FMF patients without Amyloidosis diagnosis (FMF(+) A (-)), and healthy controls (HC). The mean ages, TREM-1, C - reactive protein (CRP), and Creatinine levels of each group are shown in Table 1. TREM-1 levels were found to be significantly higher in A(+) FMF(+) group than FMF(+) A (-), and healthy control groups (p= 0.001 and 0.002). Nevertheless, this difference was not found in between A(+) FMF(+) and FMF(-) A(+) (p= 0.447). In addition, the TREM-1 levels of FMF(+) A (-), and healthy control groups were not different (0.532). In A(+) FMF(+) group, 36 patients used colchicine with the mean dose of 1.9±0.8 mg/day, 14 patients used anakinra, and 9 patients used canakinumab. In FMF(+) A (-) group all 20 patients used colchicine with the mean dose of 2.8±0.9 mg/day, 1 patient used anakinra, and 2 patients used canakinumab.

Table 1. Clinical Features of Patients and TREM-1 levels

	A(+) FMF(+) (n= 42)	FMF(-) A(+) (n=5)	FMF(+) A (-) (n=20)	HC (n=20)
Age	43.9±12.9	54.8±19	35.3±9.64	35.4±6.57
TREM-1	735.3±566.5	1247.1±1349.2	414.3±142.3	439.2±104.6
CRP	11.1±14.2	51.3±98.3	25.8±54	1.8±1.7
Creatinine	1.6±1.8	3.28±4.17	0.7±0.15	0.7±0.15

Conclusion: In conclusion, TREM-1 is a proinflammatory marker found significantly high in AA-amyloidosis patients regardless of their FMF diagnosis. TREM-1 may be useful in AA-amyloidosis follow-up and early diagnosis since currently there is a deficit of an early diagnostic marker of amyloidosis. This study is a cross-sectional one so it is hard to reach a conclusion on the effectiveness of TREM-1 during regular FMF follow-up for the secondary prevention of amyloidosis. However, the sensitivity of TREM-1 as a marker cannot be denied in amyloidosis.

Disclosure of Interests: None declared

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AB0056 NATURAL ANTIBODIES AGAINST PHOSPHORYLCHOLINE AND MALONDIALDEHYDE DURING THE FIRST TWO YEARS OF LIFE: IMPLICATIONS FOR RHEUMATIC DISEASE

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Background: Antibodies against phosphorylcholine (anti-PC) have potentially protective properties in both atherosclerosis and rheumatic disease. IgM anti-PC could play a role in SLE being associated with protection, also in relation to atherosclerotic plaques and vulnerable plaques in SLE[1] and being a non-responder to biologics in RA. [1] We reported potential mechanisms by which anti-PC could be protective: 1:anti-inflammatory; 2: inhibits uptake of oxLDL in macrophages, 3: inhibits cell death.[1] 4: anti-PC (and anti-MDA) increases clearance of human dead cells which could be of importance not especially in SLE;[2] 5: anti-PC increases T regulatory cells in SLE-patients´ T cells from a low level and also in atherosclerosis, with implications for both conditions.[3] Also antibodies against malondialdehyde (anti-MDA) have interesting properties

Objectives: It is not known how these antibodies develop early in life and what may cause low levels. The objective is to determine this.

Methods: Antibodies were studied by ELISA in healthy pregnant women (n=105; Born into life study) and their newborn children. Women were recruited before conception. Informed consent, questionnaires from parents and plasma sample was collected from children at birth from cord blood, at 1-year and 2 years after birth. Extracted antibodies were compared using a proteomics de novo sequencing approach.

Results: Children were born with very low levels of IgM anti-PC, while IgM anti-MDA was present at birth,. Both IgM anti-PC and anti-MDA increased during the first two years of life, but IgM anti-PC in contrast to IgM anti-MDA was still significantly lower than mothers´. IgG anti-PC decreased after 1 year, but reached similar levels as mothers´ after 2 years while IgG anti-MDA reached similar levels as mothers´ already after one year. Proteomics peptide sequencing analysis indicates large peptide sequence variation without specific clone expression during early stage of life compared to the adult stage for which specific peptide sequences dominated.

Conclusion: IgM anti-PC levels develop much slower than anti-MDA and are still relatively low at 2 years. We hypothesize that anti-PC is developed by a combination of pre-programming and exposure to the external world, where infectious agents may play a role. For anti-MDA pre-programming is likely to play a major role and at an earlier stage than for anti-PC.

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AB0057 THE ROLE OF NON-INFLAMMATORY MACROPHAGES INITIATING AND RESOLVING THE INFLAMMATORY RESPONSE OF MONOSODIUM URATE IN MICE

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Background: Gout is the most common chronic inflammatory arthritis around the world which is associated with many conditions that affect longevity and well-being, such as metabolic syndrome, cardiovascular diseases, and renal diseases. [1]

Objectives: Gout is the most common inflammatory arthritis around the world. Innate immunity has been implicated in gout inflammation in recent years. However, the phenomena of maintenance of 'asymptomatic' hyperuricemia, the spontaneous resolution of acute gouty arthritis and non-inflammatory tophus and its association with immune regulation are still elusive. We propose that resident macrophages may be a key player in the suppression of gouty inflammation. To address this hypothesis, we used non-inflammatory macrophages to explore the role played in initiating or resolving the inflammatory response.

Methods: Bone marrow-derived macrophages (BMDM) were stimulated with monosodium rate (MSU), then analyzed the expression of RNA and protein of inflammatory cytokines, including IL-1β, IL-18 and TNF-α. In addition, we also observe the ability of macrophage to phagocyte and hydrolyze MSU crystal.

Results: Our results indicate that MSU alone could not induce IL-1β, IL-18, and TNF-α mRNA expression and protein production. However, when macrophages were pre-stimulated with lipopolysaccharide (LPS) or MSU, as well as in combination the production of IL-1β and IL-18 were significantly increased. Furthermore, MSU maybe amplifies LPS-induced protein production of IL-1β and IL-18 but not in TNF-α. A temporal delay in the correlation between mRNA expression and protein production was shown. The results also indicated that macrophages could not only phagocytize MSU crystals but may also hydrolyze MSU crystals.

Conclusion: These data indicate that MSU crystal alone is insufficient to induce pro-inflammatory cytokines production, however, when exposed to an infectious source of infection, it will amplify the inflammatory response and there is a synergistic effect between MSU and LPS. This may explain the diverse clinic phenomena of 'asymptomatic' hyperuricemia, non-inflammatory tophus and acute gouty arthritis. The high efficiency of phagocytosis and hydrolyzed MSU crystals of macrophages may explain the spontaneous regression of acute gouty arthritis. These findings may provide a new therapy for the prevention and treatment of acute gout attacks.

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