Abstract PS7:140 Table 1 Baseline characteristics

Age (years) #	42.26 (38.67-48.03)
Sex (female)	16 (89%)
Age at SLE diagnosis #	34.33 (26.56-45.91)
Disedase duration (years) #	8.05 (3.54-14.02)
Previous sDMARD #	2.50 (2.00-4.00)
- Hydroxycloroquine	16
- Methotrexate	12
- Mycophenolate mofetil	9
- Lefhunomide	7
- Azatioprine	7
Previous Cyclophosphamide	4
Previous bDMARD	
- Rituximab	7
- Efalizumab	1
- Abatacept	1
Concomitant sDMARD	
- Hydroxycloroquine	13
- Methotrexate	7
- Azatioprine	1
- Mycophenolate mofetil	1
- Lefhunomide	1
ANA #	1/360 (1/160-1/1280)
- Anti-Ro	9
- Anti-La	4
- Anti-Sm	4
- Anti-RNP	4
Rheumatoid factor	5 (27.8%)
Anti-CCP	1 (5.6%)
Clinical APS	4 (22.2%)
Anti DNA	9 (50%)

Median (IQR)
Disease duration: years from diagnosis to BLM start
sDMARD: synthetic Disease Modifying Anti-Rheumatic Drugs
bDMARD: biologic Disease Modifying Anti-Rheumatic Drugs
APS: Antiphospholipid Syndrome

Abstract PS7:140 Table 2 One year follow up after BLM treatment

	T0	T3	T6	T12
N° of patients	18	16	14	7
Prednisone dose (mg/day)	6.9 (4.4-12.5)	7.5 (3.1-10.0)	5.0 (5.0-7.5)**	7.5 (1.5-10.0)
SLEDAI	10.0 (8.0-13.7)	4.5 (2.7-10.7)*	5.0 (2.0-10.2)**	5.0 (2.0-10.4)***
C3	73.5 (67.2-101.7)	92.5 (71.0-105.0)*	91.0 (72.7-110.0)**	89 (58.0-102.0)
C4	11.0 (7.75-19.75)	11.0 (8.0-21.2)	15.5 (10.0-24.7)**	11 (2.0-14.0)
Anti-DNA levels	11.7 (1.7-97.5)	17.0 (3.3-115.7)	31 (4.55-107)	29 (5.3-486.0)

All values are Median (IQR)
*p < 0.05 (T0-T3); **p < 0.05 (T0-T6); *** p < 0.05 (T0-T12)
T0: Baseline before BLM begin;
T3: 3 months after BLM;
T6: 6 months after BLM;
T12: 12 months after BLM.

At BLMin 5 patients had renal involvement; 3 of them improved after 3 months of treatment. Among 14 patients with joint involvement, 3 improved by month 3, 2 by month 6 and 1 by month 7 after treatment beginning. 3 patients had heart and lung disease; no one of them improved with BLM. Among 9 patients with skin disease only one showed improvement after 12 months of treatment. 9 patients had haematological disorders: 1 improved by month one, 1 by month 3, one by month 5 and 2 my month 6 of treatment.

Conclusion In this series BLM improved SLEDAI and complement levels allowing sparing of maintenance corticosteroid in SLE patients.

PS7:141 SAFETY AND TOLERABILITY OF OMALIZUMAB IN SUBJECTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS: PRELIMINARY OUTCOMES

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Background Studies performed in Lyn-/- mice indicate that IgE autoantibodies induce a lupus nephritis-like disease. In SLE, these autoantibodies are associated with increased disease activity. We propose that omalizumab, a monoclonal antibody that binds IgE, will reduce SLE activity by reducing circulating IgE autoantibodies, subsequently blocking basophil activation and immune dysregulation. The primary aim of this study was to determine the safety and tolerability of omalizumab in mild to moderate SLE.

Methods SLE participants (n=16) with SLEDAI >4 were randomised to receive omalizumab or placebo (2:1) added to their baseline SLE therapy for 16 weeks, followed by a 16 week open label and 4 week washout period. The regimen was a loading dose of omalizumab 600 mg dose followed by 300 mg every 4 weeks. Type I Interferon (IFN) gene signature was determined using a previously validated four gene interferon score (IFI27, IFI44, IFI44L, RSAD2) using quantitative PCR.

Demographics Variables n=16

Age (Years)
- Mean 43.4
- STD 13.2
- Range 22–69

Disease Duration (years)
- Mean 16
- STD 13.3
- Range 4–50

Race/Ethnicity, N (%)
- African American 3 (18.8%)
- Asian 2 (12.5%)
- Caucasian 4 (25.0%)
- Hispanic 7 (43.8%)

Sex, N (%)
- Female 16
- Male 0

Safety: There were a total of 52 adverse events, with the majority (49) being classified as mild. Three events met the criteria for SAE (primary varicella, pulmonary embolism, and bronchitis). No local or systemic allergic reactions were observed.

Clinical: There was a trend during the first 16 weeks of therapy toward reduction in the SLEDAI 2K score (p=0.077).

Biological: There was a trend in the reduction in the type I IFN signature in subjects treated in the first 16 weeks (p=0.11), especially in those subjects with high baseline IFN scores (p=0.052).

Conclusion The use of omalizumab in subjects with SLE appears to be well tolerated. There was a trend showing efficacy of omalizumab. The type I IFN signature also trended down, particularly in subjects with high signatures at baseline. The data analysis of other clinical parameters and mechanistic studies is still ongoing.