two patients had alopecia. There were no cases of type 1 diabetes, vitiligo or thyroid disorders. Interestingly, four patients had lung involvement, two of them with granulomatous-lymphocytic interstitial lung disease (GLILD), one with organising pneumonia and one with usual interstitial pneumonia. Five of the eleven patients required immunosuppressive treatment, mostly with steroids. One case of AIT required concomitant treatment with azathioprine and the patient with psoriasis was treated with methotrexate. In six patients, autoimmune disorder was the first manifestation.

Conclusion: 33% of the CVID patients had autoimmune or inflammatory manifestations in our cohort; six patients had one immune/inflammatory phenomena, four had two different disorders and one patient presented with three different ones. The most frequent manifestation was AIT, as seen in previous reports. In six cases, the autoimmune-inflammatory manifestation was the first symptom attributable to CVID. In conclusion, AD are common in CVID patients, so clinicians must be aware of possible immunodeficiencies in this type of patients.

REFERENCES:

- [1] Amaya-Urbe L, Rojas M, Azizi G, Anaya JM, Gershwin ME. Primary immunodeficiency and autoimmunity: A comprehensive review. *J Autoimmun.* 2019 May;99:52-72.
- [2] ESID Registry – Working Definitions for Clinical Diagnosis of PID, January 22, 2019.

Disclosure of Interests: None declared

DOI: 10.1136/annrheumdis-2021-eular.2696

What the rheumatologist needs to know to interpret studies in 2021

OP0005

DIETARY HYPERINSULINEMIC POTENTIAL AND RISK OF INCIDENT GOUT: 3 PROSPECTIVE COHORT STUDIES OF US MEN AND WOMEN

N. McCormick^{1,2,3,4}, C. Yokose^{1,3,4}, L. Lu^{2,5}, A. Joshi^{3,4,6}, H. Choi^{1,2,3,4}

¹Massachusetts General Hospital, Clinical Epidemiology Program, Division of Rheumatology, Allergy, and Immunology, Boston, United States of America;

²Arthritis Research Canada, Richmond, Canada; ³Massachusetts General Hospital, The Mongan Institute, Boston, United States of America; ⁴Harvard Medical School, Medicine, Boston, United States of America; ⁵Brigham and Women's Hospital, Channing Division of Network Medicine, Boston, United States of America; ⁶Massachusetts General Hospital, Clinical & Translational Epidemiology Unit, Boston, United States of America

Background: Gout and the metabolic (insulin resistance) syndrome frequently coexist. Intravenous insulin has been shown to raise serum urate (SU) levels in physiologic studies and a Mendelian Randomization study also showed a causal role of insulin on the risk of gout. However, it is unknown whether habitual hyperinsulinemic dietary intake confers gout risk.

Objectives: Prospectively examine the relation between two distinct insulin-related dietary indices and risk of incident gout in three large cohorts of US women and men over 30 years.

Methods: We studied 164,090 women from Nurses Health Study I (1986-2016) and II (1989-2017) and 40,598 men from Health Professionals Follow-up Study (1986-2016), who were free of gout at baseline. Dietary intake and covariates were assessed by validated questionnaires every 4 years. Insulinemic potential of diet was evaluated using 1) food-based empirical dietary index for

hyperinsulinemia (EDIH) score that was pre-defined based on circulating C-peptide levels¹ and reflects *insulin resistance*²; and 2) dietary insulin index (DII), which reflects *transient, postprandial insulin secretion*.² We assigned EDIH and DII scores for each participant, adjusted for total energy intake, and prospectively examined the association between scores and incident gout (using ACR survey criteria for gout³), adjusting for potential confounders.

Results: We ascertained 2,874 incident gout cases over 5,124,490 person-years of follow-up. In pooled multivariable-adjusted analyses, those in the highest EDIH quintile had 1.76-times (95% CI: 1.56 to 1.99) higher gout risk, compared with the lowest (Table 1). This attenuated with further adjustment for BMI (a likely causal intermediate) but remained positive (RR 1.30, 1.15 to 1.48). DII scores were inversely associated with gout risk (RR 0.66, 0.58 to 0.74) (Table 1).

