Home and Personal Product Chemical Exposure and Flare Events Of SLE

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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 symptom exacerbation effectors, however, reasons for flare occurrence often remain unclear.

Household product chemicals, time spent indoors, and household 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 triggered 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:

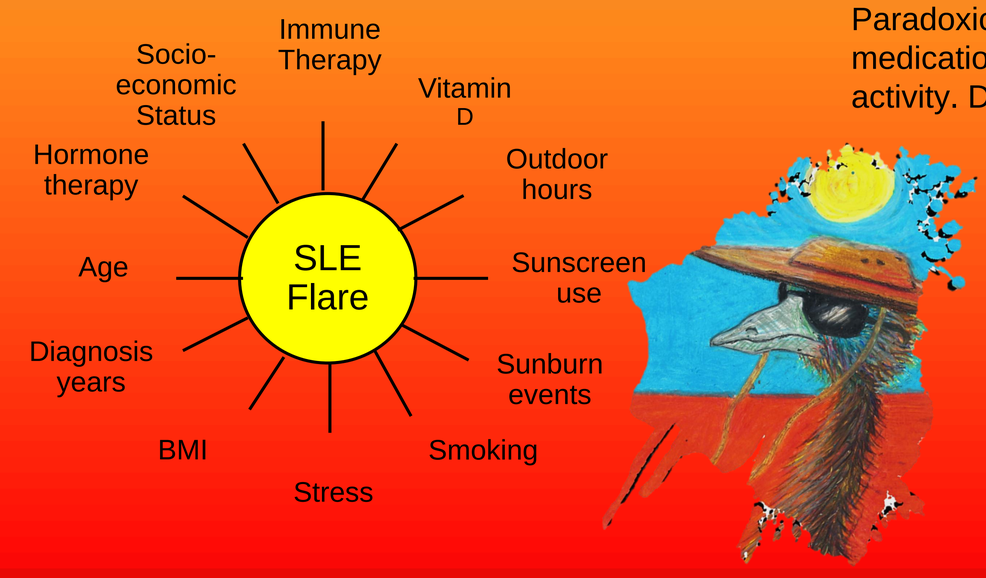
Illness flares in SLE patients can be triggered by the inhalation, ingestion and contact with specific environmental agents found and used routinely in the indoors living and work environment.

Poser CM et al. Ann Neurol 1983

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 ("absence"/"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($p \leq 0.05$).



Environmental Product Groups

Adhesives	Cleaners Carpet	Automated Dishwashing	Solid Fuel	Cleaning acid	Beauty colourant
Air freshener/ deodoriser	Cleaners Beauty	Disinfectants / antiseptics	Fixatives	Powder beauty	Perfume
Cleaners general	Ammonia	Cleaners caustic	Herbicides Insecticides pesticides	Solvent	Flame retardant
Bleach	Degreasers	Dry Cleaning	Paint	Bath oil	Fluoride
Cleaner furniture	Deodorant beauty	Equipment Oil & petrol	Make-up	Powdered salt	
Protector wax / polish	Hair remover	Fertilizer	Cleaner laundry	Hydrocarbon (accelerant)	

34 Product groups

Environmental Chemical Groups & Subgroups

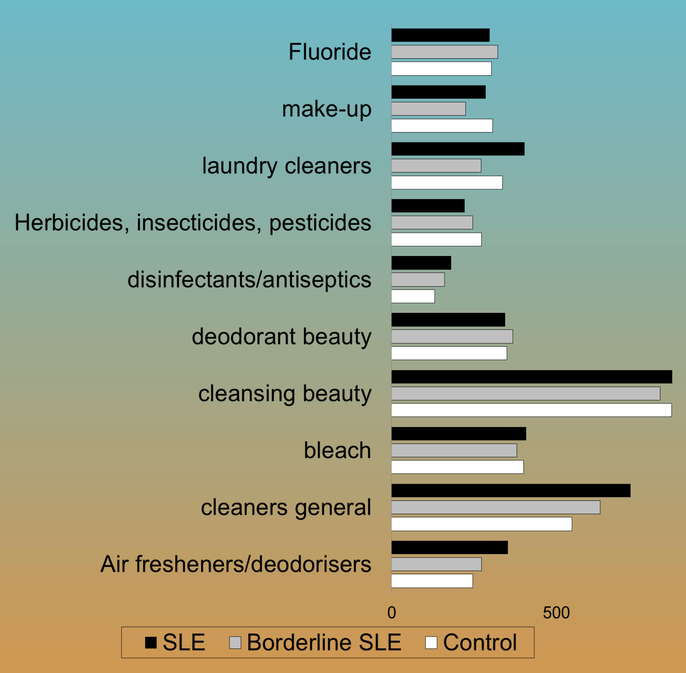
Epoxy Resin	Solvents	Formaldehyde	Fragrance	Hydrocarbon	Organo-chlorines
Surfactants	Organotins	Bleach	Organo-phosphates	Paradichloro-benzene	Disinfectants
Preservatives	Pesticides	Fixatives	Parabens	Phthalates	Bisphenol A
Perflourinates	Brominated Flame retardants	Triclosan	Phenols	Acids	Petroleum distillates
Pigments	Alkalis	Fluoride	Aromatic Amines	Silicones	

29 Chemical groups & sub groups

Results

SLE and non-SLE product group usage comparison showed pattern consistency in regard to the top 10 frequently used products. The SLE group had significantly higher usage of general cleaning products ($p=0.015$), however differences between groups were not significant for any other product group.

Mean Top 10 Product Group Exposure Days



Multivariate models for SLE participant flare days and product group exposure days showed significance ($p \leq 0.05$) for 7 out of 34 product groups and 9 out of 29 chemical groups.

Paradoxically, the use of immune therapy medications indicated an elevated risk of flare activity. Dose-response curves for products and chemicals displayed nonlinear non-monotonic responses with consistent patterns within products and some chemical components.

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, admixture 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.

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