European Cystic Fibrosis Society

Journal of Cystic Fibrosis
The Official Journal of the European Cystic Fibrosis Society

Cystic Fibrosis Research News

Title:

Differential Expression of Genes and Receptors in Monocytes from Patients with Cystic Fibrosis.

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What was your research question?

Macrophages, a white blood cell, are not able to switch off inflammation in patients with CF. Another white blood cell, the monocytes, made in the bone marrow turn into macrophages when needed to fight infection. We wanted to see whether monocytes were responsible for the macrophage problem in CF.

Why is this important?

Lung disease starts very early in life in children with CF but we don't know why. White bloods cells go to the lungs to fight infections by causing inflammation. Too much inflammation contributes to lung damage and the loss of lung function that is seen in older children and adults with CF. Macrophages are also responsible for switching off inflammation, but this does not seem to happen in CF lungs. If we can understand why macrophages can't switch off inflammation, we may be able to design treatments to fix this and slow down or prevent lung disease progression.

What did you do?

We took blood samples from patients with CF and from healthy subjects and separated out the monocytes. We then measured markers on the surface of the monocytes to tell what type

they were, measured what genes were active inside them and what cellular pathways were active.

What did you find?

We found that CF monocytes were different from those from the healthy subjects in some important ways. We found no differences in the types of monocytes in CF. We did, however, find that some of the active genes were different, some different genes were switched on and some active cellular pathways were different in CF monocytes, compared to monocytes from healthy subjects.

What does this mean and reasons for caution?

Our findings suggest that some of the differences we found maybe part of CF. If this is true we might be able to correct these by restoring normal function of CFTR, the protein that is not made normally in CF and that is responsible for the problems we see in CF. However, we did not prove that abnormal CFTR function was responsible for the differences. No new treatments will come from our research, at least not yet. Right now this is an interesting observation that needs further study to understand what it means.

What's next?

Our next steps are to look closely at how monocytes turn into macrophages and to see whether we can influence them to turn into the "switch-off" inflammation type. We will look at potential treatments that work by influencing control pathways inside the monocytes and don't rely on normal CFTR function.

Original manuscript citation in PubMed

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