Suppression of Allergic Lung Inflammation and Hyperactivity

Tech ID: 24415 / UC Case 2014-228-0

ABSTRACT

Phosphorylated Myrisolyated Alanine-rich C Kinase Substrate (MARCKS) is elevated in human and animal asthmatic tissues. In addition, unphosphorylated MARCKS is also the key molecule to trap PIP2 and to interact with PI3K and Src at cell membrane. Researchers at the University of California, Davis have developed a novel, non-toxic, and water soluble peptide, targeting MARCKS' phosphorylation site domain, that inhibits MARCKS activity and retains PIP2 pool to suppress PIP3 production. As a result, the peptide treatment is capable of suppression of allergic lung inflammation and hyper-reactivity.

FULL DESCRIPTION

Myrisolyated alanine-rich C kinase substrate (MARCKS), a substrate of protein kinase C, is a key regulatory molecule controlling mucus granule secretion by airway epithelial cells as well as directed migration of leukocytes, stem cells, and fibroblasts. Previous research has shown that the non-canonical function of MARCKS relies on its ability to stay in membrane to trap PIP2 in allergic asthma. This activity is lost if MARCKS is phosphorylated and dissociated from the membrane. UC Davis researchers have shown elevated phosphorylated MARCKS associated with human and animal asthma tissues. Thus, inhibiting MARCKS signaling activity can potentially be therapeutic points of intervention for treating asthma.

Researchers at the University of California, Davis have developed a novel non-toxic and water soluble peptide which targets MARCKS' phosphorylation site domain and inhibits MARCKS phosphorylation and its activity. UC Davis researchers have shown that this novel peptide is able to suppress allergic lung asthma symptoms including airway Inflammation, hyper-reactivity and mucous cell metaplasia in mice models *in vivo*. The peptide is effective at a nanomole level with no cytotoxicity to normal as well as asthmatic airway epithelia. Mechanistically, this peptide can reduce PIP3 pools and phospho-MARCKS levels through trapping membrane PIP2, leading to blocking PIP3-mediated signaling networks such as PI3k/AKT, PDK1, GRP1 and ARNO signaling.

APPLICATIONS

- Therapeutic potential to suppress allergic airway asthma

FEATURES/BENEFITS

- Non toxic
- Water soluble
- Effectiveness at nanomole level
- Enhanced therapeutic potential

PATENT STATUS

Country	Type	Number	Dated	Case
United States Of America	Issued Patent	11,007,248	05/18/2021	2014-228
United States Of America	Issued Patent	10,314,889	06/11/2019	2014-228

ADDITIONAL TECHNOLOGIES BY THESE INVENTORS

- Disease Markers: Mucin 5B Monoclonal Antibodies
- Controlling Tumor Growth And Malignancy
- Peptide Inhibitors of Idiopathic Pulmonary Fibrosis
- Mucin-Specific Monoclonal Antibodies

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OTHER INFORMATION

KEYWORDS

asthma, peptide, proteomics, inhibitor, pkc, akt/slug, PI3k/AKT, allergic lung inflammation

CATEGORIZED AS

- **Biotechnology**
  - Health
  - Proteomics
- **Medical**
  - Disease: Autoimmune and Inflammation
  - Disease: Cancer
  - Disease: Cardiovascular and Circulatory System
  - Therapeutics

RELATED CASES

2014-228-0

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