vivo study. Toll-like receptor (TLR)-stimulated human PBMCs or murine bone marrow-derived dendritic cells were used for in vitro study.

**Results** Our study demonstrated that DZ2002 exerted a therapeutic effect on NZB/W F1 mice with established nephritis. The mechanism involves the modulation of T cell development in lupus by interfering with TLR-triggered APC function. Further study explored the regulatory mechanisms of SAHH on DC function in both innate and adaptive immune system, using the SAHH inhibitor with definite target and potent immunosuppressive activity.

**Conclusions** The present issue demonstrated that the reversible SAHH inhibitor DZ2002 effectively ameliorates lupus syndrome in NZB/W F1 mice by regulating TLR signaling-mediated APC responses. This compound is prospective to become a novel drug for SLE treatment with Hcy as a potential biomarker in autoimmune disease.
Results  Then, anasarca and nephrosis was improved and S-Cr was decreased to 1.20 mg/dL in proportion to the reduction in activated Th17 cells in PD.

Conclusions  Although recent studies have begun to shed light on the role of IL-17 in the pathogenesis of SLE, there is no convincing evidence in actual patients. In this case, improvement of disease activity of SLE was correlated with the decrease of activated Th17. This is the first report that the IL-17-targeted therapy for SLE was shown to be effective in a patient skewing towards Th17-phenotype.

DEVELOPMENT OF ARTEMISININ ANALOGUE ANALOG SM934 IN THE TREATMENT OF SYSTEMIC LUPUS ERYTHEMATOSUS

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Background and aims  Besides their outstanding antimalarial activity, artemisinin and its derivatives also possess immunosuppressive activities and are clinical used to treat SLE. β-aminooarteether maleate (SM934), a water soluble artemisinin derivative, got the approval documents authorised by Chinese FDA for clinical trials. This study investigated the curative effects of SM934 on lupus-prone mice and explored its underlying therapeutic mechanisms.

Methods  In vivo, SM934 was given orally to female NZB/W F1 and MRL/lpr mice; renal injury, peripheral lymphoid organ disease and serological changes were evaluated. Meanwhile, correlative pathological mechanisms were studied using different aged mice. Besides, the effects of SM934 on human PBMCs were also assessed.

Results  We demonstrated that SM934 treatment could significantly improve SLE syndrome in lupus-prone animal models, including delayed the progression of glomerulonephritis; ameliorated proteinuria and renal lesion severity; increased the survival rate; decreased levels of BUN and serum anti–double-stranded DNA antibodies. Furthermore, clinical improvement was accompanied with decreased Th1-related anti-dsDNA IgG2a and IgG3 Abs, serum IL-17, and increased Th2-related anti-dsDNA IgG1 Ab, serum IL-10 and IL-4. Moreover, SM934 could significantly inhibit both of Th1 and Th17 responses, elevate Treg percentage and lower the percentage of CD3+ B220+ CD4+ CD8− (double negative) T cells in MRL/lpr mice. We further elucidate that SM934 treatment restored the compartment of B cells in the spleen of MRL/lpr mice by increasing quiescent B cells, maintaining germinal centre B cells, decreasing activated B cells and reducing PCs.

Conclusions  This work provides new evidence and clues for research about artemisinin compounds in the field of autoimmune diseases.

INCREASED NEUTROPHILS AND NEUTROPHIL SERINE PROTEASES IN THE SPLEENS OF ESTROGEN-TREATED C57BL/6 MICE AND IN SEVERAL STRAINS OF SPONTANEOUS LUPUS-PRONE MICE

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Background and aims  Oestrogen, a natural immunomodulator, regulates the development and function of diverse immune cell types and has been implicated in lupus development.

Methods  To determine the regulatory role of oestrogen on neutrophil development and function, we treated B6 mice with placebo- or oestrogen implants for 6–8 weeks, and then analysed splenic neutrophil serine proteases (NSPs, such as

Abstract 112 Figure 1  Estrogen treatment increases neutrophil number, NSP and MPO expression in wild type B6 mice. (A) Flow cytometry analysis of splenic neutrophil percentage in placebo- and estrogen-treated B6 mice. (B) The total splenocytes count in placebo- and estrogen-treated mice. (C) The total CD11b+GR1+ splenic neutrophil counts in placebo- and estrogen-treated mice. (D) and (E) Real-time RT-PCR analysis the expression of NSPs (D), and MPO (E) in splenocytes from placebo- and estrogen- treated mice. The graphs show means±SEMs (n ≥ 4). Unpaired students t tests (placebo vs estrogen) were performed.*, p<0.05; **, p<0.01; and ***, p<0.001.