ANOSIM). Detailly, at the genera level, IIM patients had a higher abundance of Enterococcus, Veillonella, Streptococcus, et al. and a lower abundance of Roseburia, Lachnospira, Klebsiella, et al(Figure 1D, $p < 0.05$). In IIM patients, Fusobacteriota correlated positively with the ratio of Th1 cells (Figure 1E, $p < 0.01$), and there was a significant positive correlation between Synergistota and B lymphocyte (Figure 1E, $p < 0.01$). Besides, Euryarchaeota and Cyanobacteria were both positively and significantly related to IL-6, IFN-γ and C-reactive protein (CRP) (Figure 1E, $p < 0.001$).

Conclusion: Richness and diversity of intestinal flora in IIM patients were impaired, which might participate in the pathogenesis of IIM by disturbing lymphocyte subpopulations and cytokines. Regulating intestinal flora and restoring homeostasis might become a critical therapeutic methods of IIM.

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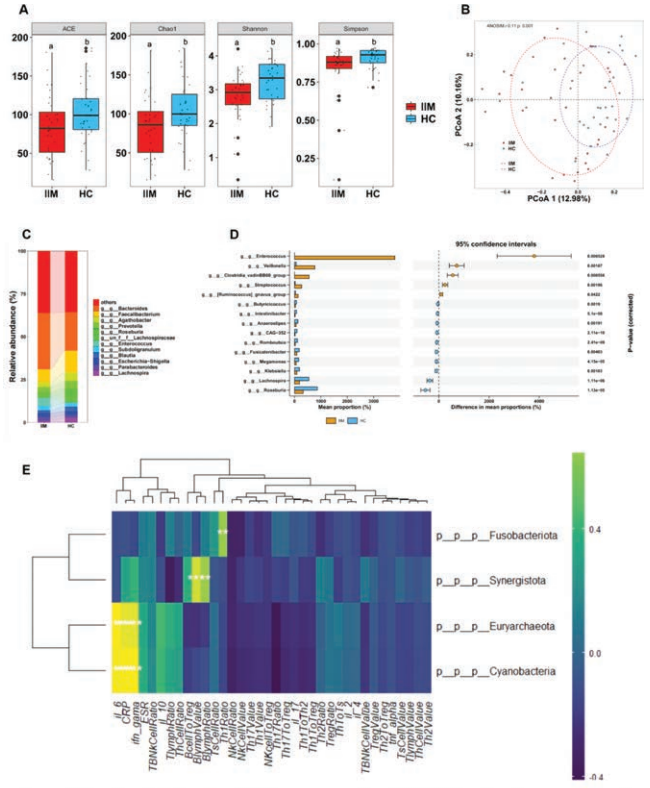

Figure 1: Feature of gut microbiota in IIM patients and HCs. (A) Alpha-diversity assessed by richness (Chao1, ACE) and diversity (Shannon, Simpson), Median estimates compared across cohorts. (B) Principal component analysis (PCoA) plot generated from the Bray-Curtis distances. (C) Microbiota composition based on relative abundance at the genera levels. (D) Panel demonstrated the average relative abundance of different genus in IIM and HCs. (E) Correlation analysis of these differential phylum and lymphocyte subsets and clinical indicators between any two groups using Spearman's correlation analysis.The correlation effect is indicated by a color gradient from green (positive correlation) to purple (negative correlation). *p<0.05. **p<0.01. ***p<0.001.

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POS0144 NOVEL ANTIBODY BIOMARKERS THAT PREDICT FAILURE TO ACHIEVE EARLY AND SUSTAINED DISEASE REMISSION OR LOW DISEASE ACTIVITY AFTER INTENSIVE FIRST-LINE THERAPY IN RHEUMATOID ARTHRITIS

P. Vandormael[1], A. Pues[1], E. Sleurs[1], J. Liesenborgs[2], P. Verschueren[3], V. Somers[1]. *[1]Hasselt University, Biomedical Research Institute, Immunology and Infection, Diepenbeek, Belgium; [2]Hasselt University, Expertise Centre for Digital Media, Computational Science, Diepenbeek, Belgium; [3]KULeuven, University Hospital Leuven, Rheumatology, Leuven, Belgium*

Background: Current EULAR guidelines for the management of rheumatoid arthritis (RA) recommend the use of classical synthetic disease-modifying anti-rheumatic drugs (csDMARDs), combined with short-term use of glucocorticoids (GC) as first-line therapy. Still, finding out which patients will show a poor response to such intensive first-line therapy is currently based on trial and error. Therefore, we recently performed a screening for novel antibody biomarkers that could predict the lack of response to csDMARD/GC combination therapy, resulting in identification of antibodies to 6 novel University Hasselt (UH)-RA antigens.

