APS, pDC and mDC produced BAFF and expressed chemo-
kine receptors.

Conclusion pDC and mDC are differentially affected by IFNξ in SLE and APS. IFNξ primes pDC for enhanced IFNξ pro-
duction which potentiates T-cell activation by mDC, thereby sustaing the IFN signature in SLE and APS.

**PS5:92** CLARIFICATION OF THE ROLE OF DNASE 1 ON THE ONSET OF SYSTEMIC LUPUS ERYTHEMATOSUS IN A MURINE MODEL

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Systemic lupus erythematosus (SLE) is a prototypical autoim-
mune disease resulting in multi-organ damage and a high rate
of morbidity. Onset of SLE is characterised by dysregulated
activation of T and B lymphocytes and the production of
autoantibodies directed against nuclear components. The auto-
antibodies generated during the onset of SLE often recognise
components released by neutrophils during NETosis, a type of
cell death defined by the generation of neutrophil extracellular
traps (NETs). The endonuclease DNaseI has been shown to be
involved in the clearance of NET components. The sera of
SLE patients contain inhibitors of DNase 1 and/or anti-NET
antibodies that block the ability of DNase 1 to degrade NETs.
Thus, whilst NETs are important for clearing infection they
must be tightly regulated and degraded to prevent the onset of autoimmunity.

In this study we monitored the production of auto-antibod-
ies in the serum of wild type and DNase 1-deficient mice from
the age of 2 to 12 months, along with proteinuria levels and the development of glomerulonephritis. We show that
DNase 1-deficient mice develop a SLE-like phenotype with
elevated auto-antibody production and kidney damage by 12
months. This model also demonstrates the female bias in SLE
under study were excluded. The DuraClone IM panel (Beck-
man Coulter) with software analysis using Kaluza version
1.2: data obtained were expressed in relative% of PB lympho-
cytes and absolute values x10–6/L. Parallel analysis of serologi-
cal SLE biomarkers included C3, C4, ANA/IF (maximal titre),
ANA/ELISA, anti-dsDNA/IFCL (maximal titre), andi-dsDNA/ELISA and antinucleosome Abs. Data obtained were statisti-
cally processed using Medcalc-Statistical Software programme.

Results Significant differences (p<0.001) were obtained between absolute values of CD19 +CD27+IgM+B cells in
HC (median 31.36, 95% CI: 24.49 to 63.35) and SLE month
O’ (median 9.82, 95% CI: 6.01 to 14.26), and also SLE month
12’ (median 10.09 95% CI: 7.12 to 14.42), but not between values obtained in SLE month O’ and month 12’
(p>0.05); not significant differences were found in analysis
using relative% of PB lymphocytes (p>0.05). In SLE month
O’ was found a slight significant correlation between absolute
values of CD19 +CD27+IgM+B cells and anti-dsDNA/ELISA
Abs (rs=−0.28, p=0.034) without a confirmation in month
12’ control (rs=−0.09, p=0.491).

Conclusions The data obtained demonstrated persistent char-
acter of marginal-zone-like B cells deficiency in peripheral blood,
and are suggesting as possible biomarker of functional hypo-
plenism/asplenia in SLE.

**REFERENCE**


Acknowledgement Supported by the research project PRO-
GRES Q40–15.

**PS5:94** CHARACTERISATION OF SLE B CELLS FROM PATIENTS IN REMISSION – PERSISTENT IL-10 SECRETORY DEFECT

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Background SLE is an autoimmune disorder characterised by polyclonal Bcell activation, the production of anti-double stranded (ds) DNA autoantibodies and cytokines. Molecular
and clinical studies regarding SLE often address clinically
active patientsand not patients in remission. This study reports
on immunoglobulin, anti-dsDNA-aab and IL-10 secretory
capacity of cultures of CD19 +lymphocytes from SLE patients
in remission in comparison to normal donors. The aim was to
evaluate whether endogenous factors (BAFF, CD40, IL4),
exogenous factors (CpG-ODN-motifs, SAC) or their combina-
tions differentially influence immunoglobulin, cytokine and anti-dsDNA-aab production in not active SLE patients vs
healthy controls.

Methods Blood samples were obtained from a group of 13 SLE
patients attending clinics at the rheumatology unit at the Heinrich-

LUPUS 2018;5(Suppl 1):A1–A129