shown to enhance their inhibitory effect of CD4+ T cell pro-inflammatory function and replication. We have reported a decreased serum levels of sema3A in systemic lupus erythematosus (SLE) compared to healthy controls and in correlation with SLE disease activity [1]. Sema3A was found to be over expressed in podo-
cyes and epithelial cells in in animal models with diabetic nephropathy. Increased
urinary sema3A was also detected in diabetic patients with proteinuria and in con-
trast-induced acute renal injury [2-3].

Objectives: To assess urine sema3A secretion in SLE patients with and without
renal involvement compared to rheumatoid arthritis patients as disease control-
and healthy controls.

Methods: 50 ml of fresh urine samples were collected, centrifuged and the super-
natant was then concentrated up to 50 times the initial concentration and sub-
jected to specific human Sema3A ELISA kit (MBS5236222, San Diego, CA, USA).

Results: Thirty-eight lupus patients fulfilling the 2012 SLICC criteria were
recruited, 33 (87%) of whom were women, at a mean age of 35±12 years. Eight
patients had active nephritis (21%) and additional 5 had a history of nephritis but
were in remission. APLA was diagnosed in 13 (34%) of patients. Disease activity
was evaluated by the Systemic lupus erythematosus disease activity index 2000
(SLEDAI 2K) and was 8±7.7.

Sema3A was lower in lupus patients compared to rheumatoid arthritis and healthy
controls, 4.9±3.9 nm/g, 8.5±2.7 nm/g, 9.855±1.7 nm/g, p=0.0006. Lupus neph-
ritis patients demonstrated lower urine sema3A concentration compared to lupus
patients without renal involvement 4±3.4 nm/g, 6.5±3.8 nm/g, p=0.03. Sema 3A
reversely correlated with proteinuria r=-0.43 p=0.006 and SLEDIA2K -0.3, p=0.04,
which was higher in patients with nephritis (21%).

Conclusion: Urinary excretion of Sema 3A was found to be decreased in the
SLE patients with renal disease, reversely correlating with disease activity and
proteinuria. These findings are in line with previous reports of decreased serum
level of Sema3A in SLE, that may result in reduced efficacy of regulatory T cells,
driving autoimmunity and kidney damage. The discrepancy between low semea3A
urinary excretion in SLE nephropathy and increased urinary secretion in other
"non-auto immune" conditions with renal damage, suggests that sema3A in the
kidneys is protective in autoimmune diseases and detrimental in "non-auto
immune" conditions. This differential effect of semea3A may have to do with different
populations of effector cells and different expression of semea3A receptors (nuropi-
lin1). Further studies should evaluate semaphorin 3A role in lupus nephritis and its
potential as a treatment option.

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controls with diabetic proteinuria and mediates diabetic nephropathy and

predict contrast-induced acute kidney injury in patients undergoing percuta-

Disclosure of Interests: None declared

CROSSTALK BETWEEN SALIVARY GLAND EPITHELIAL CELLS AND B LYMPHOCYTES IN PRIMARY SJÖGREN’S SYNDROME

Elodie Rivière1,2, Julie Pascaud1, Nicolas Tchitchek1, Saida Boudaoud1, Audrey Padelleti1, Birna Ly1, Alice Thai1, Norm Allaire1, Bernd Jagla4, Elodie Rivière1,2, Juliette Pascaud1, Nicolas Tchitchek1, Saida Boudaoud1, Audrey Padelleti1, Birna Ly1, Alice Thai1, Norm Allaire1, Bernd Jagla4, Elodie Rivière1,2, Juliette Pascaud1, Nicolas Tchitchek1, Saida Boudaoud1, Audrey Padelleti1, Birna Ly1, Alice Thai1, Norm Allaire1, Bernd Jagla4

Background: Primary Sjögren’s syndrome (pSS) is an auto-immune disorder
characterized by lymphocytic infiltrates and destruction of the salivary glands.
Mechanisms leading to B lymphocytes chronic activation remain partially under-
stood and we assumed that salivary gland epithelial cells (SGECs) might play a
key role in B lymphocytes activation and differentiation.

Objectives: We aimed to study the interactions between SGECs from pSS
patients or controls and B lymphocytes.

Methods: Patients with pSS according to 2016 EULAR/ACR criteria and controls
with sicca symptoms were studied. RNASeq analysis was performed on SGECs
and B lymphocytes sorted from salivary gland and blood using a FACs ARIA.
Enrichment analysis was performed using Ingenuity Pathway Analysis software.
Validation of the results was performed by qPCR (Biomark technology).

Figure 1. A: Difference between the percentage of alive B lymphocytes co-cultured
with SGECs and the percentage of alive B lymphocytes cultured alone at day 5. B: Difference
between the percentage of CD38+ B lymphocytes co-cultured with SGECs and the
percentage of CD38+ B lymphocytes cultured alone at day 5. Green for SGECs from
controls, in orange for SGECs from pSS patients. One asterisk indicates p-value <0.05, two
asterisks indicate p value <0.01.

Conclusion: Epithelial cells from patients with pSS have better ability than con-
trols to stimulate survival and activation of B cells. According to preliminary
results, this effect could be mediated at least partially by soluble factors. The path-
ways responsible for this stimulation are being determined by RNASeq analysis
of purified epithelial cells and B lymphocytes and inhibitory experiments that can rep-
resent new therapeutic perspectives are ongoing.

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Endothelin-1 level in patients with antiphospholipid syndrome, its association with endothelial dysfunction and atherosclerosis

Serai Shevchuk1, Inna Kuvkova1, Yulia Sheheda1, Olena Galusina2, National Pirogov Memorial Medical University, Vinitsya, Ukraine;2Scientific and Research
Institute of Invalid Rehabilitation on the base of National Pirogov Memorial Medical
University, rheumatology, Vinitsya, Ukraine;3Scientific and Research Institute of
Invalid Rehabilitation on the base of National Pirogov Memorial Medical University,
rheumatology, Vinitsya, Ukraine

Background: It is known that endothelin-1 is one of the leading factors in the
development of coronary artery disease, acute myocardial infarction, atheroscle-
rosis of cerebral and peripheral vessels, pulmonary hypertension, ischemic lesion

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