Objectives: The aim of this study was to validate these novel antibody biomarkers for their potential to identify at baseline, which RA patients fail to reach remission (rem-), or low disease activity (LDA), after intensive csDMARD therapy.

Methods: Presence of antibodies to the identified UH-RA antigens was measured using ELISA in 179 baseline samples from participants of the CareRA trial, which evaluated the efficacy of different first-line combination therapies[1]. Baseline antibody reactivity was correlated with remission or LDA, over a two-year follow-up period, according to the Disease Activity Score based on 28 joints with C-reactive protein (DAS28CRP), the DAS28 with erythrocyte sedimentation rate (DAS28ESR), and the clinical/simplified disease activity index (CDAI/SDAI).

Results: Baseline antibody reactivity against a panel of 3 antigens was higher in patients failing to reach DAS28CRP (31 vs 15%, p=0.007) or DAS28ESR (30 vs 14%, p=0.007) remission at week 8, or those failing to reach SDAI (36 vs 17%, p=0.018) or CDAI (37 vs 17%, p= 0.01) LDA at week 8, compared to RA patients that did reach these respective disease states. Baseline antibody reactivity correlated with lack of DAS28CRP remission after 8, 16, 40 and 52 weeks of first-line therapy, and was present in one in three DAS28CRP rem- patients, and in one in two DAS28CRP rem- patients that were seronegative for RF and ACPA. For each of the remission criteria studied (DAS28CRP, DAS28ESR, SDAI and CDAI), baseline antibody reactivity against these antigens was significantly higher in RA patients that did not maintain sustained remission (resp. 25 vs 12%, p=0.029; 24 vs 11%, p=0.041; 24 vs 4%, p=0.009; 24 vs 4%, 0.011), or sustained LDA (resp. 29 vs 13%, p=0.007; 27 vs 11%, p=0.008; 29 vs 14%, p=0.018; 29 vs 14%, p=0.013) between week 8 and week 52, compared to patients that did reach these respective disease states.

Conclusion: We have identified a set of 3 antibody biomarkers that predict the failure to achieve early and sustained remission and LDA in response to intensive first-line RA therapy. Therefore, the presence of these antibodies might indicate the need for another first-line treatment option in a personalized medicine approach.

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POS0145 CLINICAL RESPONSE TO RITUXIMAB IS ASSOCIATED WITH PREVENTION OF B-CELL DRIVEN SALIVARY GLAND INFLAMMATION AND EPITHELIAL RESTORATION AS REVEALED BY MOLECULAR PATHOLOGY: RESULTS FROM THE TRACTISS TRIAL IN PRIMARY SJOGREN'S SYNDROME

E. Pontarini[1], F. Chowdhury[1], E. Sciacca[1], S. Grigoriadou[1], W. Murray-Brown[1], F. Rivellese[1], D. Lucchesi[1], K. Goldmann[1], L. Fossati-Jimack[1], E. Jaworska[1], G. M. Ghirardi[1], A. Nerviani[1], P. Emery[2], W. F. Ng[3], N. Sutcliffe[4], A. Tappuni[5], M. Lewis[1], S. Arends[6], L. De Wolff[6], H. Bootsma[6], C. Pitzalis[1], S. J. Bowman[7], M. Bombardieri[1]. *[1]William Harvey Research Institute, Queen Mary University of London, Experimental Medicine and Rheumatology, London, United Kingdom; [2]University of Leeds, Chapel Allerton Hospital, Leeds Institute of Rheumatic and Musculoskeletal Medicine, Leeds, United Kingdom; [3]Newcastle University, Translational and Clinical Research Institute, Newcastle-upon-Tyne, United Kingdom; [4]Bart's Health NHS Trust, Royal London Hospital, London, United Kingdom; [5]Institute of Dentistry, Queen Mary University of London, Oral Immunobiology and Regenerative Medicine, London, United Kingdom; [6]University of Groningen, Department of Rheumatology and Clinical Immunology, Groningen, Netherlands; [7]University Hospitals Birmingham NHS Foundation Trust, Rheumatology Department, Birmingham, United Kingdom*

Background: The TRial for Anti-B-Cell Therapy In patients with pSS (TRACTISS) is the largest multi-centre, placebo-controlled, phase-III trial with the administration of 2 cycles of Rituximab (RTX) or placebo at week 0 and 24, with trial clinical endpoints at week 48. Despite the primary endpoints (30% reduction in fatigue or oral dryness) were not met, RTX treated patients showed an improvement in secondary endpoints, such as unstimulated whole salivary flow