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THU0085 RHEUMATOID ARTHRITIS AND ALLERGIC DISEASES

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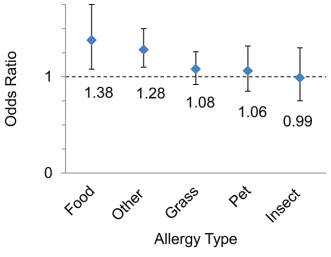
Background: Historically, RA was considered a T_H1 disease while asthma and allergy were considered T_H2 diseases (1). However, several studies show an association between RA and asthma (2), and more recently, allergic disease (3).
Objectives: We first aimed to determine the association between RA and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

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

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Background: Tumour necrosis factor-αlpha inhibitors (TNFαi) 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, TNFα 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 TNFαi. Biomarkers offer an opportunity to identify before starting or soon after starting treatment with TNFαi 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 TNFαi early treatment responders, (c) TNFαi 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-1β, 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, GMCSF, IFNγ, MIP-3α, TNFα and TNFβ. γ, MIP-3α, TNFα and TNFβ. We evaluated 14 patients with RA starting on a DMARD and 26 patients with RA starting on a TNFαi after failing DMARDs. These cytokines were assayed monthly 2 or 3 months prior to starting a TNFαi 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 TNFαi 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 TNFαi+/-DMARD), we found that in patients on DMARDs, IL-6, IL-1β, IL-28A, and TNFβ were associated with treatment response. However, in TNFαi treated patients, TNF-a, 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 TNFαi in a longitudinal cohort of 26 patients with a total of 202 samples measuring TNFα and GM-CSF may predict early TNFαi responders.
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THU0087 UTILITY OF INFRARED THERMOGRAPHY FOR THE EVALUATION OF RHEUMATOID ARTHRITIS

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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.