

there are only a few changes necessary to make it function even better. And another 50% felt that it functions quite well on the whole, and there are only a few changes necessary to make it function even better. Assessment using a validated disease activity measure for SLE was regularly performed by 66.7% of the respondents and they all used Systemic Lupus Erythematosus Disease Activity Index (SLEDAI) with 2 responders using both SLEDAI and British Isles Lupus Activity Group (BILAG). Eighty-eight percent responded that mycophenolate mofetil (MMF) was approved for treatment of SLE in their country with 72.2% responding that it was reimbursed. It was 83.3% and 94.4% for intravenous (IV) cyclophosphamide, 50.0% and 80.0% for tacrolimus, 72.2% and 5.6% for belimumab and 33.3% and 33.3% for rituximab, respectively. MMF was most commonly used in induction therapy for lupus nephritis (40.0%, IQR 25.0 65.0), followed by IV cyclophosphamide National Institute of Health (NIH) protocol (20.0%, IQR 4.0 40.0), IV cyclophosphamide Euro-Lupus Nephritis Trial protocol (15.0%, IQR 5.0 27.5), tacrolimus plus MMF (2.5%, IQR 0.0 8.8), tacrolimus (0.0%, IQR 0.0 5.0), and oral cyclophosphamide (0.0%, IQR 0.0 2.0).

**Conclusions** There are disparities influencing the management of SLE in Asia and Pacific countries. Some of the recommended treatments for SLE are not approved and not reimbursed for management of SLE in some Asia and Pacific countries.

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## 260 MIR-326 PROMOTES RENAL INJURY IN MURINE LUPUS NEPHRITIS

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**Background** MicroRNAs play vital role in the immunopathogenesis of human and experimental lupus nephritis, but the contributions of miR-326 to renal injury in systemic lupus erythematosus (SLE) remain to be demonstrated. Here we characterize the function of the miR-326 in MRL/lpr mice of lupus nephritis.

**Methods** We generated MRL/lpr mice overexpression or silence in miR-326 and analyzed the clinical course of the nephritis with respect to albuminuria. In addition, renal Th17/Treg cells and IL-17A/TGF- expression were detected by flow cytometry and immune-histochemistry respectively.

**Results** miR-326 overexpression did increase the development of albuminuria in MRL/lpr mice. In contrast, miR-326 silence decreased the development of albuminuria. The characterization of renal CD4 +T cells in miR-326 overexpression mice revealed high numbers of infiltrating Th17 cells and low numbers of infiltrating Tregs. IL-17A and TGF- expression respectively increased and decreased in miR-326 overexpression mice.

**Conclusions** miR-326 overexpression plays major role in the immunopathogenesis of lupus nephritis in MRL/lpr mice. Thus, our results suggest that miR-326 may be an intriguing new therapeutic approach for patients with lupus nephritis.

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## 261 MIR-326 REGULATES CD4+T CELLS DIFFERENTIATION IN LUPUS DISEASE OF MRL/LPR MICE

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**Background** CD4 +T cells play a major role in systemic lupus erythematosus (SLE). Many aberrations in miR-326 expression have been described as related to abnormal T cell activation in SLE. The aim of this study was to investigate the effect of miR-326 expression on the differentiation of CD4 +T cells in MRL/lpr mice.

**Methods** 3 groups of female MRL/lpr mice were injected with lentivirus-miR-326 (LV-326) or lentivirus-miR-326 specific inhibitor (LV-sponge) to increase or inhibit miR-326 expression, respectively, and lentivirus-no-encoding (LV-ctrl) as control 10 mice per group. The percentage of Th17, Th1, and Treg cells in spleen were determined by flow cytometry, the expression levels of CD4 +T related cytokines were determined by CBA and ELISA.

**Results** The results showed that, compared with LV-ctrl mice and LV-sponge mice, LV-326 mice had higher percentage of Th17 cells, and lower percentage of Tregs and Th1 cells in splenic CD4 +T cells. In contrast, LV-sponge mice had lower percentage of Th17 cells as well as higher percentage of Tregs and Th1 cells than LV-ctrl mice in splenic CD4 +T cells. Moreover, serum levels of IL-17A were significantly increased in LV-326 mice, compared with LV-ctrl mice and LV-sponge mice. Serum levels of IL-2 and TGF- were decreased in LV-326 mice compared with LV-ctrl mice.

**Conclusions** These findings suggesting that miR-326 regulates CD4 +T cells differentiation and inflammatory-related cytokines production in lupus model mouse. Implying that miR-326 may play a vital role in SLE pathogenesis by regulating CD4 +T cells differentiation.

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## 262 IMMUNOLOGICAL PATHWAYS IN SYSTEMIC LUPUS ERYTHEMATOSUS DISEASE MANIFESTATION: CEREBRAL LUPUS

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**Background** Multiple organ systems including skin, musculoskeletal, neurological, renal and hematologic systems can be involved in patients with SLE. To date extensive research has been conducted to identify unique gene expression signatures using heterogeneous SLE cohorts, however little research has been conducted to delineate SLE signatures in patients with less common disease manifestations, such as cerebral lupus. The diagnosis and monitoring of patients with cerebral lupus is particularly challenging as traditional markers of lupus disease activity in peripheral blood are often negative, and the clinical symptoms overlap with many other non-immunological diseases. The aim of this study was to identify and interrogate immunological pathways that may be aberrant in this particular SLE disease subtype.

