increased pyroptosis related upstream regulating genes including IRF1, GBP1, CASP4 and CASP1. Furthermore, this subset highly expressed inflammasome gene such as NLRC5 not NLRP3.

Conclusions There are ISG high expressed CD4 T cells across tissues of lupus. Those cells highly expressed pyroptosis pathway related genes. Further investigation is needed to characterize association between ISG and pyroptosis.

LP-093 ROLE OF LEUKOCYTE IMMUNOGLOBULIN-LIKE RECEPTOR A3 (LILRA3) IN THE PATHOGENESIS OF LUPUS-LIKE DISEASE

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Background Leukocyte immunoglobulin-like receptor A3 (LILRA3) is the soluble protein of LILRs family. LILRA3 exhibits a 6.7-kb deletion variation among individuals. The deletion removes all of four Ig-like domains, resulting in a 'non-functional LILRA3'. Our research group identified the functional LILRA3 as a novel genetic risk for systemic lupus erythematosus. The aim of this study was to functionally characterize the impact of LILRA3 in the pathogenesis of SLE.

Methods We constructed a humanized LILRA3 knock-in (LILRA3-KI) mouse in C57BL/6 (B6) mice background. The lupus-like disease was induced by repeated epicutaneous stimulation with Toll-like receptor (TLR)-7 agonist imiquimod (IMQ) on both ears of mice. All of the mice were euthanized four weeks after the initiation of treatment. The spleen and serum samples were collected. Immunocyte populations were determined by staining with fluorescently-conjugated antibodies and analysed by flow cytometry. Serum antibodies were detected by enzyme-linked immunosorbent assay (ELISA).

Results The mice developed lupus-like disease following 4 weeks of treatment with imiquimod. LILRA3-KI mice exposed to imiquimod showed increased innate and adaptive immune response, including splenomegaly ($p < 0.05$) and elevated levels of serum anti-dsDNA IgG ($p < 0.05$), compared to WT mice. The frequencies of M1 macrophage, M2 macrophage, T follicular helper cells (Tfh), germinal center (GC) B, and plasma B cells were increased in KI mice ($p < 0.05$).

Conclusions Our data demonstrated that LILRA3 promoted the TLR7-driven lupus autoimmunity with the excessive expansion of macrophages, Tfh, GC B, and plasma B cells.

LP-094 REPRESSION OF ID3 AGGRAVATES LUPUS PHENOTYPES IN MURINE MODELS VIA ABERRANT B CELL DIFFERENTIATION

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Background Id3 is a member of the inhibitor of DNA binding (Id) family which is a helix-loop-helix protein acting as a transcriptional regulator. Previous studies have reported that Id3 plays an essential role in the development and function of regulatory T cells in lupus. However, its role in B cells remains unclarified.

Methods To investigate the role of Id3 in B cells, we generated C57BL/6 mice with a CD19Cre-mediated B cell-specific depletion of Id3 as well as mice (Id3-/-) with a conventional knockout of Id3. In these mice, we evaluated the presentation of lupus-mimicking phenotypes and changes in immune cell populations. Additionally, we assessed the influences of B cell-specific Id3 depletion in lupus-induced mice by R848.

Results In Id3-/- mice, proportions of effector T cells such as Th1, Th2, and Th17 cells were elevated whereas those of regulatory T cells were lower than in control mice. Furthermore, proportions of plasma cells were significantly elevated in Id3-/- mice. These mice presented with increased inflammation in kidney tissues, resembling lupus nephritis. An elevated proportion of plasma cells was replicated in mice with B cell-specific Id3 depletion. Induction using R848 exacerbated lupus-like manifestations including increased proteinuria and higher serum immunoglobulin levels in B cell-specific Id3-depleted mice than in control mice. In an in vitro study, CD19+ B cells from Id3-/- mice were more differentiated into plasma cells than those from control mice. In contrast, there were no significant differences in other B cell subsets.

Conclusions Genetic suppression of Id3 in murine models exacerbated lupus-like phenotypes with aberrant B cell differentiation. These findings may imply a potential role of Id3 in the pathogenesis of lupus.

LP-095 MACROPHAGE ACTIVATION IN UNTREATED PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS (PRELIMINARY DATA)

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Background The study of the ability of monocytes to activate associated with the clinical activity of immunological markers of inflammation in systemic lupus erythematosus (SLE) will provide important and fundamentally new information on the involvement of these cells in the development of autoimmune rheumatic diseases.

Objectives To study macrophage activation in untreated systemic lupus erythematosus (SLE) patients (pts).

Methods A total of 21 active SLE pts (female/male 15/6) were enrolled in the study (median age was 35[24; 41] years; disease duration was 8 [2; 14] months; SLADAI-2K was 7 [6;16]). The control group consisted of 29 volunteers (23F/6M, median age 38 [35; 48] years).

Isolation of monocytes was carried out according to the standard procedure for obtaining a leukocyte fraction in a Ficoll gradient and subsequent selection of CD14 + cells using magnetic separation. After isolation, the cells were cultured in X-Vivo medium. To assess the degree of monocyte activation, cells were stimulated by the addition of LPS. The value of monocyte activation was expressed as a ratio of the level of secretion of proinflammatory cytokines by monocytes cultured with and without LPS addition. Secretion levels were determined by ELISA. The belonging of the isolated cells to

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