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Cystic Fibrosis Research News

Title:

Mucoid Pseudomonas aeruginosa and regional inflammation in the cystic fibrosis lung

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What was your research question?

In this study, we asked two main research questions: 1) Where are nonmucoid and mucoid forms of the bacterium, *Pseudomonas aeruginosa*, located in the lobes of cystic fibrosis (CF) lungs? 2) How do these types of Pseudomonas affect inflammation in different lobes of the CF lungs?

Why is this important?

Long-term bacterial infections and an inflammation response by the immune system cause lung tissue damage in CF. Pseudomonas is one bacterium that commonly infects CF patients. There are at least two forms of Pseudomonas found in CF: nonmucoid and the more aggressive, mucoid form. Research suggests that the upper lobes of CF lungs are more damaged compared to the lower lobes; however, the reasons for upper-lobe concentrated disease is poorly understood. We predicted that nonmucoid and mucoid Pseudomonas are found in different lung lobes, have distinct effects on inflammation, and may explain upper/lower lobe differences in CF lung disease.

What did you do?

We obtained fluid from the lungs of CF patients during a routine procedure called bronchoscopy. This procedure involves a bronchoscope, a flexible tube which is passed through the mouth and used to enter each lobe of the patient's lung. Then, fluid was instilled and removed from each lung lobe. That fluid was analyzed for the presence of mucoid and nonmucoid Pseudomonas as well as indicators of the inflammation immune response.

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What did you find?

Mucoid and nonmucoid Pseudomonas were found in all lobes of the CF lung, with no preference for upper or lower lobes. Additionally, without considering the type of bacterial infection, inflammation was slightly elevated in the upper lobes compared to the lower lobes. Importantly, any lobes infected with mucoid Pseudomonas were more inflamed compared to those infected with nonmucoid Pseudomonas (or with no Pseudomonas).

What does this mean and reasons for caution?

We showed that infection with mucoid Pseudomonas, regardless of the lung lobe, causes inflammation in the CF lung. This is important because inflammation is linked to tissue damage and if mucoid Pseudomonas is a culprit in this process, these bacteria should be specifically targeted with existing and new therapies for CF patients. Our study was limited by a small set of 14 patients. Additionally, bacteria other than Pseudomonas, which also infect CF patients, were not investigated here.

What's next?

In future studies, we hope to recruit a larger group of patients and to consider bacteria other than Pseudomonas, which may also affect inflammation in CF lung lobes. Additionally, we may collect fluid from CF patients over time to see how infection and the immune response change with age.

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