A96 REGULATION OF AUTOIMMUNE INFLAMMATION BY MYELOID-DERIVED SUPPRESSOR CELLS

E Kontaki,[1,2] M Ioannou,[1,2] T Alissafi,[1,2] K A Papadakis,[1] D Boumpas,[1,2] P Verginis[1,2] *[1]Medical School, University of Crete, Heraklion, Greece; [2]Institute of Molecular Biology and Biotechnology, Foundation for Research and Technology, Heraklion, Greece*

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Background and objectives Restoration of immune homeostasis and self-tolerance represent the ultimate goal of autoimmune diseases. Although a variety of therapeutic targets are employed, a large number of patients with autoimmune syndromes fail to either respond to current therapy or to achieve long-lasting remission after its cessation. Myeloid-derived suppressor cells (MDSCs) encompass a newly described population of cells that potently suppress immune responses however their role in autoimmune inflammatory diseases is poorly understood.

Materials and methods Using flow cytometry, the authors assessed the presence of CD11b Gr1+ MDSCs in the spleen and draining lymph nodes of mice with trinitrobenzene sulfonate (TNBS)-induced colitis and control-treated mice. Furthermore, the authors monitored the expansion of the monocytic (CD11b Ly6C+ Ly6G_) and granulocytic (CD11b Ly6G+ Ly6C_) subsets of MDSCs in the peripheral lymphoid compartments.

Results The authors observed an increased accumulation of CD11b Gr1+ MDSCs in the spleen of TNBS-treated mice, compared to control group. Among the MDSCs, the granulocytic Ly6G+ subset was significantly enriched in TNBS-treated mice both in spleen and lymph nodes. Extensive phenotypic characterisation of sorted Ly6G+ MDSCs revealed an increased expression of the inhibitory molecule PD-L1, but not PD-L2, indicating that MDSCs might mediate their function via the PD-1/PD-L1 pathway.

Conclusions This data demonstrate a preferential expansion of granulocytic Ly6G+ MDSCs during the effector phase of the inflammatory response. Ongoing work would address the potential role of this cell subset in inhibiting established disease and elucidate the mechanisms used by MDSCs for the suppression of autoimmune inflammatory diseases.