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Cystic Fibrosis Research News

Title:

Mast cell tryptase changes with Aspergillus fumigatus – host crosstalk in cystic fibrosis patients

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What was your research question?

In people with cystic fibrosis (CF), Aspergillus fumigatus (AF), a common airborne fungus, can cause allergic or infectious disease of their lungs. Mast cells (MC) are cells lining the bronchial tissue of the lung and are involved in host immune system defense and allergy. We sought for a biological marker of altered MC activity, which would indicate possible infection of CF sufferers with AF disease.

Why is this important?

Lung function is a major determinant of quality of life for people with CF. Recognizing and harnessing MC activation during AF disease might help preserve lung function in CF patients. We have previously shown that in people with CF, the allergic effects of AF, can be tracked by analysing particular antibodies in patients (IgE and IgG).

What did you do?

We took advantage of the availability of a blood serum marker of MC activity, the enzyme tryptase, in order to study the effect of AF on MC activity in CF patients. We measured

tryptase, IgG and IgE responses to AF molecules and collected clinical and laboratory data in 76 CF patients (children, adults with native lungs, and adults with lung transplantation).

What did you find?

Overall, we identified a decrease in circulating blood tryptase in adults (with native or transplanted lungs) and children with CF, who were sensitized to AF, i.e. who display an immune response (IgE+) to AF compared to those who were not sensitized to AF (i.e. IgE-). Second, we found that in people who are not sensitized to AF (IgE-) but instead display an IgG immune response (IgE+) to this mold, there was an association between levels of tryptase and IgG to AF. The authors speculate that lung transplantation acts as a rest-to-baseline event for lung responses to AF.

What does this mean and reasons for caution?

Sensitization, i.e. making IgE, is necessary for allergy. Our findings suggest that during this phase, MC activity is impaired, as reflected by a lower level of the enzyme tryptase. Our data also suggest that MC are involved in the changes in antibody ratios observed (IgG-IgE transition) during the interaction between AF and its CF host. Changes in tryptase levels could be a biological marker of ongoing disease processes involving AF. Caution is needed because such complex processes need larger-scale studies and validation in different patient populations to be completely proven.

What's next?

We aim at extending our research with studies looking at how the MC and AF mold interact at a molecular level as well as search for more biomarkers involved in MC activity.

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