Conclusion: EDIH scores, reflecting chronic hyperinsulinemia (i.e., greater insulin resistance with reduced clearance), were positively associated with the risk of incident gout, even beyond the pathway through adiposity. Conversely, higher DII scores, which reflect short-term, postprandial elevations in insulin levels (and also greater insulin clearance and sensitivity) conferred a lower risk. This corroborates human physiologic experiments and Mendelian Randomization studies showing insulin resistance can increase SU levels by decreasing renal excretion of urate, and supports lowering insulinemic potential of diet as a strategy to reduce gout risk.

REFERENCES:

- [1] Tabung *et al.* PMID 27821188
- [2] Lee *et al.* PMID 32618519
- [3] Wallace *et al.* PMID 856219

Disclosure of Interests: Natalie McCormick: None declared, Chio Yokose: None declared, Leo Lu: None declared, Amit Joshi: None declared, Hyon Choi Consultant of: Ironwood, Selecta, Horizon, Takeda, Kowa, Vaxart, Grant/research support from: Ironwood, Horizon

DOI: 10.1136/annrheumdis-2021-eular.719

Opening Plenary Abstract session

OP0006

ASSOCIATIONS OF BASELINE USE OF BIOLOGIC OR TARGETED SYNTHETIC DMARDS WITH COVID-19 SEVERITY IN RHEUMATOID ARTHRITIS: RESULTS FROM THE COVID-19 GLOBAL RHEUMATOLOGY ALLIANCE

J. Sparks^{1,2}, Z. Wallace^{2,3}, A. Seet⁴, M. Gianfrancesco⁴, Z. Izadi^{4,5}, K. Hyrich^{6,7}, A. Strangfeld⁸, L. Gossec^{9,10}, L. Carmona¹¹, E. Mateus^{12,13}, S. Lawson-Tovey^{7,14}, L. Trupin⁴, S. Rush⁴, G. Schmajuk^{4,15}, P. Katz⁴, L. Jacobsohn⁴, S. Al Emadi¹⁶, L. Wise¹⁷, E. Gilbert¹⁸, A. Duarte-Garcia¹⁹, M. Valenzuela-Almada²⁰, T. Hsu¹, K. D'silva^{2,3}, N. Serling-Boyd^{2,3}, P. Dieudé²¹, E. Nikiphorou²², V. Kronzer¹⁸, N. Singh²³, M. F. Ugarte-Gil²⁴, B. Wallace^{25,26}, A. Akpabio²⁷, R. Thomas²⁸, S. Bhana²⁹, W. Costello³⁰, R. Grainger³¹, J. Hausmann^{2,32,33}, J. Liew³⁴, E. Siroitch^{35,36}, P. Sufka³⁷, P. Robinson^{38,39}, P. Machado^{40,41,42}, J. Yazdany⁴ on behalf of COVID-19 Global Rheumatology Alliance. ¹Brigham And Women's Hospital, Division of Rheumatology, Inflammation, and Immunity, Boston, United States of America; ²Harvard Medical School, Division of Rheumatology, Inflammation, and Immunity, Boston, United States of America; ³Massachusetts General Hospital, Division of Rheumatology, Allergy, and Immunology, Boston, United States of America; ⁴University of California, San Francisco, Division of Rheumatology, San Francisco, United States of America; ⁵University of

Table 1. Risk Ratio (95% CI) of Gout According to Quintiles of Insulin-Related Dietary Index

