

Cystic Fibrosis Research News

Title:

Key inflammatory markers in bronchoalveolar lavage predict bronchiectasis progression in young children with CF

Lay Title:

Markers of inflammation in lung fluid can predict lung damage in young children with Cystic Fibrosis (CF)?

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What was your research question?

Can markers of inflammation in lung fluid predict progression of lung damage in young children with CF?

Why is this important?

The most important reason for shortened life expectancy in CF is the progressive damage to the lungs caused by a vicious circle of infection and inflammation. It has been shown that lung

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damage already starts at a young age, and that by the age of 5 years around 60% already show abnormal widening of the airways, which are called bronchiectasis. Prevention of lung damage is an important goal for the care of children with CF. Markers that can identify children more at risk for developing bronchiectasis would help in the decisions for treatment and can help improve their long-term outcome.

What did you do?

In 37 children aged 1 or 3 years we collected lung fluid, by doing bronchoscopies, and images of the lungs by making chest-CT scans. Chest CT scans were repeated 2 years later. In the lung fluid we measured several markers of inflammation, such as neutrophils (specific inflammatory white blood cell), and cytokines, which are substances excreted by activated inflammatory cells. The 2 consecutive chest CT scans were scored for presence of bronchiectasis.

What did you find?

We found that interleukin 8 (IL-8) is the best marker of inflammation in lung fluid to predict a more than average progression of bronchiectasis 2 years later. IL-8 is a substance produced by different cells in the body that attracts neutrophils. Also other markers such as number of neutrophils, MPO (a substance excreted by activated neutrophils) in lung fluid were associated with increase of bronchiectasis.

What does this mean and reasons for caution?

The findings help in understanding the causes for development of bronchiectasis in young children with CF. It also shows that inflammation is already present at a young age, which needs intervention to prevent lung damage. Treatment should start from birth, and using inflammatory markers can help identify the children that need intervention the most. Limitations of this study is that it is done in a relatively small number of children. Furthermore, the study is done in lung fluid, which is retrieved by doing bronchoscopies. This required anaesthesia and is therefore rather invasive, and not something that is used routinely.

What's next?

Future research should focus on ways to identify inflammation in the lungs of children in a non-invasive way. Further unravelling the mechanisms of inflammation which leads to lung damage, may reveal targets for the development of new therapies.



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