as part of a treat-to-target strategy in early rheumatoid arthritis: impact of joint area, repeated injections, MRI findings, anti-CCP, IgM-RF and CRP. *Annals of the rheumatic diseases* 2012, 71(6):851-856.

Acknowledgement: This research was supported by grants from the National Research Foundation of Korea (NRF) funded by the Ministry of Science and ICT (NRF-2018R1D1A1B07045491)

Disclosure of Interests: None declared

DOI: 10.1136/annrheumdis-2019-eular.951

THU0085 RHEUMATOID ARTHRITIS AND ALLERGIC DISEASES

Vanessa Kronzer¹, Cynthia S. Crowson¹, Jeffrey Sparks², Robert Vassallo¹, John Davis¹. ¹Mayo Clinic, Rochester, United States of America; ²Brigham and Women's, Boston, United States of America

Background: Historically, RA was considered a T_H1 disease while asthma and allergy were considered T_H2 diseases (1). However, several studies show an association between RA and asthma (2), and more recently, allergic disease (3).

Objectives: We first aimed to determine the association between RA and asthma after controlling for important confounders including allergic disease, urban environment, and passive smoke exposure. Second, we aimed to determine the association between RA and various allergy types.

Methods: This case-control study identified 1,023 cases of RA (175 incident) within a single-center biobank population using a rules-based algorithm that combined self-report with two diagnosis codes. Exposures were self-reported on biobank questionnaires. Logistic regression models calculated the association of exposures with RA, adjusting for potential confounders.

Results: Asthma was associated with RA after adjusting for allergies, urban environment, and passive smoke exposure (OR 1.28, 95% CI 1.04 to 1.58). History of allergic disease was also associated with RA (OR 1.30, 95% CI 1.12 to 1.51), especially food allergy (OR 1.38, OR 1.08 to 1.75, see Figure 1). Results from the incident RA cohort mirrored these findings, though the association with asthma was not significant (OR 1.17, 95% CI 0.66 to 2.06).

Abstract THU0085 – Figure 1. Adjusted association between allergy types and RA.

Conclusion: Asthma and allergies may be associated with increased risk of RA. It is possible a broader problem with immune dysregulation underlies both RA and allergic diseases, and having one predisposes to the other.

REFERENCES:

- [1] Singh VK, Mehrotra S, Agarwal SS. The paradigm of Th1 and Th2 cytokines: its relevance to autoimmunity and allergy. *Immunol Res*. 1999;20(2):147-61.
- [2] Sheen YH, Rolfes MC, Wi CI, Crowson CS, Pendegraft RS, King KS, et al. Association of Asthma with Rheumatoid Arthritis: A Population-Based Case-Control Study. *J Allergy Clin Immunol Pract*. 2018;6(1):219-26.
- [3] Lai NS, Tsai TY, Koo M, Lu MC. Association of rheumatoid arthritis with allergic diseases: A nationwide population-based cohort study. *Allergy Asthma Proc*. 2015;36(5):99-103.

Acknowledgement: Funding for this project was provided by the Rheumatology Research Foundation Resident Research Preceptorship. We would also like to acknowledge the Mayo Clinic Center for Individualized Medicine

Disclosure of Interests: Vanessa Kronzer: None declared, Cynthia S. Crowson: None declared, Jeffrey Sparks Grant/research support from: Bristol-Myers Squibb, Amgen, Consultant for: Optum, Robert Vassallo: None declared, John Davis Grant/research support from: Pfizer

DOI: 10.1136/annrheumdis-2019-eular.4858

THU0086 PREDICTING TNFALPHA INHIBITOR TREATMENT RESPONSE USING SERUM CYTOKINES IN PATIENTS WITH RHEUMATOID ARTHRITIS

Marissa Lassere¹, Sue Baker¹, Jenny Gu². ¹UNSW Sydney, St George Hospital SESLHD, Sydney, Australia; ²UNSW Sydney, Sydney, Australia

Background: Tumour necrosis factor- α inhibitors (TNF α) are the main biologics (b-MARDs) used to treat active rheumatoid arthritis (RA) in patients that have failed disease modifying treatment (DMARD). However, 10% of patients with rheumatoid arthritis, TNF α inhibitors do not work at all. Patients are continued on this treatment for several months risking side-effects in the hope that the treatment will work. Another 40% of patients respond partially to this treatment and have to also be treated with other drugs such as methotrexate and prednisone in addition to treatment with TNF α . Biomarkers offer an opportunity to identify before starting or soon after starting treatment with TNF α which patients will be responders and whether prednisone and other drugs can be reduced and optimise the risk-benefit of treatment.

