excluded from this study. All patients underwent Tc-99m ECD SPECT and were classified by the number of positive antiphospholipid antibodies they carried. The heterogeneity of brain perfusion was defined as the coefficient of variation. Analysis of variance (ANOVA) was used to evaluate the differences between groups.

Results Total 60 adult patients were included in this study. There were 54 patients in the case group and 6 patients in the control group. The mean age was 38.3±11.5 years. There were 52 women and 8 men. There was no significant difference in mean brain perfusion between groups (p=0.69). However, Tc-99m ECD SPECT demonstrated significant heterogeneity of brain perfusion in relation to the number of antiphospholipid antibodies (p=0.01).

Conclusions This is the first study to show that Tc-99m ECD SPECT can detect the increased heterogeneity of brain circulation in non-criteria antiphospholipid antibody carriers with neuropsychiatric manifestations.

15 THE ROLE OF INTRACISTERNAL DEPOSITED IGG IN THE PATHOGENESIS OF NEUROPSYCHIATRIC SYMPTOMS OF SYSTEMIC LUPUS ERYTHEMATOSUS

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Background and Aims Systemic lupus erythematosus (SLE) is a chronic autoimmune disease characterised by high levels of autoantibodies and multi-organ tissue damage including damage of the central system. Nerve system disease in SLE includes neurologic and psychiatric events, collectively termed neuropsychiatric SLE (NPSLE). Currently, the pathogenesis of NPSLE is unclear. We investigated pathogenesis of NPSLE in this study.

Methods To understand the pathogenesis of NPSLE, we analysed the medical records of 1131 patients with SLE, and conducted experiments by using lupus-prone mice and mice with intracisternal injections of lupus serum containing high level of autoantibody and mice with gene deficiency.

Results There are 59 patients with NPSLE clinical manifestation including headaches, seizure disorder, cognitive dysfunction, cerebrovascular disease, etc. MRI examination in 14 patients with NPSLE indicates 50% normal and 50% abnormal signals. Lupus-prone mice spontaneously develop meningitis. Meningitis was developed in normal mice with intracisternal injection of lupus serum but not healthy serum; IgG is a major contributor in this meningitis. Monocyte/macrophage, complement and selectin are involved in the development of meningitis induced by lupus serum. This meningitis is dependent on dose of IgG but not associated with kinds of autoantibodies and systemic disease activity. Severity of meningitis induced by lupus serum IgG was significantly reduced in TNF deficient mice compared to wild mice.

Conclusions The deposited IgG or IgG contained immune complex in brain tissue exerts an important role in the pathogenesis of meningitis. This finding will promote development of effective therapeutic strategy to patients with NPSLE.

Parallel Session 2: Cell targeting in SLE

16 HIGH SALT PROMOTES SYSTEMIC LUPUS ERYTHEMATOSUS BY TET2-INDUCED DNA DEMETHYLATION AND DRIVING THE DIFFERENTIATION OF TFH CELLS

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Background and Aims Systemic lupus erythematosus (SLE) is an autoimmune disorder that is characterised by the presence of autoantibodies and immune dysregulation. The pathogenesis of SLE has not been elucidated. The induction of epigenetic changes by environmental factors such as diet may also be relevant. A high-salt diet is considered an important contributor to cardiovascular and renal diseases, and recent research has indicated that a high-salt diet can induce autoimmunity.

Methods In this study, the effects of high salt on various immune cells and in MLR/lpr mice were observed, and the underlying mechanisms were investigated by flow cytometry, high-throughput sequencing, DNA methylation map, ChIP-QPCR.

Results In this study, high salt (sodium chloride, NaCl), under physiological conditions, was demonstrated to increase the differentiation of Tfh. A high-salt diet markedly increased lupus features in MRL/lpr mice. The mechanism is NaCl-induced DNA demethylation via the recruitment of the hydroxytransferase Ten-Eleven Translocation 2 (TET2). Gene silencing of TET2 obviously diminished NaCl-induced Tfh cell polarisation *in vitro*. In addition, the gene expression of sh2d1a, map3k1, spn and stat5b was enhanced after NaCl treatment, consistent with the findings in lupus $CD4^{+}$T cells. However, only spn was directly regulated by TET2, and spn was not the sole target for NaCl.

Conclusions High-salt treatment promotes SLE in mice and the underlying mechanism might be NaCl enhancing Tfh cell differentiation by TET2 inducing global and gene specific DNA demethylation. Our findings not only explain the epigenetic mechanisms of high-salt induced autoimmunity but also provide an attractive molecular target for intervention strategies of SLE.

Parallel Session 4: Lupus reflections across the continents: are we addressing the needs of our patients?

17 CAPABILITIES OF EUROPEAN LUPUS GROUPS: MEMBERS OF LUPUS EUROPE

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Background Lupus patient organizations(POs) are becoming increasingly important stakeholders in political and medical healthcare decision-making processes. LUPUS EUROPE is an umbrella organization of national lupus groups in Europe.

