analyzed using flow cytometry. The PBMCs were incubated with anti-CD3/CD28 beads, supplemented with transforming growth factor-β and interleukin-2 to induce differentiation of Tregs, with or without tunicamycin for 36 hours.

**Results** The percentage of Tregs in the PBMCs of SLE patients was lower than that in the HCs (1.8 ± 0.9 versus 2.6 ± 0.7%, \( p=0.02 \)). The induced differentiation of Tregs increased in both groups, and the increased proportion was greater in the SLE group (600 ± 351 versus 252 ± 95%, \( p=0.01 \)). Incubation with tunicamycin in the Treg differentiation process also increased the proportion of Tregs in both groups (385 ± 259 versus 166 ± 105%, \( p=0.006 \)), and the increased proportion was higher in the SLE group.

**Conclusions** The baseline percentage of Tregs was lower in SLE patients than in HCs. However, when Treg differentiation was induced, the differentiation of Tregs was more pronounced in the SLE group. This exaggerated differentiation may reflect the paradoxical response to the diminished suppressive capacity of Tregs in SLE patients.

**CD11c+T-BET+ B CELL IS CRITICAL FOR ANTI-CHROMATIN TGG2A PRODUCTION IN THE DEVELOPMENT OF LUPUS**

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**Background and aims** A hallmark of systemic lupus erythematosus is high titers of circulating autoantibodies. Recently a novel CD11c+ B cell subset has been identified in aged female mice that is critical for the development of autoimmunity. Transfer of MHC II-mismatched splenocytes from Bm12 mice into B6 mice causes a chronic graft versus host reaction (cGVHD), which is characterised by the production of high titers of autoantibodies and immunopathology that closely resemble SLE. The aim of this study was to figure out the role of CD11c+ B cell in the production of autoantibodies during the development of lupus induced by cGVHD.

**Methods** We developed and validated cGVHD model by spleenocytes transfer of Bm12 mice into B6 mice and identified CD11c+ B cell by flow cytometry and examined anti-chromatin antibody by ELISA. We also identified CD11c+T-bet+ B cell of peripheral blood mononuclear cells obtained from SLE patients and healthy controls.

**Results** CD11c+T-bet+ B cell was significantly increased in the development of lupus induced by cGVHD. CD138+CD11c+ B cell produced large amounts of anti-chromatin IgG2a upon in vitro stimulation. Depletion of CD11c+ B cells significantly ameliorated anti-chromatin IgG2a production in vivo. T-bet deficiency impaired the expression of CD11c in B cells and anti-chromatin autoantibodies production in the process of cGVHD. The accumulation of T-bet+CD11c+ B cell was found in lupus patients.

**Conclusions** Our data demonstrated the aberrant activation and differentiation of CD11c+T-bet+ B cell, which produced large amounts of anti-chromatin IgG2a in lupus murine model and patients.