Journal of Immunology Research 2017 vol.2017

Cytokine Profile of Patients with Allergic Rhinitis Caused by Pollen, Mite, and Microbial Allergen Sensitization

Tyurin Y., Lissovskaya S., Fassahov R., Mustafin I., Shamsutdinov A., Shilova M., Rizvanov A.
Kazan Federal University, 420008, Kremlevskaya 18, Kazan, Russia

Abstract

© 2017 Yury A. Tyurin et al. Allergic rhinitis (AR) is especially prevalent among the population of large cities. Immunologically, the airway epithelium is a region where the population of allergen-presenting cells concentrates. These cells actively express a group of receptors of the innate immune system. A specific cytokine profile is its representation. The study was aimed at evaluating the cytokine profile in patients with seasonal and perennial allergic rhinitis. The cytokine profile of nasal secretion and blood serum of 44 patients with AR was studied. 24 of them had seasonal allergic rhinitis (SAR), and 20 patients suffered from perennial allergic rhinitis (PAR). The patients' age ranged from 4 to 60 years. It was determined in our study that the activation of the GM-CSF production retained in patients with PAR sensitized to mite allergen components (Dermatophagoides pteronyssinus). There was a higher production profile of TNF-α and TSLP in nasal secretion in the patients with perennial allergic rhinitis and additional high sensitization to SEs. Sensitization to mold fungal allergen components significantly increases in patients with seasonal allergic rhinitis. They demonstrated high level of sensitization to the Aspergillus fumigatus component m3. Thus, along with other clinical trials, the study performed would clarify some aspects of molecular pathogenesis of human allergic rhinitis.

http://dx.doi.org/10.1155/2017/3054217

References

[1] K. Thestrup-Pedersen, "Atopic eczema. What has caused the epidemic in industrialised countries and can early intervention modify the natural history of atopic eczema?," Journal of Cosmetic Dermatology, vol. 2, no. 3-4, pp. 202-210, 2003.

[2] J. Bousquet, N. Khaltaev, A. A. Cruz et al. , "Allergic rhinitis and its impact on asthma (ARIA) 2008 update (in collaboration with the World Health Organization, GA2LEN and Aller-Gen)," Allergy: European Journal of Allergy and Clinical Immunology, vol. 63, Supplement 86, pp. 8-160, 2008.

[3] D. J. Nelson, C. McMenamin, A. S. McWilliam, M. Brenan, and P. G. Holt, "Development of the airway intraepithelial dendritic cell network in the rat from class II major histocompatibility (Ia)-negative precursors: differential regulation of Ia expression at different levels of the respiratory tract," The Journal of Experimental Medicine, vol. 179, pp. 203-212, 1994.

[4] S. McWilliam, D. Nelson, J. A. Thomas, and P. G. Holt, "Rapid dendritic cell recruitment is a hallmark of the acute inflammatory response at mucosal surfaces," The Journal of Experimental Medicine, vol. 179, pp. 1331-1336, 1994.

[5] A. Trompette, S. Divanovic, A. Visintin et al. , "Allergenicity resulting from functional mimicry of a Toll-like receptor complex protein," Nature, vol. 457, pp. 585-588, 2009.

[6] S. Akira, S. Uematsu, and O. Takeuchi, "Pathogen recognition and innate immunity," Cell, vol. 124, pp. 783-801, 2006.

[7] M. A> Willart and B. N. Lambrecht, "The danger within: endogenous danger signals, atopy and asthma," Clinical & Experimental Allergy, vol. 39, pp. 12-19, 2009.

[8] J. L. Bro?ek, J. Bousquet, C. E. Baena-Cagnani et al. , "Allergic rhinitis and its impact on asthma (ARIA) guidelines: 2010 revision," The Journal of Allergy and Clinical Immunology, vol. 126, pp. 466-476, 2010.

[9] E. Sallmann, B. Reininger, S. Brandt et al. , "High-affinity IgE receptors on dendritic cells exacerbate TH2-dependent inflammation," The Journal of Immunology, vol. 187, pp. 164-171, 2011.

[10] H. Hammad, M. Plantinga, K. Deswarte et al. , "Inflammatory dendritic cells?not basophils?are necessary and sufficient for induction of Th2 immunity to inhaled house dust mite allergen," The Journal of Experimental Medicine, vol. 207, pp. 2097-2111, 2010.

[11] R. P. Schleimer, A. Kato, R. Kern, D. Kuperman, and P. C. Avila, "Epithelium: at the interface of innate and adaptive immune responses," The Journal of Allergy and Clinical Immunology, vol. 120, pp. 1279-1284, 2007.

[12] K. Bulek, S. Swaidani, M. Aronica, and X. Li, "Epithelium: the interplay between innate and Th2 immunity," Immunology & Cell Biology, vol. 88, pp. 257-268, 2010.

[13] P. D. Mitchell and P. M. O'Byrne, "Epithelial-derived cytokines in asthma," Chest, vol. 151, 2017.

[14] D. Lam, N. Ng, S. Lee, G. Batzer, and A. A. Horner, "Airway house dust extract exposures modify allergen-induced airway hypersensitivity responses by TLR4-dependent and independent pathways," The Journal of Immunology, vol. 181, pp. 2925-2932, 2008.

[15] F. Perros, H. C. Hoogsteden, A. J. Coyle, B> N. Lambrecht, and H. Hammad, "Blockade of CCR4 in a humanized model of asthma reveals a critical role for DC-derived CCL17 and CCL22 in attracting Th2 cells and inducing airway inflammation," Allergy, vol. 64, pp. 995-1002, 2009.

[16] H. Bowen, A. Kelly, T. Lee, and P. Lavender, "Control of cytokine gene transcription in Th1 and Th2 cells," Clinical & Experimental Allergy, vol. 38, pp. 1422-1431, 2008.

[17] M. L. Kapsenberg, "Dendritic-cell control of pathogen-driven T-cell polarization," Nature Reviews Immunology, vol. 3, pp. 984-993, 2003.