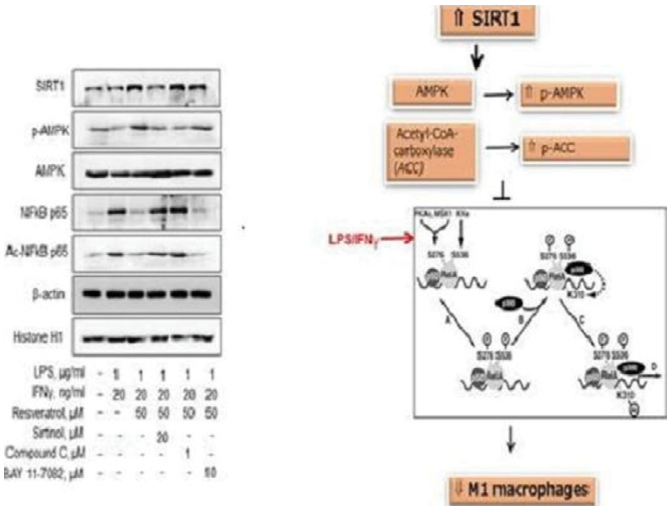

Disclosure of Interest: None declared
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AB0108 DECREASED EXPRESSION OF PTPN22 GENE IN PATIENTS WITH RHEUMATOID ARTHRITIS CARRYING THE RISK ALLELE OF PTPN22 RS2488457 POLYMORPHISM

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Background: Mutations in the protein tyrosine phosphatase non-receptor 22 (*PTPN22*) gene are associated with numerous connective tissue and autoimmune diseases [1]. In particular, *PTPN22* has been recognized as the main non-HLA genetic risk factor involved in rheumatoid arthritis (RA) susceptibility [2]. Moreover, it has been suggested that *PTPN22* modulation may influence on inflammatory processes associated with RA [3,4].
Objectives: To determine if *PTPN22* (rs2476601, rs33996649 and rs2488457) polymorphisms, associated with RA, may influence on *PTPN22* expression in RA patients compared to healthy controls. Moreover, the association between *PTPN22* expression in patients with RA and their clinical characteristics was studied.
Methods: *PTPN22* messenger RNA (mRNA) expression was quantified by quantitative real-time PCR in peripheral blood samples from 42 RA patients and 24 healthy controls. *PTPN22* rs2476601 (G>A), *PTPN22* rs3399649 (C>T), and *PTPN22* rs2488457 (C>G) single-nucleotide polymorphisms (SNP) were genotyped by TaqMan SNP genotyping assays. Differences in *PTPN22* expression between patients and controls were analyzed by Student's t test, according to their genotype. Correlation coefficients were also assessed between *PTPN22* expression in RA patients and their clinical characteristics.
Results: A significant down-regulation of *PTPN22* expression in patients with RA carrying *PTPN22* rs2488457 risk allele (G) compared to controls was observed (relative mean values of *PTPN22* mRNA levels ± standard deviation: 2.93±0.76 vs 4.33±0.63, p=0.0004). Furthermore, an inverse relationship between *PTPN22* expression and disease duration (r=-0.38, p=0.03) was found. These results were adjusted by sex, age at time of study and cardiovascular risk factors.
Conclusions: Our study shows for the first time that the risk allele of *PTPN22* rs2488457 polymorphism influences on the down-regulation of *PTPN22* in patients with RA. This result suggests a transcriptional suppression of *PTPN22* gene in RA, which in turn may play an important role in disease diagnosis and progression.
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AB0109 AUTOPHAGY INHIBITOR REGULATES APOPTOSIS AND PROLIFERATION OF SYNOVIAL FIBROBLASTS THROUGH THE INHIBITION OF PI3K/AKT PATHWAY IN COLLAGEN-INDUCED ARTHRITIS RAT MODEL

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Background: Mounting studies have illustrated an important role of autophagy in various diseases, but few studies have reported its contribution to rheumatoid arthritis (RA) and the underlying mechanism is largely unknown.
Objectives: This study aimed to investigate whether autophagy inhibitors could regulate apoptosis and proliferation through PI3K/AKT pathway in RA.
Methods: RA animal model was established by collagen induction. General observations and degree of joint swelling were observed. Inflammatory response, cell survival related factors and apoptosis were also detected in synovial fibroblasts. In addition, cultured rheumatoid arthritis fibroblast-like synoviocytes (RA-FLS) were subjected to TNF-α treatment *in vitro*, and TNF-α induced cell autophagy, synovial cell proliferation and apoptosis were detected. Moreover, cell cycle and cytokine secretion protein, along with the above parameters, were analyzed.
Results: Results from the animal model showed that autophagy inhibitors could attenuate inflammatory reaction and synovial hyperplasia, while promoted synovial fibroblasts apoptosis. Meanwhile, inhibition of autophagy promoted cell apoptosis and reversed cell proliferation *in vitro*, also blocked cell in the G2/M arrest and reduced the S phase cells. Furthermore, inhibition of PI3K/AKT pathway reversed TNF-α mediated autophagy and cytokine secretion.
Conclusions: autophagy inhibitors could mitigate inflammation response, inhibiting RA-FLS cell proliferation while promoting cell apoptosis by PI3K/AKT pathway.
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AB0110 EVALUATION OF THE EFFECT OF CHUANTENGTONGBI DECOCTION ON DBA / 1 MICE CIA MODEL

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Background: DBA/1 mouse ($H2^q$ type) CIA model as a mature model of rheumatoid arthritis is widely used in pharmacology and pharmacodynamics research[1]. Chinese medicine treatment of rheumatoid arthritis has accumulated rich experience. The study of Tripterygium glycosides is quite representative, and it has been widely used in the past 30 years. Some Clinical studies showed that tripterygium glycosides treatment of rheumatoid arthritis had good effect[2]. ChuanTengTongBi decoction is also the effective prescription commonly used for the treatment of rheumatoid arthritis.
Objectives: To investigate the pathological damage degree of DBA/1 mouse CIA model and the effects of different doses of ChuanTengTongBi decoction on the CIA model mice.
Methods: The mice were divided into normal group, model group, leflunomide group (3.11mg/kg/d), low-dose of ChuanTengTongBi group (0.44g/ml/d), medium-dose group (0.88g/ml/d) and high-dose group (1.76g/ml/d).
Results: The arthritis index (AI) was evaluated every week to determine whether the model was successful. We defined AI score ≥4 as successful model (AI score