Mucous breakthrough in mice holds promise for cystic fibrosis

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A London, Canada scientist studying cystic fibrosis (CF) has successfully corrected the defect which causes the overproduction of intestinal mucous in mice. This discovery by Dr. Richard Rozmahel, a scientist with the Lawson Health Research Institute, affiliated with The University of Western Ontario, has clear implications to understanding and treating this facet of the disease in humans. CF is a fatal, genetic disease characterized by an overproduction of mucous in the lungs and digestive system.

Rozmahel and his colleagues are identifying secondary genes that could contribute to CF, and measuring their impact on the disease. More specifically, they are investigating the potential of a gene found in mice, mCLCA3, which is similar to one in humans that exhibits abnormal levels in CF. The mCLCA3 gene is expressed by cells that produce and secrete mucous.

The researchers discovered that mCLCA3 plays an important role in the property of mucous, thereby allowing it to be cleared rather than result in the blockages that underlie CF. By correcting the abnormal levels of mCLCA3 in CF mice they were able to overcome the mucous lesions. Whereas CF mice normally do not survive more than 4 weeks as a consequence of the mucous disease, the animals where mCLCA3 levels were corrected could live a normal lifespan.

"It's my hope to understand what is causing the exaggerated mucous production and secretion in CF patients," says Dr. Rozmahel. "From

there, we can figure out ways to correct it."

Source: University of Western Ontario