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/1.73m². 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

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