Interferon might help asthma patients breathe easier, study suggests

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An immune-system protein already used to treat diseases like multiple sclerosis, hepatitis C and a variety of cancers might also aid asthma patients, UT Southwestern Medical Center researchers have found.

The investigators determined that the protein interferon blocks the development of a population of immune cells known to cause asthma. These cells are members of a class of T lymphocytes, called T helper 2 cells, or Th2 cells. Under normal circumstances, Th2 cells help protect against infections by secreting chemicals that induce inflammation; however, in some individuals, these Th2 cells can also promote allergic responses to normally harmless substances, including animal dander, pollens and pollutants. Once Th2 cells become reactive to these substances, they promote all of the inflammatory processes common to allergic diseases like asthma and atopic dermatitis.

The findings, available online and in the July 15 issue of the *Journal of Immunology*, suggest that interferon might be a valuable and readily available therapy for individuals with asthma.

"This finding is incredibly important, because humans are being treated with interferon for a variety of diseases, yet no one has tried treating asthma patients with interferon," said Dr. J. David Farrar, assistant professor of immunology and molecular biology at UT Southwestern and senior author of the study. "The current therapies for asthma are inhalers and steroids, both of which offer only temporary relief."

Asthma results in approximately 200,000 pediatric hospitalizations each year, more than for any other childhood disease. About 20 million people have been diagnosed with asthma in the U.S.

In the current study, the researchers showed in isolated human cells that interferon blocks the development of nascent Th2 cells and inhibits cells that already have become Th2 cells by interfering with a regulatory protein called GATA3, a transcription factor Th2 cells express to regulate their function.

"Interferon is blocking the development of these cells and their stability, and it's doing this by targeting the very transcription factor that regulates their development and stability in the first place," Dr. Farrar said. "By targeting this transcription factor, we've turned off the key component that regulates the entire process."

The findings, he said, provide proof-of-principle that targeting this particular group of cells with interferon might be an effective therapy for those with asthma.

"The study has confirmed that it's the Th2 cells that you really want to target," he said. "If you can stop a Th2 cell from ever developing, and if you can take a Th2 cell that has already become one and stop it from secreting these asthma-causing chemicals, then that's really the 'Holy Grail' of treating asthma."

The next step, Dr. Farrar said, is to study whether interferon will prevent Th2 cells taken straight from asthma patients from secreting the chemicals known to induce asthma.

"If interferon works against these cells, I think that would be an excellent basis for beginning a clinical trial and treating asthma patients," Dr. Farrar said. "We've been treating humans with interferon for a long time,

so we don't have to go through early-phase safety trials. We already have information about its toxicity."

Provided by UT Southwestern Medical Center