Introduction

Environmental and intrinsic factors play a pathogenic role in SLE through modulation of immune system regulatory responses. Stimuli such as UV sunlight, hormones, infection and stress are accepted symptoms exacerbation effectors, however, reasons for flare occurrence often remain unclear.

Household product chemicals, time spent indoors, and housed and personal hygiene practises may increase an individual’s potential for environmental chemical exposure (ECE). This study concentrates on household product usage patterns in 80 SLE participants defined by American College of Rheumatology (ACR) criteria, examining the correlation of product and chemical exposure with flare event days over one year.

Study Hypothesis

Illness flares in SLE patients can be triggerd by the inhalation, ingestion and contact with environmental agents found and used routinely in the indoors living and work environment.

Method

ECE was established through retrospective self-report questionnaires responses (flare history, home environment, commercial product usage). Questionnaires were analysed to establish counts of SLE flare and ECE days over 1 year. Definition and explanatory example of ‘Flare’ was included in the methods to standardise participant understanding of what constitutes a flare.

Flare definition:

Illness flares in SLE patients can be triggered by the inhalation, ingestion and contact with specific environmental agents found and used routinely in the indoors living and work environment.


ECE was estimated after collation of self-reported product/chemical exposure activity. A product and chemical exposure matrix (PACEM) was developed as part of this study: product chemical component groups were assigned using published literature databases, labelled ingredients and material safety data sheets. Products were coded into 32 product groups and 29 chemical groups.

Product and chemical groups were allocated a binary score (“absent”/”presence”). Weighted scores were not assigned due to insufficient data regarding chemical concentrations within nominated products.

General linear modelling (negative binomial robust link function) was performed for flare, product and chemical exposure day counts adjusted for significant covariates(p ≤ 0.05).

Conclusions

Relative risk increase associated with immune-modulation therapy suggests that participants on therapy have more severe disease with sub-optimal clinical benefit from therapy.

The UV-protective effects of makeup and makeup pigments may reduce the number of flare days in photosensitive lupus patients. The lack of clear correlation between ECE and flares for other chemicals/products may be explained by such factors as small sample size, self-reporting bias, and the lack of accounting for chemical concentration, adimixure and chemical multiplicative toxicity. Study size precluded modelling for observed dose-response nonlinear non-monotonicity.

A wider study incorporating biological and environmental sample analysis would strengthen ECE quantification and validate the PACEM tool. This would provide direct assessment of ECE and correlation with lupus flare activity.

Our study suggests that lupus flares reflect complex interactive chemical effects requiring nonlinear modelling.

Acknowledgements

• Assoc Prof John Atia
• Assoc Prof Howard Bridgman
• ARRC staff and study participants
• Val Badham University of Newcastle Foundation Scholarship for Immunology

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