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

S Yung. [1]*The University of Hong Kong, Department of Medicine, Hong kong, Hong Kong S. A.R*

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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?

[1]AR Kitching*. [1]*Monash University/Monash Health, Centre for Inflammatory Diseases- Dept Medicine, Clayton- Melbourne, Australia*

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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

[1]RB Huizinga*, [2]R Yahya, [3]AHA Gafor, [1]N Solomons, [1]L Veasey. [1]*Aurinia Pharmaceuticals Inc, Clinical Affairs, Victoria, Canada*; [2]*Hospital Kuala Lumpur, Department of Nephrology, Kuala Lumpur, Malaysia*; [3]*Jalan Yaakob Latif- Bandar Tun Razak, Department of Nephrology, Kuala Lumpur, Malaysia*

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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

with MMF (1–2 g/day) and reducing corticosteroid dose. We report 24 week data.

Results In this study, 7/10 (70%) subjects achieved CR at 24 weeks. Of the 10 subjects that achieved a≥25% reduction in UPCR at 8 weeks, 80% were responders (61% reduction in UPCR over baseline) at 24 weeks. In addition, inflammatory markers such as C3, C4 and anti-dsDNA all continued to normalise to 24 weeks. Renal function remained stable. VCS was well-tolerated with no unexpected safety signals observed.

Conclusions The results suggest that early response to therapy of VCS in combination with MMF may predict 24 week CR in the presence of low steroids in active LN. 48 week CR data will be presented at the meeting.

Parallel Session 7: Manifestations, comorbidities and complications

21 INFECTIONS IN THE ASIA PACIFIC REGION

W Louthrenoo*. *Faculty of Medicine- Chiang Mai University, Division of Rheumatology-Department of Internal Medicine, Chiang Mai, Thailand*

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Infections in the Asia Pacific Region Infections in patients with systemic lupus erythematosus (SLE) are not uncommon, and are major causes of morbidity and mortality. The prevalence of infections is high among developing countries, and those with low socioeconomic status, particularly in Asia. Dysregulation of the immune system by the disease itself and the use of corticosteroids and immunosuppressive drugs increase susceptibility to infection, which can cause by both usual and opportunistic pathogens. Infections caused by viruses, bacteria, mycobacterium, fungi, and parasites have been described. Varicella zoster, Salmonella spp., both Mycobacterium tuberculosis and non-tuberculosis, Nocardia spp., Aspergillus spp., Pneumocystis jiroveci, etc. are common opportunistic pathogens.

Diagnosing infections in SLE is sometimes difficult. Acute infections can cause protean manifestations that sometimes simulate disease flare. Atypical presentations are not uncommon. Fever and leukocytosis might not be present due to the use of corticosteroids and immunosuppressive drugs. Occult infections can be overlooked if not searched for carefully. Furthermore, infections themselves can trigger disease flare. A high level of hsCRP correlates well with infection. Procalcitonin can be used as a marker for bacterial infection.

Treatment of infections in SLE also is problematic. Use of high dose corticosteroids and immunosuppressive drugs to control SLE activity can reactivate latent infections, or exacerbate current infections, making them more difficulty to control. Infections should be suspicious in SLE patients with fever or clinical presentations that do not respond to appropriate SLE treatment. Appropriate evaluation is needed and treatment should be started immediately to cover pathogens most likely possible, and prevent morbidity and mortality.

22 MYOCARDIAL DIFFUSION WEIGHTED IMAGING REVEALS SUBCLINICAL MYOCARDIAL INFLAMMATION IN PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS

[1]Q Yan*, [2]L Wu, [1]Q Guo. *[1]RenJi Hospital- School of Medicine- Shanghai JiaoTong University, Department of Rheumatology, Shanghai, China; [2]RenJi Hospital- School of Medicine- Shanghai JiaoTong University, Department of Radiology, Shanghai, China*

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Background and Aims To evaluate whether diffusion weighted imaging can assess myocardial oedema in patients with systemic lupus erythematosus (SLE).

Methods 32 patients (mean age 36±8 years) with SLE and 20 controls (mean age 47±6 years) underwent cardiac MRI at 3.0 T. Standard cine images were obtained. DWI and T2 mapping were acquired in a mid-cavity short-axis plane. Late gadolinium enhancement (LGE) images were obtained 15 min after 0.2 mmol/kg of contrast. all patient were subdivided in to late gadolinium enhancement-positive (LGE+) and LGE-negative (LGE-) group according to the presence and absence of enhancement on LGE image.

Results SLE patients had low disease activity (mean SLE disease activity index score 0.74±0.5). There were no differences in LV size or function between SLE patients and controls. Only 11 subjects had LGE. SLE LGE+ subjects had highest ADC value among the three groups. SLE LGE+ subjects had higher ADC (apparent diffusion coefficient) than LGE- subjects. SLE LGE- subjects had higher ADC than control ($p<0.05$). T2 value of SLE LGE+ was no significant difference with SLE LGE- subjects. Repeated measures were highly correlated by linear regression for both inter- and intraobserver analysis (both $R=0.75$, $p<0.001$). ADC mapping identifies increased in SLE patients, likely due to subclinical myocardial oedema.

Conclusions These findings suggest that even in SLE patients with inactive disease and normal cardiac function, ADC mapping as a novel quantitative and highly reproducible technique can detected low grade myocardial inflammation.

Parallel Session 8: Innate immunity and interferon

23 CYCLIN DEPENDENT KINASE 1 : A NOVEL REGULATOR CONTROLLING TYPE I INTERFERON SIGNALING AND POTENTIAL TARGET FOR THERAPEUTIC INTERVENTION IN SLE

[1]L Wu*, [1]B Qu, [1]Y Qin, [1,2]N Shen. *[1]Renji Hospital- School of Medicine- Shanghai Jiao Tong University, Shanghai Institute of Rheumatology, Shanghai, China; [2]Medical Centre, The Centre for Autoimmune Genomics and Aetiology CAGE, Cincinnati- Ohio, USACincinnati Children's Hospital*

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Background and Aims Abnormal epigenetic changes are involved in over-activated pathogenic IFN signalling in SLE. However, the mechanisms are still not clear. We tried to identify novel epigenetic regulators of IFN signalling pathways in SLE.