

Experimental nonsteroidal treatment of asthma shows promise

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A new nonsteroidal, anti-inflammatory therapy made from a human protein significantly decreases disease signs of asthma in mice, opening the possibility of a new asthma therapy for patients who do not respond to current steroid treatments. Results of this therapy in an animal model were presented at The Endocrine Society's 92nd Annual Meeting in San Diego.

The protein, insulin-like growth factor binding protein-3 (IGFBP-3), uniquely inhibits specific physiological consequences of <u>asthma</u> examined in asthmatic mice, said Youngman Oh, PhD, a study co-author and a professor of <u>pathology</u> at Virginia Commonwealth University, Richmond, Virginia.

IGFBP-3 reportedly targets a key cellular pathway called nuclear factor kappa B, or NF- κ B that plays a role in inflammation. The IGFBP-3 protein interferes with its cellular signaling and suppresses NF- κ B activity.

"This novel mechanism has never been identified before. Our findings could have major implications not only for asthma but also other inflammatory diseases that NF- κ B plays a role in, such as atherosclerosis and rheumatoid arthritis," Oh said.

In asthma, when the airways become inflamed, they become hyperreactive, or overly sensitive, to "triggers," such as dust, smoke and pet dander. This leads to a chain of reactions that elicit an asthma



"attack". According to the American Lung Association nearly 23 million people have asthma, of which 9 million are children.

"Anti-inflammatory corticosteroid medicines are an important part of asthma management for many people, but an estimated 20 percent of patients with asthma are resistant to existing steroid medications and there is a critical need for alternate therapies," Oh said.

Using a mouse model, Oh and his colleagues showed that IGFBP-3 production is suppressed in asthma. They measured NF- κ B inflammatory activity, using molecular and cellular techniques, and found that treatment with IGFBP-3 blocked NF- κ B activity.

The researchers administered IGFBP-3 to the mice by spraying a synthetic form of the <u>protein</u> into their opened trachea. The treatment "reduced all physiological manifestations of asthma," including airway inflammation and hyperreactivity, Oh said. His research team plans to study IGFBP-3 treatment in asthmatic canine models next.

Provided by The Endocrine Society

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