

Researchers make promising finding in severe lung disease

29 June 2008

Researchers at the University of Illinois at Chicago have identified a novel function for an enzyme that plays a role in the tissue injury in acute respiratory distress syndrome, also known as ARDS.

The finding offers a new therapeutic target for the prevention and treatment of lung inflammation and injury. The research will be published in the journal *Nature Immunology* later this year and online June 29.

ARDS is an often fatal complication of bacterial infections, blood transfusions, overdoses of some medications, or traumatic injury. According to the National Heart, Lung, and Blood Institute, it affects nearly 150,000 people each year in the United States.

In ARDS, the lungs become swollen with water and protein, and breathing becomes impossible, leading to death in 30 percent to 40 percent of cases. There is no effective treatment.

It has previously been shown that the enzyme, called nonmuscle myosin light-chain kinase, or MYLK, plays a pivotal role in the disruption of the endothelial barrier -- a single thin layer of cells that line blood vessels -- which prevents water and protein from accumulating in tissues.

In addition to the disruption of the endothelial barrier and build-up of water in lungs in ARDS, a circulating blood cell, the neutrophil, "migrates into lung tissue and, when activated, can cause profound injury," said Jingsong Xu, assistant professor in pharmacology and dermatology and lead author of the paper.

Neutrophils are the most common type of white blood cells and are critical to what is called the innate immune response. They normally engulf and destroy invading bacteria and fungi and act as the first line of immune system defense.

In acute respiratory distress syndrome, they misfire and attack healthy tissue.

"Although there have been many studies into how MYLK disrupts the endothelial barrier, no one has investigated how MYLK functions to regulate the neutrophil transmigration into tissues," said Xu. "We decided to look at this."

The researchers found that MYLK was essential to the movement of neutrophils through the endothelial barrier. It unleashes a cascade of molecular events inside the neutrophil that changes the cell's shape, which is necessary for adhesion and migration.

"To our surprise, the pathway was a completely novel one that did not involve the well-studied and expected target of (the enzyme)," Xu said.

The unexpected finding of a novel pathway "opens up a completely new set of possible therapeutic targets for the prevention and treatment of this deadly disease," said Dr. Asrar Malik, distinguished professor, head of pharmacology and co-author on the paper.

Source: University of Illinois at Chicago

APA citation: Researchers make promising finding in severe lung disease (2008, June 29) retrieved 11 October 2022 from <https://medicalxpress.com/news/2008-06-severe-lung-disease.html>

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