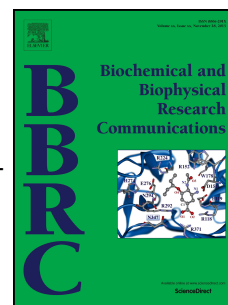


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## **Inhibition of TRPC3 Downregulates Airway Hyperresponsiveness, Remodeling of OVA-sensitized mouse**

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### **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

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