Background The 1997 Update of the 1982 American College of Rheumatology Revised Criteria for Classification of SLE includes two autoantibody criteria: #10, abnormal level of anti-DNA, anti-Sm, or antiphospholipid; #11 positive antinuclear antibody (ANA). Thus, ANA positivity is counted as 1 of the 11 criteria and a person shall be said to have SLE if any 4 or more of the 11 criteria are present, serially or simultaneously, during any interval of observation. The immunofluorescence pattern observed in the ANA test provides a direct initial assessment of the immunological status of the patient’s immune system. The ANA pattern, by definition, indicates the presence of autoantibodies. The accuracy of the ANA test, expressed as the frequency of false positive and false negative results, is influenced by many factors, including the type of cell used, the antibody detecting system and the observer. The immunofluorescence pattern observed in the ANA test provides a direct initial assessment of the immunological status of the patient’s immune system. The ANA pattern, by definition, indicates the presence of autoantibodies. The accuracy of the ANA test, expressed as the frequency of false positive and false negative results, is influenced by many factors, including the type of cell used, the antibody detecting system and the observer. The immunofluorescence pattern observed in the ANA test provides a direct initial assessment of the immunological status of the patient’s immune system. The ANA pattern, by definition, indicates the presence of autoantibodies. The accuracy of the ANA test, expressed as the frequency of false positive and false negative results, is influenced by many factors, including the type of cell used, the antibody detecting system and the observer.

Methods Collective issues on ANA nomenclature were raised at the annual ICAP workshops. The 2016 meeting was attended by 120 participants from 34 countries. The workshop exchanges arrived at consensus on a few, but clearly important issues that are of great interest to the ANA community. These issues include: (1) the need to adopt a nomenclature and classification tree categorised in three major types as demonstrated by: a) decreased production of anti-dsDNA autoAb, b) decreased number and proliferation of GC B cells, c) decreased number of IgG anti-dsDNA secreting PC and d) decreased IRF4 and Prdm1 mRNA expression. RGC-32 KO recipients. Host B cell number and activation, anti-dsDNA Ab production, germinal centre (GC) B cell number and proliferation, PC number, expression of transcription factors IRF4 and Blimp1 were assessed at 2 and 4 weeks.

Results RGC-32 mRNA was upregulated in B cells by lps, anti-CD40 mAb, IL-21 and IL-6, IL-4 or TGFβ and RGC-32 mRNA and protein expression was determined. TLR-dependent and T independent B cell differentiation to plasma cells (PC) was induced with lps and with CD40mAb plus IL-4. cGVHD was induced with 100×10^6 Bm12 splenocytes injected into WT or RGC-32 KO recipients. Host B cell number and activation, anti-dsDNA Ab production, germinal centre (GC) B cell number and proliferation, PC number, expression of transcription factors IRF4 and Blimp1 were assessed at 2 and 4 weeks.

Conclusions These results suggest that expression of RGC-32 in B cells is critical for optimal GC proliferation, PC differentiation and autoantibody production in a murine model of lupus. These data support the idea that RGC-32 blockade has the potential to attenuate autoimmune parameters of cGVHD and possibly reverse abnormalities in the T and B cell pathways that contribute to lupus pathogenesis.

Dalazatide, an inhibitor of the Kv1.3 channel on activated effector memory T cells, has immunotherapy potential against systemic lupus erythematosus

1,2Anne Stevens*, 3Megan Yuasa, 5Chelsea Olsen, 6Shawn Iadonato, 7Peter Probst, 7Seattle Children’s Research Institute, Seattle, WA, USA; 8University of Washington, Seattle, WA, USA; 9Kineta, Inc., Seattle, WA, USA; 10KPI Therapeutics, Inc, Seattle, WA, USA

Abstracts