiology at the David Geffen School of Medicine at UCLA.

For the study, one group of mice was exposed to vehicle emissions for two weeks and then filtered air for one week, a second was exposed to two weeks of emissions with no filtered air, and a third was exposed to only clean, filtered air for two weeks. This part of the collaborative research took place at the Northlake Exposure Facility at the University of Washington, headed by study author Michael E. Rosenfeld.

"The biggest surprise was finding that after two weeks of exposure to vehicle emissions, one week of breathing clean filtered air was not enough to reverse the damage," said Rosenfeld, a professor of environmental and occupational health sciences and pathology at the University of Washington.

Mice were exposed for a few hours, several days a week, to whole diesel exhaust at a particulate mass concentration within the range of what mine workers usually are exposed to.

After the exposures, UCLA scientists analyzed blood and tissue specimens and checked to see if the protective antioxidant and anti-inflammatory properties of HDL, known as "good" cholesterol, were still intact. They used special analytical laboratory procedures originally developed by study author Mohamad Navab at UCLA to evaluate how "good" or "bad" HDL had become. The team found that many of the

positive properties of HDL were markedly altered after the air-pollutant exposure.

For example, the HDL of mice exposed to two weeks of vehicle emissions, including those that received a subsequent week of filtered air, had a much-decreased ability to protect against oxidation and inflammation induced by low-density lipoprotein (LDL) cholesterol, known as "bad" cholesterol, than the mice that had only been exposed to filtered air.

According to researchers, without HDL's ability to inhibit LDL, along with other factors, the oxidation process may run unchecked. Moreover, not only was the HDL of the mice exposed to diesel exhaust unable to protect against oxidation, but, in fact, it further enhanced the oxidative process and even worked in tandem with the LDL to promote even more oxidative damage.

Researchers also found a twofold to threefold increase of additional oxidation products in the blood of mice exposed to vehicle emissions, as well as activation of oxidation pathways in the liver. The degree of HDL dysfunction was correlated with the level of these oxidation markers.

"We suggest that people try to limit their exposure to air pollutants, as they may induce damage that starts during the exposure and continues long after it ends," said first author Fen Yin, a researcher in the division of cardiology at the Geffen School of Medicine.

The current research builds on the team's previous findings that ambient ultrafine particles commonly found in air pollution, including vehicle emissions, enhance the build-up of cholesterol plaques in the arteries and that HDL may play a role.

"Our research helps confirm that the functionality of HDL may be as

important to check as the levels," said study author Dr. Alan Fogelman, executive chair of the department of medicine and director of the atherosclerosis research unit at the Geffen School of Medicine.

Provided by University of California, Los Angeles