New asthma biomarkers identified from lung bacteria

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Dr. Patricia Finn, the Earl M. Bane Professor of Medicine at the UIC College of Medicine Credit: UIC/Jenny Fontaine

While the microbiome has gained significant attention for its impact on digestive health in recent years, its effect on lung disease has largely

remained unstudied.

Dr. Patricia Finn says this is a missed opportunity.

"The microbiome is the ecosystem of good and bad bacteria living in the body," said Finn, the Earl M. Bane Professor of Medicine at the University of Illinois at Chicago. "Because the lungs continuously and automatically draw air, and any number of environmental agents, into the body, the composition and balance of microbes in the lungs may have a profound effect on many respiratory conditions."

New research from Finn and her colleagues in the UIC College of Medicine suggests that the lung microbiome plays a significant role in asthma severity and response to treatment.

Asthma is a chronic disease in which lung airways become swollen and narrow, making it difficult for air to move in and out of lungs. Because people with asthma have inflamed airways, they experience a range of symptoms, including shortness of breath, coughing, wheezing and chest tightness.

In a group of clinically similar patients with asthma, researchers identified two asthma phenotypes by assessing the microbiome and airway inflammation. The patients were ages 18 to 30 with mild or moderate atopic asthma.

"This tells us the microbiome has relevance beyond the gut, and that it is a potential biomarker for asthma," said Dr. David Perkins, professor of medicine and surgery at UIC, who jointly operates the lab with Finn.

These two phenotypes, called asthma phenotype one and two, or AP1 and AP2, are demarcated by the prevalence and dominance of different bacteria in the lung. When compared, patients in the two groups

performed differently on pulmonary function tests.

AP1 was associated with less [severe asthma](link); it showed decreased T helper cytokines and increased enterococcus bacteria, but normal pulmonary function tests. In contrast, AP2 was associated with increased pro-inflammatory cytokines, increased oral taxa and strep pneumonia bacteria, and decreased [pulmonary function tests](link), or more severe asthma.

In both AP1 and AP2, the associations between the composition of the microbiome and specific inflammatory cytokines were decreased after treatment with an inhaled corticosteroid, a common asthma therapy. Researchers say this suggests that ICS may function by dampening responses to microbes.

"The data suggest that further study of the microbiome may help to develop more personalized treatment recommendations for patients with asthma," said Finn, the senior author on the paper, which is published in *PLOS ONE*.

Finn says that asthma research has increasingly focused on the differences between seemingly similar patients, and that this study adds to the growing body of evidence that patients benefit from precision medicine approaches to common chronic diseases, such as asthma.

"If we can better understand how the individual's lung microbiome affects asthma and identify likely microbial culprits, we may get to a point where we can predict and control asthma development and severity by shifting the microbiome early in life," Finn said. "This could be as simple as diet, probiotics or medication."

A second study from the Finn-Perkins lab at UIC, recently published in the *American Journal of Respiratory Cell and Molecular Biology*, showed

that even prior to birth, exposure to specific compositions of bacteria may elicit an inflammatory immune response that could predispose individuals to asthma.

"This is an exciting time in scientific discovery," Finn said. "We are learning that the lung microbiome potentially plays a role in many parts of the body including cord blood cells, which may impact disease onset or symptoms."

More information: Benjamin A. Turturice et al, Atopic asthmatic immune phenotypes associated with airway microbiota and airway obstruction, *PLOS ONE* (2017). DOI: 10.1371/journal.pone.0184566

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