MedicalXpress

Study reveals COPD linked to increased bacterial invasion

April 27 2016

Chronic obstructive pulmonary disease (COPD) is a common smoking-related lung illness and the third leading cause of death in the United States. Scientists have long believed that inhaling toxic gases and particles from tobacco smoke causes inflammation of the small airways in the lungs, leading to the development of COPD. However, the theory doesn't explain why airway inflammation and disease progression continue even after the patient stops smoking.

Now a study led by Vanderbilt University Medical Center (VUMC) investigators finds that persistent inflammation in COPD may result from a defect in the immune system that allows airway bacteria to invade deeper into the lung. The research led by first author Bradley Richmond, M.D., instructor in the Division of Allergy, Pulmonary and Critical Care, and senior authors Timothy Blackwell, M.D. and Vasiliy Polosukhin, M.D., Ph.D. was published online April 5, 2016 in *Nature Communications*.

The investigators were interested in the function of the body's mucus membranes like those found in the nose and the lining of the lungs. Mucosal surfaces normally have an elaborate barrier that prevents bacteria from invading into deeper tissues. Having previously shown that patients with COPD often lack a key component of this barrier called secretory immunoglobulin A (secretory IgA), the investigators studied mice lacking secretory IgA. While these mice appeared healthy at birth, as they aged they developed increased bacterial invasion, chronic inflammation, and damage in the lungs similar to patients with COPD.

These changes occurred without cigarette smoke or other exposures."These findings suggest that over time changes in the airways resulting from cigarette smoke exposure make the lungs more susceptible to bacterial invasion. This may explain why inflammation persists in COPD even after patients stop smoking," said Richmond.

Treatment with the anti-inflammatory drug roflumilast halted lung damage in secretory IgA-deficient mice, suggesting a possible future therapy for patients with COPD and decreased secretory IgA in small airways.

Provided by Vanderbilt University Medical Center