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Novel Murine Model of Asthma Identifies Methods to Antagonize Th2 Response, Asthma and Allergic Disease

Tech ID: 23253 / UC Case 2011-208-0

BACKGROUND

Bronchial asthma is a chronic and heterogeneous inflammatory disorder of the conducting airways with immune and non-immune etiologies. Although the underlying molecular basis of asthma is not completely understood, inflammation is a key pathological feature of bronchial asthma. The increasing prevalence in Western countries (approximately 15% of children and 8% of adults) supports the vast resources deployed to find treatments and drugs that act upon pathways not targeted in current therapies are of particular interest.

TECHNOLOGY DESCRIPTION

UC inventors have developed a murine model of spontaneous asthma that closely replicates the immunologic, genetic and the clinical course of the related human disease. And, by uncovering an unknown mechanism of action, inventors have identified new means of specifically inhibiting the Th2 biased response in general, and allergic asthma in particular. This model has also enabled *in vivo* testing of new class of drugs that is able to block the sequence of events that culminate in human asthma.

APPLICATIONS

The invention claims new therapies for bronchial asthma and other allergic diseases that may respond to regulating specific T cell responses.

ADVANTAGES

By modulating an alternative pathway (unlike current therapies), one may interfere with the immune response that tees up individuals for expressed symptoms.

STATE OF DEVELOPMENT

Inventors have generated a murine model of human asthma that spontaneously reproduces the human disease. Specific, "hallmark" indicators have been assessed (serum IgE, airway hyper-reactivity, airway inflammation) to confirm the efficacy of the class of drugs claimed for their ability to abrogate the course of disease, *in vivo*.

INTELLECTUAL PROPERTY INFO

Worldwide rights available; Pending patents available under confidentiality.

RELATED MATERIALS

Li, X., et al. (2012) Divergent requirement for Gas and cAMP in the differentiation and inflammatory profile of distinct mouse Th subsets, J Clin Invest. Mar 1;122(3):963-73. - 03/01/2012

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Pulendran B, et al. (2010) Programming dendritic cells to induce T(H)2 and tolerogenic responses. Nat Immunol 11: 647-55 - 07/20/2010

Kim HY, et al. (2010) The many paths to asthma: phenotype shaped by innate and adaptive immunity. Nat Immunol 11: 577-84 -06/18/2010

Datta SK, et al. (2010) Mucosal adjuvant activity of cholera toxin requires Th17 cells and protects against inhalation anthrax. Proc Natl Acad Sci U S A 107: 10638-43 - 04/28/2010

Barrett NA and KF Austen. 2009. Innate cells and T helper 2 cell immunity in airway inflammation. Immunity 31: 425-37 - 09/18/2009

CONTACT

University of California, San Diego Office of Innovation and Commercialization innovation@ucsd.edu tel: 858.534.5815.



INVENTORS

Raz, Eyal

OTHER INFORMATION

KEYWORDS

asthma, bronchial asthma, inflammation, inflammatory, psoriasis, allergy, multiple sclerosis, immunomodulation, immunomodulation,

immune modulation, Th-2, helper, T-

cell, T cell

CATEGORIZED AS

Biotechnology

Health

Medical

Disease: Autoimmune and Inflammation

Disease: Respiratory and

Pulmonary System

RELATED CASES 2011-208-0, 2013-334-0, 2014-084-0, 2013-282-0 ▶ Lee J, et al., Cyclic AMP concentrations in dendritic cells induce and regulate Th2 immunity and allergic asthma, Proc Natl Acad Sci U S

A. 2015, 3;112(5):1529-34.

OTHER INFORMATION

E. Raz (2013) A Novel Approach to Explore Th2-Biased Immunity: Implications for Asthma and Allergic Diseases, Manuscript in

preparation (available under confidentiality)

PATENT STATUS

Country	Туре	Number	Dated	Case
United States Of America	Published Application	2014059147	04/17/2014	2011-208

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