EDIH (measure of insulin resistance)						
	Q1: lowest circulating insulin levels	Q2	Q3	Q4	Q5: highest circulating insulin levels	P for trend
N cases	430	482	598	631	733	
Person-years	1,025,129	1,025,285	1,025,574	1,025,301	1,023,651	
Age-adjusted RR	1.00 (Ref)	1.13 (1.00-1.29)	1.43 (1.26-1.61)	1.53 (1.36-1.73)	1.85 (1.64-2.09)	<.0001
MV-Adjusted [*] RR	1.00 (Ref)	1.11 (0.98-1.27)	1.39 (1.22-1.57)	1.47 (1.30-1.67)	1.76 (1.56-1.99)	<.0001
MV-Adjusted ^{**} RR (+ BMI)	1.00 (Ref)	1.03 (0.90-1.17)	1.21 (1.06-1.37)	1.21 (1.07-1.37)	1.30 (1.15-1.48)	<.0001
Dietary Insulin Index (measure of transient, post-prandial secretion and sensitivity)						
	Q1: lowest insulin sensitivity	Q2	Q3	Q4	Q5: greatest insulin sensitivity	P for trend
N cases	783	611	527	498	455	
Person-years	1,024,763	1,025,730	1,025,075	1,025,538	1,023,834	
Age-adjusted RR	1.00 (Ref)	0.79 (0.71-0.88)	0.69 (0.62-0.77)	0.65 (0.58-0.73)	0.59 (0.53-0.66)	<.0001
MV-Adjusted [*] RR	1.00 (Ref)	0.79 (0.71-0.88)	0.69 (0.62-0.77)	0.66 (0.59-0.74)	0.60 (0.53-0.67)	<.0001
MV-Adjusted ^{**} RR (+ BMI)	1.00 (Ref)	0.78 (0.70-0.87)	0.69 (0.62-0.77)	0.67 (0.60-0.75)	0.66 (0.58-0.74)	<.0001

*Multivariable (MV) models adjusted for age (month), White race, smoking, menopause (women only), hormone use (women only), physical activity, history of hypertension, and diuretic use **MV + BMI models further adjusted for BMI (a likely causal intermediate)

California, San Francisco, Department of Epidemiology and Biostatistics, San Francisco, United States of America;⁶The University of Manchester, Centre for Epidemiology Versus Arthritis, Manchester, United Kingdom;⁷Manchester University NHS FT, National Institute of Health Research Manchester Biomedical Research Centre, Manchester, United Kingdom;⁸Deutsches Rheuma-Forschungszentrum Berlin (DRFZ), ein Institut der Leibniz-Gemeinschaft, German Rheumatism Research Center, Berlin, Germany;⁹Institute Pierre Louis Epidemiology And Public Health, INSERM, Paris, France;¹⁰University Hospitals Pitié Salpêtrière - Charles Foix, Rheumatology Department, Paris, France;¹¹Instituto de Salud Musculoesquelética (InMusc), Rheumatology Department, Madrid, Spain;¹²Portuguese League Against Rheumatic Diseases, Rheumatology Department, Lisbon, Portugal;¹³European League Against Rheumatism (EULAR) Standing Committee of People with Arthritis/Rheumatism in Europe (PARE), Department of Rheumatology, Kilchberg, Switzerland;¹⁴The University of Manchester, Centre for Genetics and Genomics Versus Arthritis, Manchester, United Kingdom;¹⁵San Francisco VA Medical Center, Division of Rheumatology, San Francisco, United States of America;¹⁶Hamad Medical Corporation, Rheumatology Department, Doha, Qatar;¹⁷University of Southern California, Division of Rheumatology, Los Angeles, United States of America;¹⁸Mayo Clinic, Division of Rheumatology, Rochester, United States of America;¹⁹Mayo Clinic, Division of Rheumatology and Robert D. and Patricia E. Kern Center for the Science of Health Care Delivery, Rochester, United States of America;²⁰Mayo Clinic, Division of Rheumatology, Rochester, United States of America;²¹Bichat-Claude Bernard Hospital, Rheumatology Department, Paris, France;²²King's College London, Centre for Rheumatic Diseases, London, United Kingdom;²³University of Washington, Division of Rheumatology, Seattle, United States of America;²⁴Scientific University of the South, Rheumatology Department, Lima, Peru;²⁵University of Michigan, