for heating, and living in an agricultural area), and for SLE, disease damage (Brief Index of Lupus Damage, BILD) score and age of lupus diagnosis. Multivariable logistic regression models assessed the association of each exposure with SLE status, and linear regression models to examine the association of each exposure with disease damage (i.e. BILD score) for SLE patients, both adjusting for age, sex, race/ethnicity and education.

Results We included 359 SLE patients with an average diagnosis age of 32±11 years and 106 healthy controls. Ninety-one percent of SLE patients and 89% of controls were female. SLE patients were older than controls, with a mean age of 49±14 vs. 41±16 (p<0.05). Both groups were racially/ethnically diverse, with over 65% identified as non-White. Over half of SLE patients (56.7%) reported any exposure compared to 40% of controls (p<0.05). Specific exposures of the SLE group ranged from 8.8% for kerosene to 28.9% for agricultural exposure (see table 1). There was a higher prevalence of any exposures among SLE patients vs. controls (OR 1.9, 95% CI 1.2–3.0, p<0.05), as well as specific exposures to pesticides (exterminator) (OR=1.9, 95% CI 1.0–3.7), other home insecticide use (OR=2.1, 95% CI 1.1–4.1, p<0.05) and kerosene (OR=2.8, 95% CI 0.8–9.7). We found no association between residential exposures and BILD score in the SLE-only analysis.

Conclusions SLE diagnosis was associated with increased odds of residential exposures, but this study is under powered to fully examine individual exposures. Lack of information on timing of exposures is another study limitation. While this study demonstrates the prevalence of exposures, the observed trend warrants further investigation with assessments of exposure timing relative to age and SLE diagnosis.

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258 NK GENE SIGNATURE IN SLE

1Nicole I. Fewings*, 2Sanjay Swaminathan, 3David Booth, 4Ming Wei Lin. 1Westmead Institute for Medical Research; 2Department of Clinical Immunology, Westmead Hospital, Sydney, New South Wales, Australia Department of Medicine, University of Sydney, Sydney, New South Wales, Australia Department of Medicine, Western Sydney University, Sydney, New South Wales; 3Centre for Immunology and Allergy Research, Westmead Institute for Medical Research, University of Sydney, Westmead NSW, Australia; 4Dept of Clinical Immunology and Immunopathology, Westmead Hospital, Westmead NSW 2145; and Faculty of Medicine, Sydney Medical School, University of Sydney, NSW 2000

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259 HEALTHCARE SYSTEM AFFECTING SYSTEMIC LUPUS ERYTHEMATOSUS IN ASIA-PACIFIC COUNTRIES

1Chan-Bum Choi*, 2Sheereen Oon, 3Mandana Nikpour, 4Sang-Cheol Bae. 1Hanyang University Hospital for Rheumatic Diseases; 2Melbourne University; 3Department of Rheumatology, Hanyang University Hospital for Rheumatic Diseases

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Background SLE has traditionally been considered a disease of dysregulated B cells and the production of pathogenic autoantibodies. Evidence that other cell types contribute to the observed pathology is being recognised with the advent of a new techniques providing focused avenues of exploration. The aim of this study was to determine which genes are differentially expressed in patients compared to healthy autoimmune controls. This may then be lead to downstream intracellular signalling pathways and aberrant cytokine production which may contribute to the understanding of the pathogenesis of SLE.

Methods Whole blood RNA was extracted from 46 SLE patients with heterogeneous disease manifestations and at different stages of disease activity, 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.

Results Gene expression analysis unsurprisingly showed higher expression of interferon-related genes in SLE (MX1, GBP1, IRF7, IFIT2), however we also found that there were genes that were under-expressed. An NK signature with genes such as KLRC2, KLRC1, KLRB1, KLRF1, KLRG1, PRF1 and IL2RB shows the potential value of the approach to discern the important cell types in the development of SLE.

Conclusions The most significantly differentially expressed genes in SLE show an NK signature, with genes that encode activating and inhibitory NK receptors having reduced expression compared to healthy controls. This NK signature recognises a role for this arm of the innate immune response as a unique fingerprint of SLE.

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