**Methods** Whole blood RNA was extracted from 46 SLE patients, 5 Autoimmune encephalitis and 20 healthy controls. Gene expression was measured using a Nanostring nCounter mRNA expression assay incorporating over 500 immunological genes. Data was analysed using Partek Genomics Suite to identify differentially expressed genes and find pathways that may be of interest to interrogate further in the context of SLE disease manifestations.

**Results** Clear differences in gene expression between healthy controls and SLE patients were evident. In our cohort of 46 SLE patients, seven had symptoms of cerebral lupus (seizure and/or psychosis). Cerebral lupus patients showed significantly different expression in 5 genes. TNFRSF1B, CD14 and IL1B expression was increased in cerebral lupus compared to other SLE, while PSMB7 and IKBKB was decreased in cerebral lupus compared to other SLE.

**Conclusions** The differences observed in this group of genes (CD14, PSMB7, IKBKB, IL1B and TNFRSF1B) implicate myeloid cell dysfunction as a possible point of difference between cerebral lupus and other forms of SLE. This preliminary finding may help explain the overlap between the innate and the adaptive immune dysregulation in lupus pathogenesis, and the

use of TNF inhibition acutely in patients with a similar autoimmune cerebral disease, cerebral vasculitis.

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## 263 TIME OF RESPONSE TO IMMUNOSUPPRESSIVE THERAPY FOR LUPUS NEPHRITIS PATIENTS

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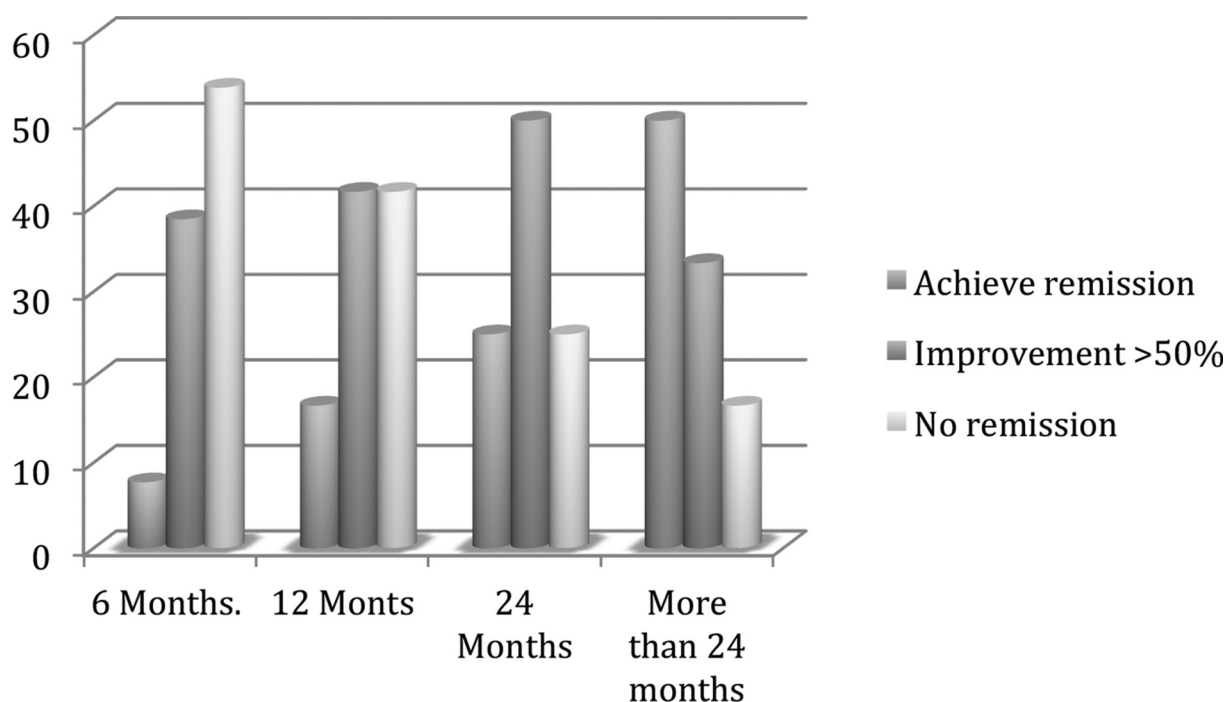
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**Background** Lupus Nephropathy (LN) is an important cause of morbidity and mortality in patients with Systemic Lupus Erythematosus (SLE). The goal of LN treatment is to suppress inflammation and preserve renal structure and function to prevent progression to kidney failure, in addition to minimizing side effects. Currently therapy for severe LN is based on high doses of glucocorticoids and different immunosuppressive drugs.

**Objective** To determine the response time and immunosuppressive drugs used in a series of patients with LN.

**Methods** Retrospective analysis of Lupus patients in a single center with renal disease. The variables recorded were: the number of immunosuppressive drugs used from the diagnosis of LN until remission and the response evaluated regarding 24 hour proteinuria (achieve remission, improvement greater than or equal to 50% respect to baseline and/or no improvement).

**Results** In a series of 80 patients with SLE, 17 were diagnosed with NL, and of these, 14 proliferative diffuse glomerulonephritis (GN IV). The sample consists of 2 men and 12 women between 30 and 65 years of age, with a follow-up time of 6 to 55 months and 9 Caucasian and 5 Latin Americans. All patients were treated with hydroxychloroquine (HCQ),



**Abstract 263 Figure 1** Response time of the LN with immunosuppressive treatment regarding to 24h proteinuria. Blue: normal value of proteinuria 24 h, Red: improvement 50% compared to baseline, Green: no improvement or remission