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Serum IgE induced airway smooth muscle cell remodeling is independent of allergens and is prevented by omalizumab

Micheal Roth

University Hospital Basel, Switzerland

Background: Airway wall remodeling is a major pathology of allergic asthma and is independent of inflammation. This pathology can be induced by purified human IgE, without the presence of allergens by activating airway smooth muscle cells (ASMC) to proliferation and depose extracellular matrix.

Objective: We assessed if circulating IgE obtained from allergic asthma patients in the absence of allergens stimulates ASMC remodeling and if this can be prevented by anti-IgE antibodies.

Methods: *In vitro*, human ASMC were exposed to serum of either healthy controls, or patients with allergic asthma, non-allergic asthma, or atopic non-asthma patients. Proliferation and deposition of collagens and fibronectin were determined after 3 and 5 days.

Results: Serum from patients with allergies significantly stimulated ASMC proliferation at 3 and 5 days, the deposition of collagen type-I within 48 hours and of fibronectin within 24 hours. One hour pre-incubation of sera with omalizumab prevented these effects in allergic serum. In contrast, the anti-IgE antibody had no significant effect on serum from healthy donors or patients with non-allergic asthma. Furthermore, these effects were not modified by the presence of allergens.

Conclusion: The data provides experimental evidence that anti-IgE antibodies neutralize the pro-remodeling activity suggesting that IgE is a major contributor ASMC mediated airway wall remodeling in asthma.

Biography

Michael Roth is currently working as the Head of Pulmonary Cell Research, Pneumology, University Hospital Basel, Switzerland. He has completed his PhD from University of Basel. He has worked as a Visiting Professor and Associate Professor for University of Sydney for 2 years. He has published 148 articles in reputed journals.

Michael.Roth@usb.ch

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