

○ How traffic pollution damages the heart

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Living close to a busy road can damage your heart - and now we're closer to understanding why.

Previous studies had suggested that people living in [polluted areas are more at risk of heart disease](#). For example, a study published in *The New England Journal of Medicine* last year showed that women in 36 American cities were [more likely to develop heart disease](#) if the air they breathed was rich in particles measuring 2.5 micrometres or less in diameter - known as PM2.5s - which are present in car exhaust fumes.

It now seems that a greater hazard may be posed by so-called "ultrafine" particles, about a dozen times smaller at 0.18 micrometres wide. The latest study in mice has shown that they clog up arteries with fatty atherosclerotic deposits, and chemically alter "good" cholesterol, or high-density lipoprotein (HDL) cholesterol, reducing its beneficial effects.

The results pose a dilemma for air quality regulators, for unlike PM2.5 particles and larger PM10s, which are already monitored and restricted, ultrafine particles are almost too small to detect or filter. "It has been shown before that PM2.5 is more dangerous than larger particles," says Andre Nel of the University of California at Los Angeles, who led the study. "Now we've added that ultrafines are probably even more dangerous than PM2.5."

For their experiment, Nel's team parked a special mobile laboratory near highway 110, one of the busiest roads in Los Angeles. For five weeks, mice breathed in fumes captured from the passing traffic, which had been concentrated into three separate streams. One contained clean air filtered of almost all its particles. A second stream contained the ultrafine particles and the third contained PM2.5s.

When they examined the blood vessels of mice exposed to the ultrafine particles, they found 55 per cent more atherosclerotic plaque than in mice exposed to clean air and 25 per cent more than in mice exposed to PM2.5s.

Moreover, lab tests on cells taken from human arteries showed that HDL cholesterol from the blood of mice that had breathed the ultrafine particles was less effective than normal at blocking inflammation triggered by oxidative damage to tissue (*Circulation Research*, [DOI: 10.1161/CIRCRESAHA.107.164970](#)). Such damage occurs throughout life as a result of normal cellular activity, but is usually repaired. The mice also showed evidence of oxidative damage to fat, and abnormally high activity of genes in the liver that combat oxidative damage.

The results imply that the ultrafine particles are coated with the products of fuel combustion, such as polyaromatic hydrocarbons, which cause oxidative damage in the body. For example, HDL loses its protective effect if it is oxidised, says Nel.

"They've confirmed prior findings that concentrating particles can accelerate atherosclerosis, and shown that the smallest particles seem most effective at doing this," says Joel Kaufmann of the University of Washington in Seattle, who led the *New England Journal of Medicine* study.

Ultrafine particles might cause more damage than PM2.5s because their collective surface area is greater, meaning that they can transport more tissue-damaging chemicals into the body, Nel says. There are between 100 and 1000 times more ultrafine particles than PM2.5s in car exhaust fumes. "They are

also smaller, and so penetrate deeper into the lung, and they're retained there longer," says Nel.

The big question is whether the ultrafine particles are so small that they could sneak through pores in the lung lining and into the bloodstream. "Animal studies suggest they can, but human studies are inconclusive," says Jon Ayres at the University of Aberdeen, UK, who chairs the UK government's Committee on the Medical Effects of Air Pollutants. Two years ago the committee produced a report warning of a possible link between ultrafine particles and cardiovascular disease.

"This latest study suggests that the ultrafine fraction plays a particularly strong role in the oxidative and bloodstream inflammatory effects," says Nino Künzli of the Centre for Research in Environmental Epidemiology in Barcelona, Spain, who showed in 2005 that women in Los Angeles who were exposed to PM_{2.5} particles in their homes had thicker artery walls - a marker of atherosclerosis.

"This study suggests that ultrafine pollution plays a particularly strong role in oxidative and blood inflammatory effects"

However, Künzli and others warn that it is too early to presume that what happens in mice will also happen in people. For example, because the particles were concentrated before the mice inhaled them, the levels the mice breathed in were up to six times as high as those to which commuters in Los Angeles are regularly exposed. Also, the mice had been genetically engineered to develop plaques more quickly than normal, meaning it would likely take longer for similar effects to materialise in humans.

The findings nevertheless pose awkward questions for regulators and car manufacturers, which are already struggling to enforce or meet existing standards for PM_{2.5}.

"This new, important study shows that ultrafine particles may be of greater concern than larger ones," says George Gray of the US Environmental Protection Agency's Office of Research and Development. "This study will be carefully considered as the EPA's Office of Air moves forward with its regular review of the existing PM regulations," he says.

The other bad news is that anti-pollution masks won't block out ultrafine particles, although some car manufacturers in Europe are now fitting vehicles with electrostatic devices that can precipitate them out.