Objectives To identify the different structures and capabilities among European lupus groups.

Methods An online survey was made for the LUPUS EUROPE then 24 member groups from 22 countries.
Results 14 groups (58%) responded from Belgium (2), Cyprus, Denmark, Finland, Greece, Italy, Iceland, Netherlands, Norway, Spain, UK, Sweden and Switzerland.

Key results included:

- 13/14 groups have an elected board of volunteers, 11/14 are run by volunteers
- 9 of the 14 groups are affiliated with the national arthritis and/or rheumatism associations
- 12/14 groups cited membership subscriptions as the main source of funding
- 5/14 groups have an established medical advisory board mainly involved with educational activities on lupus
- Lack of lupus awareness amongst doctors and time to diagnosis were cited as major challenges for people with lupus
- 8/12 groups identified need for capacity building in political lobby activities

More than 2/3rds of the groups expect LUPUS EUROPE to support member groups in their advocacy work and provide scene and opportunity to have more people educated and engaged in improving lupus patient interests in research and political work.
Conclusions There is a diverse range of capabilities and needs amongst national European lupus groups; some are very well established with significant capabilities, while others need capacity building in priority areas.

Parallel Session 5: Lupus nephritis

18 RESIDENT KIDNEY CELLS IN THE PATHOGENESIS OF LUPUS NEPHRIT

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Lupus nephritis is a severe cause of acute kidney injury and an important cause of end-stage renal failure in some regions such as Asia. It is characterised by aberrant innate and adaptive immune responses, autoantibody production and their deposition in the kidney parenchyma, triggering complement activation, increased proliferation of resident renal cells and upregulation of pro-inflammatory molecules leading to inflammatory cell infiltration, all of which culminate in the destruction of normal nephrons and their replacement by fibrous tissue. Anti-dsDNA antibodies are specific to SLE and their level often correlates with disease activity. Apart from mediating pathogenic process through the formation of immune complexes, there is evidence that pathogenic anti-dsDNA antibodies can bind to resident renal cells and induce downstream inflammatory and fibrotic processes. Though clinically effective, current treatment for lupus nephritis entails the use of non-specific immunosuppressive agents and the anti-inflammatory action of high-dose corticosteroids. The clinical and histological impact of novel biologics targeting pro-inflammatory molecules remain to be fully defined. Insight into the underlying mechanisms that induce inflammatory and fibrotic processes in the kidney in lupus nephritis could offer opportunities for novel therapeutic options to improve clinical outcome. This lecture will discuss recent advances in the understanding of pathogenic mechanisms leading to inflammation and fibrosis in the kidney in lupus nephritis, with particular focus on the contribution of resident renal cells.

19 WHAT CAN WE LEARN FROM STUDIES OF VASCULITIS?

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The kidney, particularly the glomerulus, is vulnerable to immune and inflammatory injury via a variety of humoral and cellular mechanisms. In lupus nephritis, both arms of the adaptive effector response, together with innate effectors, can be prominent participants. Furthermore, in systemic lupus erythematosus (SLE) there is reactivity to multiple autoantigens that can be planted in glomeruli, be deposited as components of circulating immune complexes, or be intrinsic to the glomerulus itself.

Other forms of autoimmune renal disease are characterised by autoimmunity to a more restricted range of autoantigens. Thus, examining effector mechanisms in autoimmune diseases such as myeloperoxidase anti-neutrophil associated glomerulonephritis (MPO-ANCA) associated nephritis and autoimmune anti-glomerular basement membrane (GBM) disease can take arguably a more reductionist approach compared to lupus nephritis.

Published and unpublished data in studies in experimental models of these forms of renal vasculitis will be discussed, focusing on the role of cell mediated responses and renal injury in these diseases. The potential relevance of these studies to SLE and lupus nephritis will be highlighted.

20 AURION STUDY: 24-WEEK DATA OF MULTI-TARGET THERAPY WITH VOCLOSPORIN, MMF AND STEROIDS FOR ACTIVE LUPUS NEPHRITIS

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Background and Aims In lupus nephritis (LN), complete remission (CR) or partial remission is associated with better patient and renal survival. Subjects who do not achieve a 25% reduction in proteinuria within 8 weeks of starting induction immunosuppression are unlikely to achieve even a PR. Voclosporin (VCS) is a novel CNI demonstrating less pharmacokinetic–pharmacodynamic variability and a potentially improved safety profile compared with other CNIs.
Methods
Entry criteria renal biopsy within 24 months (Class III; IV-S, IV-G (A) or (A/C); V, III/V, IV/V, ISN/RPS); urine protein:creatinine ratio (UPCR) $\geq$1.0 mg/mg (III/IV) or UPCR $\geq$1.5 mg/mg (V); serologic evidence of active LN; and eGFR >45 mL/min/1.73m^2. AURION assessed the ability of biomarkers at 8 weeks to predict clinical response over 24 and 48 weeks when taking voclosporin (VCS) 23.7 mg po BID in combination