responders but a low type I IFN signature and higher TNF-alpha expression in both HCQ/QC-nonresponders and HCQ/QC-responders.

Conclusions An increased number of mDCs may contribute to HCQ-refractoriness and predict a better response to treatment with both HCQ and QC but do not contribute to HCQ/QC-refractoriness. The significant correlation between macrophages and CLASI scores in the HCQ/QC-nonresponders, a finding not seen in either HCQ or HCQ/QC-responders, may also indicate that macrophages are more involved in antimalarial-refractory skin disease. The difference between the responders and nonresponders is further confirmed by the cytokine staining and mRNA expression. Our data is an initial step in determining the activation pathways that account for the lack of response to antimalarials.

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### Abstracts

**II-10 ROLE OF MACROPHAGE-DRIVEN AUTOINFLAMMATION IN SLE**

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**Background** Although SLE is a prototype of autoimmune (T cell/B cell-mediated) disease, there is increasing evidence implicating myeloid cells in its pathogenesis. We examined the role of macrophages (MΦ) in human SLE and the pristane-induced lupus model.

**Methods** Tissues of SLE patients and mice with pristane-induced lupus were examined by double immunohistochemistry. MΦ were analyzed by flow cytometry, phagocytosis assay, real-time PCR, Seahorse assay, and RNA-Seq. Mice were injected daily with the liver X receptor (LXR) agonist T0901317.

**Results** SLE patients’ bone marrow contained numerous activated caspase-3+ apoptotic cells located outside of MΦ. In contrast, in leukemia patients undergoing bone marrow ablation prior to transplantation, all caspase-3+ cells were inside MΦ, suggesting that lupus is associated with impaired clearance of apoptotic cells. MΦ in pristane-lupus took up fluorescently-labeled apoptotic cells poorly compared with controls. Studies in pristane-treated knock-out mice indicated that the phagocytosis of dead cells in the lung by ‘natural’ IgM (DAH absent in μMT mice, restored by infusion of IgM), C3, and C3b receptors (absent in C3-/- and CD18-/- mice, prevented by complement depletion with cobra venom factor). DAH also was prevented by MΦ depletion (clodronate liposomes) but not neutrophil depletion (anti-Ly6G). MΦ in pristane-induced lupus exhibited features of classical activation (impaired phagocytosis of apoptotic cells, high glycolysis, low oxidative phosphorylation, increased HIF1α expression, M1 surface markers, and TNFα production), whereas control MΦ from mice treated with mineral oil (do not develop DAH) were M2-like, with high phagocytic activity, low glycolysis, high OxPhos, increased LXRx expression, M2 surface markers, and IL-10 production. In both pristane-induced lupus MΦ and SLE patients’ monocytes, we found low levels of the LXRx-regulated reverse cholesterol transporter ABCA1. We hypothesized that it might be possible to treat DAH by ‘re-polarizing’ MΦ using a synthetic LXR agonist. Consistent with that idea, pristane-treated mice receiving T0901317 did not develop DAH and MΦ from mice receiving T0901317 exhibited an M2-like surface phenotype and decreased TNFα production compared with controls.

**Conclusions** Our data suggest that lupus-associated DAH is partly an autoinflammatory response caused by sluggish clearance of apoptotic cells by M1-like MΦ and that certain clinical manifestations (e.g. DAH) can be treated by repolarizing MΦ using an activator of the transcription factor LXRx. Low levels of LXRx-driven ABCA1 expression may also have implications for the therapy of atherosclerosis in SLE.
macrophages transfected with hY3. IFIT1 MX1, and EIF2AK2 transcripts were significantly increased in the WISH cells treated with hY3 macrophage supernatants, but not macrophage supernatants alone (n=7, p=0.02).

Conclusion These data now provide a link between IFN and the inflammatory and possibly fibrosing component of CHB and position Siglec-1 positive macrophages as integral to the process.

Living with Lupus

LL-01 SOCIAL DETERMINANTS OF TREATMENT ADHERENCE AND DISEASE SEVERITY AMONG PEOPLE LIVING WITH LUPUS IN A SMALL ISLAND DEVELOPING STATE: A REPORT FROM ST. LUCIA

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Background The occurrence of systemic lupus erythematosus (SLE) varies considerably worldwide, with documented high incidence rates among women of African descent. The clinical course is likely influenced by social determinants, including socioeconomic position (SEP), yet findings remain inconsistent, with little information from the Caribbean diaspora. This study presents the epidemiology of SLE in St. Lucia for the first time, exploring the association of SEP and SLE medication adherence and disease severity.

Methods Data have been collected from the only specialist lupus clinic in St Lucia between 1995 and 2017. We explored the effect of selected markers of SEP on disease severity (yes/no), and treatment adherence (yes/no) using logistic regression, adjusting for the effects of age, sex and years since diagnosis at all times. We used education level (primary or secondary education, tertiary education) or patients eligible for treatment cost discount or exemption (yes/no) as indicators of SEP. We also explored the effect of enrolment in a self-help programme on both regression outcomes, and the effect of treatment adherence on disease severity.

Results 143 people with SLE have registered at the clinic between 1995 and 2017. The mean age at diagnosis was 32 years (standard deviation 12 years), and 132 (92%) were female, for a female to male ratio of 12 to 1. Since 2010 (a period of full clinic operation) 66 women have been diagnosed with SLE, for a crude incidence rate of 9.3 per 100,000 person years (95% CI 7.2 to 11.8). Half (49%) had a severe clinical course, defined as having cerebritis, nephritis, or being on dialysis, and half (50%) were medication adherent at their last follow-up visit. Higher SEP was consistently associated with increased treatment adherence and decreased disease severity (treatment adherence odds ratios ranged from 2.4 to 3.4; disease severity odds ratios ranged from 1.0 to 3.5) (figure 1).

Conclusion In St Lucia, among a population of predominantly African descent, and using selected markers of SEP, patients of lower socioeconomic position have more severe disease and lower medication adherence than those of higher socioeconomic position.

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LL-02 DEPRESSION IN PATIENTS WITH CHRONIC CUTANEOUS LUPUS ERYTHEMATOSUS: PREVALENCE AND ASSOCIATED FACTORS IN A PREDOMINANTLY AFRICAN AMERICAN COHORT FROM THE SOUTHEAST U.S

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Background Chronic dermatologic diseases in general and cutaneous involvement in patients with systemic lupus erythematosus (SLE) in particular have been linked to increased depression. However, little is known about the burden of depression and its risk factors in patients with primary chronic cutaneous lupus erythematosus (CCLE), the most common type of cutaneous lupus. Additionally, previous studies examining CCLE have included predominantly white patients, despite recent findings indicating that black individuals have higher susceptibility for this condition and experience earlier damage in the disease course. We aimed to examine the prevalence of depression in patients with primary CCLE in the Southeast