weeks 16, 32 and 36. Type I Interferon (IFN) induced gene signature was determined using a previously validated four gene interferon score (IFI27, IFI44, IFI44L, RSAD2) using quantitative PCR.

Results No local or systemic allergic reactions occurred following Omalizumab injections. Of the 52 adverse events (AEs), 49 were reported as mild while 3 met the criteria for severe AEs. SLEDAI 2K scores improved significantly in the Omalizumab group at week 16 ($p=0.038$), as well as during the open label phase in subjects initially receiving placebo ($p=0.020$). No worsening in BILAG scores or PGA were detected. A total of 26% (4/15) patients achieved SRI4 response criteria with Omalizumab. IFN gene signature improved in subjects treated with Omalizumab ($p=0.11$), especially in subjects with high baseline IFN scores ($p=0.052$).

Conclusions Omalizumab in subjects with SLE is well tolerated and associated with improvement in disease activity potentially linked, at least in part, to modulation of type-I IFN responses. Larger randomized clinical trials are required to assess the role of this drug in patients with SLE.

Trial registration

Clinical Trials registration number NCT01716312

Environmental Factors

EF-01 SOCIOECONOMIC DETERMINANTS AND SLE SEVERITY IN BLACK PATIENTS IN BARBADOS

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Background SLE patients of lower socioeconomic position (SEP) exhibit increased morbidity and mortality but less clear is whether the disease incidence is increased in the setting of poverty and whether a more severe disease phenotype develops in poorer patients. Crucial etiologic factors for SLE may predominate in genetically-predisposed individuals of lower SEP. Lack of running water at age 12 and frequent childhood infections- both of which are associated with poverty, have been implicated in some groups. In our cohort of black patients in Barbados, we sought to determine by comparison with the general population if SLE clusters in persons of lower SEP and if poorer patients had a more severe disease phenotype.

Methods Patients from the Barbados National Lupus Registry (universal access to care) were included if they had a clinical diagnosis of SLE made by a rheumatologist in the 7 year period 2008–2014.

The clinical status was assessed at the end of 2017 ensuring all patients had at least 3 years of follow up – the period of time within which most severe SLE organ involvement develops. Private health insurance coverage was used a proxy for SEP. Patients were divided into 2 groups based on the presence or absence of health insurance at disease diagnosis. We investigated the effect of SEP on major organ involvement (yes/no), SLE nephritis (yes/no), neuropsychiatric disease (yes/no), and vital status in December 2017 (alive, alive with chronic kidney disease, died).

Results In a subset of 135 patients with SLE ($F=126$), 29 (21%) had private healthcare insurance at diagnosis (median age 32), the remaining 106 (79%) did not (median age 35).

This compares to a national prevalence of private healthcare of 24% (95% CI 21% to 26%). The association between health insurance status and each of the four complications/outcomes is presented in table 1. After adjusting for disease duration, patients without private health insurance at diagnosis had more complicated SLE and worse SLE outcomes (Odds Ratios >1) in all cases. For two of these clinical complications (major organ involvement and SLE nephritis) the effects were borderline statistically significant at the 5% level.

Conclusions Socioeconomic determinants appear to affect the severity of SLE in Barbados with poorer patients having a more severe disease phenotype.

The high morbidity and mortality documented in the international literature in the setting of poverty may be due to a combination of social issues and the presence of more aggressive disease from the outset.

Abstract EF-01 Table 1 The association between no health insurance at diagnosis and disease outcome

Outcome	Odds ratio	95% CI	P value
Major organ involvement †	2.69	1.00–7.26	0.05
Lupus nephritis †	3.24	1.01–10.37	0.05
Neuropsychiatric lupus †	4.40	0.55–35.16	0.16
Vital status ‡	3.83	0.84–17.45	0.08

† logistic regression, adjusting for disease duration

‡ vital status with 3 levels: 1 (alive), 2 (alive with chronic kidney disease), 3 (died). Ordinal logistic regression, adjusting for disease duration.

EF-02 LONGITUDINAL STUDY OF LONG-TERM POVERTY AND PERSISTENT DEPRESSIVE SYMPTOMS IN SLE

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Background A prior study found that persons with SLE in long-term poverty have greater accumulation of disease damage over 6 years than those exiting poverty or never in poverty.¹ The present study evaluates the effect of long-term poverty status on depressive symptoms over the same duration of time.

Methods Data are from the UCSF Lupus Outcomes Study in which persons with SLE were recruited in 2003 throughout the U.S. and interviewed annually through 2015. In each year we characterized respondents' poverty status based on household income and family size and administered the CESD measure of depressive symptoms, defining a high level of depressive symptoms using a validated SLE-specific cutpoint (≥ 24) associated with a formal diagnosis of depression.² Prevalent persistent depression was defined as having high levels of depressive symptoms for ≥ 3 years between 2009 and 2015. Incident persistent depression used the same criteria, measured only among those who had low levels of depressive symptoms between 2006 and 2009. Logistic regression was used to estimate the impact of being poor in every year from 2003–2009, permanently leaving poverty by 2009, or never being poor on prevalent and incident persistent depression, with and without adjustment for gender, age, marital status, race/ethnicity, education, disease duration, extent of accumulated damage by 2009 using the Brief Index of Lupus Damage,³ smoking status, and BMI.

