Results: In non-RA patients (n=151), PISA was correlated with total IgG and IgA in GCF ($p<0.0001$). IgA RF and IgA ACPA were present in GCF and correlated with total IgA in GCF ($p<0.05$ and $p<0.01$ respectively). In contrast to RA patients (n=72), IgA RF and IgA ACPA in GCF of non-RA patients did not correlate with IgA RF and IgA ACPA in serum. In non-RA patients, IgA ACPA positivity in GCF was more frequent in ever smokers (18%) than in never smokers (9.8%); the same held for presence or absence of periodontitis (18% and 9.3% IgA ACPA positivity, respectively). In non-RA patients PISA was correlated with IgA ACPA in serum ($p<0.01$) and GCF ($p=0.05$) (figure 1).

Conclusions: RA-associated autoantibodies are present in GCF of patients with and without RA. In contrast to RA patients, in whom this presence is probably due to leakage from serum, presence of RA-associated autoantibodies in GCF of patients without RA is presumably the result of local formation of these autoantibodies due to periodontitis.

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Disclosure of Interest: None declared

DOI: 10.1136/annrheumdis-2018-eular.3920

AB0120

ADIPONECTIN COULD BE A MEDIATOR OF THE PRESENCE OF *P. GINGIVALIS* IN PATIENTS WITH EARLY RHEUMATOID ARTHRITIS

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Background: Rheumatoid arthritis (RA) is a systemic chronic inflammatory disease characterised by joint destruction, deformity, lower functional status and decrease in life expectancy. The *Porphyromonas gingivalis* (*P. gingivalis*) has been reported to be significantly associated with RA. The adipokines are suggested to be a common link between periodontitis and RA.

Objectives: To determine the association between serum adipokines levels and presence of *P. gingivalis* in patients with early rheumatoid arthritis (eRA) compared to healthy individuals

Methods: A cross sectional study was conducted. Patients with the diagnosis of (eRA) according to the ACR/EULAR 2010 criteria were studied in Bogota-Colombia. A complete medical history related to RA was obtained. Adiponectin levels measured by Luminex technology (MILLIPLEXMAP), IL6 by chemiluminescence (Immulate 1000, Siemens) and leptin quantification by ELISA (DiaSource,), high-sensitivity CRP (hs-CRP) (Immulate 1000, Siemens) and ESR (Test 1 THL Ali FAX), and the detection of periodontopathic bacteria was carried out by qPCR from subgingival plaque samples. Cancer, autoimmune disease, infection, antibiotic use, diabetes, orthodontics treatment were exclusion criteria. An association analysis was made to evaluate the relationship between adipokines levels and periodontal, rheumatologic conditions using X2 test, Mann Whitney test and logistic regression model was performed to confirm this associations. All the results were performed with a level of significance of 95%

Results: A total of 51 patients with eRA and 51 healthy individuals were matched by age and gender. The mean age in RA patients was 48.54±10.93 years, 80.39% were female, between RA patients, 17.64% were in high activity and 27.45% in remission by DAS 28-ESR. 45.1% had APCA>20 UE. 37.25% were overweight and 13.72% were obese. 82.35% of patients had treatment, of which 80.39% received treatment with conventional disease modifying therapy, being methotrexate the most frequent one in 86.8%. In turn, 64.7% and 51% had high leptin and adiponectin levels respectively. An association of high adiponectin levels was found with Body Mass Index-BMI <25 ($p=0.017$). The presence of *P. gingivalis* in RA patients was 78.4% and in healthy individuals was 45.09% $p=0.001$. The adiponectin levels>47755 pg/ml were found associated with the absence of *P. gingivalis* (OR=0.078 95% CI 0.01–0.62) adjusted an ever smoking history of cigarette smoking, Body Mass Index>30 and high level of leptin and IL6.

Conclusions: The adiponectin plays an anti-inflammatory role in the pathophysiology of several chronic inflammatory diseases and it has been reported to inhibit LPS-induced NFκB nuclear translocation and to affect other LPS-triggered pathways. To promote the weight control in eRA could be to prevent the oral infection

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Disclosure of Interest: None declared

DOI: 10.1136/annrheumdis-2018-eular.3169

AB0121

RESVERATROL-ENHANCED AUTOPHAGIC FLUX REDUCES SEVERITY OF EXPERIMENTAL RHEUMATOID ARTHRITIS

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Background: Previously, we have demonstrated that dietary supplementation with resveratrol (RSV) lowers synovial hyperplasia, chemokines and oxidative damage in an acute antigen-induced arthritis (AIA) model developed in rats, and that autophagic cell death and limiting angiogenic response were involved¹.

Objectives: Here, we investigated the autophagic flux induced by RSV and its relation with serum interleukin (IL)-1β levels, since autophagy could regulate IL-1β via inflammasome. Moreover, C-reactive protein (CRP) and prostaglandin E₂ (PGE₂), which are also closely linked to inflammation and correlated with disease activity, were also evaluated.

Methods: Animals were randomly divided into 3 groups: healthy, AIA, and RSV-treated AIA group. RSV (12.5 mg/kg/day) was given orally 8 weeks before AIA induction until sacrifice day (48 hour after intra-articular injection). Healthy and AIA animals were administered 100 μl of water. RSV effects on autophagy were evaluated by the expression of the autophagy proteins, microtubule-associated protein 1 light chain 3 (LC3), and Beclin1 (an autophagic-related protein which up-regulation is associated with increased autophagosome formation), and p62 (an autophagic adaptor protein whose accumulation is associated with autophagic degradation dysfunction), by confocal and immunohistochemistry, respectively. Specific enzyme-linked immunosorbent assay kits and cytokine magnetic milliplex were used to measure CRP and PGE₂ and IL-1β in serum samples, respectively.

Results: At the end of the study, RSV significantly reduced the serum levels of IL-1β, CRP and PGE₂ ($p<0.01$ for IL-1β and PCR, and $p<0.05$ for PGE₂). In relation to autophagy proteins, results showed that the expression of LC3 was greater in the AIA synovial membranes than in control synovial samples, in which the presence of vesicles was easily observed. Interestingly, the synovial tissues from the RSV group showed a significantly ($p<0.001$) higher signal for LC3, compared with the AIA samples. As compared with healthy controls, AIA synovial membranes showed increased p62 expression, which was markedly inhibited by RSV treatment ($p<0.01$). The protein level of Beclin 1 was higher in AIA synovial membranes than in healthy samples; however, RSV did not increase the signal of Beclin 1. Moreover, there was a significant correlation between p62 expression and serum IL-1β levels ($p<0.05$ and $r=0.617$), PGE₂ ($p<0.01$ and $r=0.604$), and PCR ($p<0.001$ and $r=0.692$).

Conclusions: These data suggest that resveratrol is capable of inducing the non-canonical (Beclin1 independent) autophagy pathway, and in consequence, modulates the cross-talk existence with inflammation in an acute AIA model, which could also ameliorate the severity of rheumatoid arthritis.

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Disclosure of Interest: None declared

DOI: 10.1136/annrheumdis-2018-eular.4468