MedicalXpress

If smoker has COPD, quitting might not help lung function

July 27 2016, by Molly Schulson

Quitting cigarettes may not improve smokers' lung function if they have already begun to develop chronic obstructive pulmonary disease, according to new research from Weill Cornell Medicine. The findings illustrate how cigarettes affect the lungs on a cellular level – which, the investigators hope, will help persuade smokers to stop as early as possible.

"The addiction to nicotine is very powerful," said senior author Dr. Ronald Crystal, chairman of the Department of Genetic Medicine, the Bruce Webster Professor of Internal Medicine and a professor of medicine at Weill Cornell Medicine. "But the earlier you can stop smoking before developing COPD, the better chance you will have at healthy lungs."

Endothelial cells, which form the lining of blood vessels, shed small bubble-like particles of plasma membrane called circulating endothelial microparticles, or EMPs. This process becomes accelerated when programmed cell death – called apoptosis – occurs; researchers can identify apoptotic EMPs by looking for specific antibody markers in the blood.

Weill Cornell Medicine investigators previously found that smokers have elevated levels of apoptotic EMPs coming from pulmonary capillaries compared with nonsmokers. To determine what happens to a smoker's apoptotic EMP levels after they cease smoking, researchers in the new study, published July 26 in *Thorax*, assessed the total and apoptotic EMP

levels of a group of 138 nonsmokers, healthy smokers and COPD smokers. After baseline EMP levels were recorded, 17 healthy smokers and 18 COPD smokers successfully quit smoking. All subjects' EMP levels were then collected after three, six and 12 months.

Researchers found that both healthy and COPD smokers had elevated levels of apoptotic EMPs compared with nonsmokers, but only healthy smokers were positively impacted by smoking cessation. "When healthy smokers stopped smoking, their levels of apoptotic EMPs dropped back down to normal levels. But for COPD smokers, their EMP levels did not," Crystal said.

COPD, which causes partially irreversible airflow obstruction, is the third most common cause of death in the United States. Once a person develops the disease, lung function declines at an accelerated rate. "Studying the mechanisms by which COPD occurs, particularly very early on, is important so that we can target drugs that may be effective in terms of stopping the progression of the disease," Crystal said.

The Global Initiative for Chronic Lung Disease has set the standard for diagnosing COPD and determining its progression. Out of GOLD's four classifying stages of COPD, which range in intensifying severity from GOLD I to IV, most COPD participants were at GOLD I and II. "We purposely looked at people with the mildest form of the disease because it gives better insight into the mechanism," Crystal said. "When the disease is more severe, there are many pathological mechanisms, so it's harder to sort out what's going on."

Crystal added that this research is also important when it comes to developing COPD biomarkers, which are substances in the blood that provide doctors with insight into how certain organs, such as the lungs, are doing.

Provided by Cornell University