Lazaros I Sakkas, J Clin Exp Dermatol Res 2018, Volume 9
DOI: 10.4172/2155-9554-C1-067

17TH EUROPEAN DERMATOLOGY CONGRESS

March 01-03, 2018 | Paris, France

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Apremilast induces IL-10+ Bregs that are inversely correlated with IFN-γ+ and IL-17+ NKT cells in psoriatic arthritis and psoriasis

Background & Purpose: There is impairment IL-10+ regulatory B cells (IL-10+Bregs) in psoriatic arthritis (PsA) and psoriasis (Ps) and Bregs were inversely correlated with IL-17+ and IFNγ+ innate immune cells. Apremilast, a PDE4 inhibitor, increased IL-10+Bregs in PsA and Ps. Herein we explored the relationship of IL-10+Breg cells with NK and NKT cells producing the IFN-γ and IL-17 post-apremilast, a PDE4 inhibitor, treatment.

Methods: PBMCs from 8 PsA, 12 Ps patients, obtained at baseline and 6 months post-apremilast treatment, and 10 healthy controls (HCs) were studied. Flow cytometric analysis was carried out with MoAbs against CD56, CD16, CD3, CD7, CD19, CD24, CD27 and CD38. Intracellular IFN-γ, IL-17 and IL-10 following CpG (ODN2006) and PMA/ionomycin stimulation was also examined.

Results: The PsA baseline CD3+CD56+ (NKT) cells were increased compared to Ps patients and HCs (10.2±3.6 vs. 5.2±2.1 vs. 4.3±2.5 , $p<0.05$ for PsA vs. Ps and HC), but no difference in NK cells between PsA, Ps and HCs. Apremilast treatment did not significantly alter the total NKT and NK but decreased IFNγ+ and IL-17+ NKT cells from PsA and Ps patients collectively (from 52.2±12.8 pre to 35.3±7.1 for IFNγ+ NKT and from 2.2±1.06 to 0.96±0.35 for IL-17+ NKT; $p<0.05$ for both subsets). IFNγ+ and IL-17+ NKT cells from PsA and Ps patients inversely correlated with IL-10+ Bregs post-treatment. There was no correlation between memory Bregs and cytokine-producing NKT and NK cells.

Conclusion: Apremilast induces IL-10+ Bregs and decreases IFN-γ- and IL-17-producing NKT cells in PsA and Ps.

Biography

Lazaros I Sakkas is currently working as a Professor of Medicine and Rheumatology, Chairman of the Department of Rheumatology and Clinical Immunology, Faculty of Medicine, School of Health Sciences, University of Thessaly. He is also an Adjunct Assistant Professor at Temple University School of Medicine, Philadelphia, PA, USA and an Adjunct Professor, Center for Molecular Medicine, Old Dominion University, USA. He is the President at the Institute for Rheumatic Diseases.

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