*Anikó Kapitány*[1], *Gabriella Béke*[1], *Georgina Nagy*[1], *Qm.Doan-Xuan*[2], *Zsolt Bacsó*[2], *Krisztián Gáspár*[1], *Gábor Boros*[3], *Gábor Mócsai*[1], *Zsolt Dajnoki*[1], *Tamás Bíró*[4], *Éva Rajnavölgyi*[5], *Andrea Szegedi*[1]:

**Examination of peripheral blood dendritic cells in atopic dermatitis**

(Division of Dermatological Allergology, Department of Dermatology, Faculty of Medicine, University of Debrecen, Debrecen, Hungary[1], Department of Biophysics and Cell Biology, Faculty of Medicine, University of Debrecen, Debrecen, Hungary[2], Department of Dermatology, Faculty of Medicine, University of Debrecen, Debrecen, Hungary[3], MTA-DE "Lendület" Cellular Physiology Research Group, Faculty of Medicine, University of Debrecen,, Debrecen, Hungary[4], Department of Immunology, Faculty of Medicine, University of Debrecen, Debrecen, Hungary[5])

Dendritic cells (DCs) as the main antigen presenting cell type play crucial role in the pathogenesis of atopic dermatitis (AD). Characteristics of the DCs located in the skin of AD patients are well defined but less is known about their peripheral blood DC precursors. Therefore we aimed to characterise the myeloid pre-DCs separated from the blood of patients with AD, to examine their phenotypical features and chemokine production in order to determine if they differ from myeloid pre-DCs in the blood of healthy controls.

Peripheral blood myeloid pre-DCs were separated from the blood of 12 AD patients and 10 healthy controls using CD1c+/BDCA1+ magnetic separation kit. Cell surface markers were examined with Flow Cytometry while chemokine production was investigated with the help of Chemokine Antibody Array.

Focusing on CD1c+ and CD11c + cells we found, that the cells separated from the blood of patients with AD expressed more FcεRI (56,23% vs. 27,48%) and less CD206-mannose receptors (45,8% vs. 66,2%) on their surface than the cells, that were originated from healthy controls. Moreover pre-DCs from AD patients exclusively produced not just the AD related CCL17 and CCL18, but also those chemokines, which are characteristic for maturing DCs (CCL3, CCL4, CCL5, CCL19).

Our results indicate that circulating pre-DCs deriving from AD patients are more matured and activated than the cells that derive from healthy persons and therefore may contribute to the pathogenesis of AD.