B-cell subsets	1 group (n=27)		2 group (n=26)		Control group (n=29)	
	Cells, 10 ⁹ /n	Percentage, %	Cells, 10 ⁹ /n	Percentage, %	Cells, 10 ⁹ /n	Percentage, %
B-cell [CD19+]	0,144 [0,1; 0,21]	9,1 [7,5-10,9]	0,105 [0,1-0,2] **	8,35 [6-10,2]	0,2 [0,1-0,2]	8,5 [7,2-11,0]
B-memory cell [CD19+CD27+]	0,003 [0,00166; 0,0044]	2,1 [1,6-3,1]	0,0015 [0,001-0,003] *	1,25 [0,9-1,7] *,**	0,003 [0,001-0,007]	2,2 [1,1-3,0]
Switched B-cells [CD19+CD27+IgD-]	0,0187 [0,0133-0,0289]	16 [9,3-18,4]	0,01 [0,005-0,02]	6,8 [3,6-11,6] *,**	0,02 [0,01-0,04]	12,8 [9,3-17,0]
Non-switched B-cells [CD19+CD27+IgD+]	0,0073 [0,00619-0,0122]	5,9 [3,6-9,7]	0,009 [0,006-0,01]	7,45 [5,1-11,4]**	0,01 [0,005-0,02]	7,4 [3,7-11,1]
Double negative D-cells [CD19+CD27-IgD-]	0,021 [0,011-0,028]	14 [9,6-19,5]	0,02 [0,01-0,03]	15,05 [11,9-18,1]	0,02 [0,01-0,02]	13,3 [7,1-19,3]
Naive B-cells [CD19+CD27-IgD+]	0,076 [0,063-0,13]	61,6 [52,9-68,8]	0,095 [0,07-0,1]	70,85 [62,5-75,6] **	0,1 [0,06-0,1]	64,7 [57,6-72,4]
Transitioning B-cells [CD19+CD38+++CD10+IgD+CD27-]	0,000424 [0,000162-0,000624]*,**	0,2 [0,1-0,4]	0 [0-0,0001]	0 [0-0,1]	0,0001 [0-0,0003]	0,1 [0-0,1]
Plasmoblasts [CD19+CD38+++CD27+IgD-CD20-]	0,00071 [0,00023-0,00129]*,**	0,4 [0,3-0,8]*, **	0,0003 [0,00007-0,0004]	0,15 [0,1-0,3]	0,0002 [0,0001-0,0004]	0,1 [0,1-0,2]
Plasmocytes [CD19+CD38+]	0,000262 [0,000106-0,000414]*, **	0,1 [0,1-0,3]	0,0001 [0-0,0002]	0,1 [0-0,1]	0,0001 [0,00-0,0002]	0,1 [0,05-0,1]

Data presented as median and interquartile range. * - differences to control group were observed, p<0,05. ** - differences between pts. groups were observed, p<0,05. No significant difference between T-cell subsets was found. In comparison with healthy individuals, pts. with early RA had increased levels of switched B-memory cells, transitioning B-cells and plasmoblasts, the opposite was observed in progressed RA pts with addition of decrease in B-memory cells.

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AB0069

ANTIBODIES AGAINST PHOSPHORYLCHOLINE AS PROTECTION MARKERS IN AUTOIMMUNITY, ATHEROSCLEROSIS, CARDIOVASCULAR DISEASE AND CHRONIC INFLAMMATION – IMPLICATIONS FOR TREATMENT

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Background: Autoimmunity, atherosclerosis and cardiovascular disease (CVD) are chronic inflammatory conditions and represent a major part of global burden of morbidity and also mortality. The immune system plays an important role in these conditions. Further, the risk of CVD and atherosclerosis is raised in several autoimmune conditions, where SLE is an important example. Its major cause, atherosclerosis (where activated immune competent cells producing cytokines are abundant), for example, active and passive immunization can influence the degree of atherosclerosis in animal experiments, but no treatment developed to specifically ameliorate inflammation is presently available. The cause of the chronic inflammation and immune activation in atherosclerosis has not been fully defined, and there are different, non-mutually exclusive and potentially synergistic possibilities including oxidized LDL (oxLDL) heat shock proteins (HSP) and infections. We focused on the lipid moiety in oxLDL, especially phosphorylcholine (PC) which is a small hapten, immunogenic when exposed and reported that oxLDL's immunological effects is to some extent caused by PC. Antibodies against PC (anti-PC) are abundant, about 5-10% of circulating IgM is IgM anti-PC.

This is thus a theranostic concept, combining novel risk markers with possible underlying mechanisms and potentially immunization to raise anti-PC.

Objectives: We reported that anti-PC, especially IgM (and IgG) anti-PC is negatively associated with atherosclerosis progress, CVD (including both MI and stroke) in different populations, and also with autoimmune diseases, especially systemic rheumatic diseases as SLE. Potential mechanisms include anti-inflammatory effects, inhibition of uptake of oxLDL in the vascular wall, promotion of T regulatory cells, inhibition of cell death, and increased clearance of dead cells.

Methods: We have used a combination of experimental and translational/clinical studies.

Results: Based on our findings, we have proposed a novel hypothesis which is a development of the hygiene/old friends-hypothesis, namely that low exposure to microorganisms as nematodes, parasites and some bacteria which from an evolutionary point of view has been with humanity for millions of years, leads to low levels of the antibodies described and a lack of ability to prevent autoimmune

conditions. Indeed, individuals from Kitava, New Guinea leading a traditional life as hunter-gatherers and horticulturalists appear not to have CVD or rheumatic disease, but strikingly high anti-PC levels.

Conclusion: anti-PC could be a novel factor protecting against rheumatic disease.

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Spondyloarthritis - aetiology, pathogenesis and animal models

AB0070

ROLE OF VASPIN IN ATHEROSCLEROTIC DISEASE AND CARDIOVASCULAR RISK IN AXIAL SPONDYLOARTHRITIS

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