Results 535 persons with SLE were interviewed in each year from 2003 to 2015 (94% female, 65% non-Hispanic whites, mean age in 2003 50 years, range 20–83, mean disease duration 17 years, range 1–51). Between 2003 and 2009, 81% were never poor, 8% exited poverty, and 11% were poor in every year. 89 of the 535 (16.6%) met the study definition of prevalent persistent depression; 23 (7.4%) of the 312 free of high levels of depressive symptoms from 2006–2009 had incident persistent depression as of 2015. Table 1, below, indicates that those who were poor in every year had significantly higher rates of prevalent and incident persistent depression than those exiting poverty or never poor.

Conclusions Public policy to help persons with SLE stay out of poverty or to exit poverty may lower their rates of prevalent and incident persistent depression. Attention to the economic status of persons with SLE should be part of an overall treatment strategy including treatment for depression since such attention may help reduce accumulation of damage as well as reduce the prevalence and incidence of persistent depression.

Abstract EF-02 Table 1 Odds ratios (95% CI) for effect of poverty status between 2003 and 2009 on prevalent and incident persistent depression (CESD score ≥ 24 in 3 or more years), between 2009 and 2015, with and without adjustment

Outcome	Poor every year	Exited poverty	Never poor
Prevalent Persistent Depression (n=535)			
Unadjusted	3.9 (2.1, 7.2) *	1.0 (0.4–2.2)	Ref.
Adjusted†	2.9 (1.4, 6.1) *	0.8 (0.3–1.8)	Ref.
Incident Persistent Depression (n=312)‡			
Unadjusted	4.5 (1.3, 15.4) *	0.9 (0.2–3.9)	Ref.
Adjusted†	4.1 (0.9–19.1)	0.7 (0.1–3.7)	Ref.

*p<0.01

† Adjusted for gender, age, marital status, race/ethnicity, education, disease duration, extent of accumulated damage as of 2009, smoking status, and BMI

‡ Excludes persons with high levels of depressive symptoms 2006–2009

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EF-03 MICROBIOTA-ASSOCIATED TRYPTOPHAN CATABOLISM INDUCES AUTOIMMUNE ACTIVATION IN A MOUSE MODEL OF LUPUS

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Background Tryptophan (Trp) is an essential amino acid that is used for the biosynthesis of key compounds such as serotonin (5HT), kynurenine (Kyn), and AhR ligands. The gut microbiome is a critical participant in Trp metabolism through which it modulates many biological pathways, including

immune activation with the generation of AhR ligands. High Kyn and low 5HT levels have been found in patients with SLE. Disturbances in gut bacterial communities, defined as dysbiosis, have been found in lupus patients and lupus mice. Moreover, a recent study demonstrated that the translocation of gut pathobiont in a lupus mouse model as well as in SLE patients related with autoimmune pathogenesis. The hypothesis tested in this study is that the interplay between gut microbiota, Trp metabolism, and genetic susceptibility modulates systemic autoimmunity.

Methods We used the B6.Sle1.Sle2.Sle3 (TC) lupus-prone mouse model that shares over 95% of its genome with control B6 mice. Sequencing of fecal 16S rDNA was performed by standard methods. Serum and feces Trp metabolites were quantified by mass spectroscopy. Gnotobiotic (GF) B6 mice were colonized with feces from either TC or B6 mice, their immune phenotypes analyzed 4 weeks later. The immunophenotypes of TC mice analyzed after 4 month exposure to various levels of dietary Trp.

Results TC and B6 mice have a distinct gut microbiota, and transfers of TC fecal microbiota induce a transient autoimmunity in GF B6 mice. Autoimmune activation was also mitigated by horizontal microbiota transfers between co-housed TC and B6 mice. As SLE patients, TC mice present high Kyn and low 5HT levels in their serum and feces, and this metabolite imbalance was eliminated by a broad-spectrum antibiotic treatment. Furthermore, variations in dietary Trp modulated autoimmune pathogenesis: a low Trp diet prevented the development of autoimmunity while a high Trp diet accelerated disease progression, and the production of their anti-dsDNA IgG was positively correlated with their Kyn level. Finally, feces from TC mice fed with high Trp levels induced a higher autoimmune activation than feces from TC mice fed with low Trp levels in GF B6 mice.

Conclusions These results demonstrate the existence of interactions between lupus susceptibility genes and gut dysbiosis, where the full disease phenotype requires the presence of both. Furthermore, our results suggest that gut dysbiosis alters Trp metabolism in genetically lupus susceptible mice by expanding Trp-catabolizing bacteria and that some of these alterations contribute to autoimmune activation.

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EF-04 ASSOCIATION OF ULTRAVIOLET-B RADIATION AND RISK OF SLE AMONG WOMEN IN THE NURSES' HEALTH STUDIES

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Background Ultraviolet-B radiation (UV-B) exposure may lead to worsened photosensitivity, rashes, and systemic flares among SLE patients. Although UV-B radiation damages keratinocytes and may result in production of novel forms of autoantigens, it remains unknown whether UV-B exposure increases the risk of developing SLE. We aimed to examine the association of UV-B exposure with risk of incident SLE in a large