



Cystic Fibrosis Research News

Citation:

Lam, et al. Increased expression of anion transporter SLC26A9 delays diabetes onset in cystic fibrosis. J Clin Invest 2019.

What was your research question? (50 words maximum)

Our research goal was to determine how a gene called SLC26A9 modifies the age at which individuals with CF get diabetes.

Why is this important? (100 words maximum)

As individuals with CF live longer, age-dependent complications, including diabetes, are becoming more common. Cystic fibrosis-related diabetes (CFRD) affects approximately 20% of adolescents and 40-50% of adults with CF. The development of diabetes is associated with increased medical complications and risk of death. A previous study from our group suggested that SLC26A9, a chloride channel found in pancreatic cells, may be a modifier of CFRD. The current study provided new evidence that the SLC26A9 chloride channel can alter the age at onset of diabetes in CF.

What did you do? (100 words maximum)

We performed several studies to show that the SLC26A9 gene was a good candidate for delaying the age at onset of CFRD. We then performed studies in cells from a pancreas to see whether the delay was due to an increase or decrease in the amount of SLC26A9 chloride channel.

What did you find? (100 words maximum)

The study showed that increasing the level of the SLC26A9 chloride channel over the lifetime of an individual with CF delays the onset of diabetes. The study also emphasized the key role in diabetes risk played by the exocrine pancreas. This is the part of the pancreas that makes digestive enzymes and is most affected in CF.





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What does this mean and reasons for caution? (100 words maximum)

The results provide new insights into the factors that alter when a person with CF will get diabetes. However, the SLC26A9 chloride channel is only one of a number of factors that alter diabetes risk in CF.

What's next? (50 words maximum)

Identifying additional modifier genes that contribute to the risk of diabetes and developing strategies to therapeutically target SLC26A9.

Original manuscript citation in PubMed

https://pubmed.ncbi.nlm.nih.gov/31581148/