Objectives: We undertook a series of experiments with the following objectives: determine whether cytokine biomarkers will predict which patients with rheumatoid arthritis are: (a) sustained DMARD early treatment responders (b) sustained TNF α early treatment responders, (c) TNF α early treatment failures.

Methods: We used the Millipore's MILLIPLEX MAP Human Th17 Magnetic Bead kit for the simultaneous quantification of the following cytokines: IL-1 β , IL-2, IL-4, IL-5, IL-6, IL-9, IL-10, IL-12p70, IL-13, IL-15, IL-17A, IL-17E/IL-25, IL-17F, IL-21, IL-22, IL-23, IL-27, IL-28A, IL-31, IL-33, GM-CSF, IFN γ , MIP-3 α , TNF α and TNF β . γ , MIP-3 α , TNF α and TNF β . We evaluated 14 patients with RA starting on a DMARD and 26 patients with RA starting on a TNF α after failing DMARDs. These cytokines were assayed monthly 2 or 3 months prior to starting a TNF α to evaluate month-to-month cytokine variability and every month up to 5 months after initiation of treatment. RA disease activity was measured using the RA Disease Activity Score (DASCRP28) which includes joint counts, CRP and a patient-reported outcome of health status. All samples were blocked with Heteroblock to reduce rheumatoid factor and other heterophilic antibodies. Rheumatoid factor was measured before and after blocking. The same negative and positive controls were included across all plate runs. All assays were done in singlet to accommodate longitudinal samples. Mixing studies were undertaken to evaluate whether cytokine results could be analysed using quantitative statistics.

Results: We had 67 serum samples in the DMARD treated group and 202 serum samples in the TNF α treated group because of the longitudinal study design. Using mixed effects linear regression to account for longitudinal data in a model that included all 25 cytokines, treatment-time and treatment type (DMARD or TNF α +/DMARD), we found that in patients on DMARDs, IL-6, IL-1 β , IL-28A, and TNF β were associated with treatment response. However, in TNF α treated patients, TNF- α , GM-CSF and IL-6 were associated with treatment response. Only p values <0.005 are reported given that 25 cytokines were included in the model.

Conclusion: In this study of treatment response comparing DMARDs and TNF α in a longitudinal cohort of 26 patients with a total of 202 samples measuring TNF α and GM-CSF may predict early TNF α responders.

Disclosure of Interests: Marissa Lassere Grant/research support from: I have received research support with educational grant funding from Sanofi-Aventis, Pfizer, MSD, Abbvie, Sue Baker: None declared, Jenny Gu: None declared

DOI: 10.1136/annrheumdis-2019-eular.6445

THU0087 UTILITY OF INFRARED THERMOGRAPHY FOR THE EVALUATION OF RHEUMATOID ARTHRITIS

Jesús Loarce-Martos¹, Javier Bachiller-Corral¹, Ismael Fernández Cuevas², Manuel Sillero Quintana³, Mónica Vázquez Díaz¹. ¹HU Ramón y Cajal, Madrid, Spain; ²ThermoHuman, Madrid, Spain; ³Universidad Politécnica de Madrid, Madrid, Spain

Background: Rheumatoid Arthritis (RA) is a chronic inflammatory disease, which predominantly affects the hands. Currently in clinical practice, we explore the number of swollen and painful joints in order to evaluate the activity of the disease. Infrared thermography (IT) is a non-invasive, lacking ionizing radiation, operator-independent and low-cost technique that allows to measure the temperature of the cutaneous surface through the taking of a photograph.

Objectives: To determine the utility of IT in the evaluation of hands arthritis in patients with RA.