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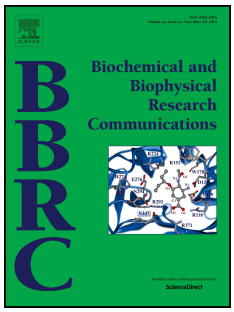

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Inhibition of TRPC3 Downregulates Airway Hyperresponsiveness, Remodeling of OVA-sensitized mouse

Lingwei Wang[#1], Jie Li[#1,2], Jian Zhang[#3], Qi He[1], Xuanwen Weng[1], Minjie Guan[1], Chen Qiu[*1]

[1]Department of Respiratory Diseases, Second Clinical medical college (Shenzhen People's Hospital), Jinan University, Shenzhen, China

[2]Integrated Chinese and Western Medicine Postdoctoral research station, Jinan University, Guangzhou, China

[3]Research Laboratory for Reproductive Health, Shenzhen Institutes of Advanced Technology, Chinese Academy of Sciences, Shenzhen, China

[#] These authors contributed equally to this work.

∗Corresponding authors Chen Qiu: Department of Respiratory Diseases, Second Clinical medical college (Shenzhen People's Hospital), Jinan University, 1017 Dongmen North Road, Luohu District, Shenzhen, Guangdong 518020, China. Tel.: +86 755 2553 3018; fax: +86 755 2553 3497. E-mail address: qiuchen@email.jnu.edu.cn.(C. Qiu).

Abstract

Airway hyperresponsiveness (AHR), airway remodeling and inflammation are the fundamental pathological alterations that occur in asthma. Transient receptor potential canonical 3 (TRPC3) has been implicated in diverse functions of airway smooth muscle cells (ASMCs) in asthma. However, the underlying mechanisms remain incompletely understood. We investigated the mRNA and protein expression of TRPC3 in ASMCs from normal and OVA-sensitized mouse. And the effects of inhibition or knockdown of TRPC3 with Ethyl-1- (4- (2,3,3-trichloroacrylamide) phenyl) -5 - (trifluoromethyl) -1H -pyrazole -4-carboxylate (Pyr3) and lentiviral shRNA on OVA-sensitized mouse AHR, airway remodeling, circulating inflammatory cytokines, cell proliferation and migration. We found that TRPC3 mRNA and protein expression levels were significantly increased in ASMCs from OVA-sensitized mouse. Inhibiting TRPC3 with continuous subcutaneous administration of Pyr3 decreased