splenocytes (effector) were incubated with K-562 (target) in a ratio of 100:1 for 4 hour once a week until the end of the experiment.

**Conclusions** The splenocytes from KRG treated porcine showed a significantly increased cytotoxicity in time dependent fashion. Whereas, splenocytes from untreated porcine showed a less toxicity. Taken together, KRG has the potential to modulate immune function and should be further investigated as an immunomodulatory agent.

**Methods** Gastric cancer cells (SNU-484) were grown in RPMI medium with 10% heat inactivated FBS at a seeding density 1*10^6 cells/mL for 3 days, supernatant was concentrated 10 fold. Splenocytes were treated with 1% (v/v) SNU-484 supernatant for various periods of time.

**Results** Flow cytometry (FCM) results suggests that the treatment do not affect the viability of the cells during the study period, further the intracellular levels of NKP30, NKP44, granzyme B, perforin were assessed using Real time-PCR (RT-PCR) and western blot techniques. RT-PCR revealed that NK cell markers were initially down-regulated during 2 days of incubation and increased several folds higher during 5th day when compared to normal control. However, no significant changes were observed in protein expression. SNU-484 cells supernatant treated splenocytes were further analysed for cytolytic activity against K562 cell line as a target with varying (1:6, 1:12, 1:25 and 1:50) target to effector ratio for a period of 24 hour.

**Conclusions** The results suggest that the treated splenocytes have significantly increased cytolytic activity (49.4%) at the lower effector to target ratio (1:25) when compared to untreated control splenocytes (38.2%). Our results indicate that gastric cancer cell micro-environment can modulate the NK cells efficacy to act against cancer.
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 TGIN G2A 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 splenocytes 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+CD11+ 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.

### IXAZOMIB, AN ORAL PROTEASOME INHIBITOR, REDUCES ANTIBODY PRODUCTION BY DEPLETING PLASMA CELLS IN A T CELL DEPENDENT ANTIGEN RESPONSE MODEL


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### Background and aims
Pathogenic auto-antibodies produced by plasma cells are key drivers of many auto-immune diseases such as Systemic Lupus Erythematosus (SLE), Rheumatoid Arthritis (RA), and Sjogren’s Syndrome (SS). In addition, solid organ transplant rejection is also mediated by antibodies produced against the donor organ. Plasma cells are highly metabolically active antibody factories and thus sensitive to depletion by proteasome inhibitors. Ixazomib, an oral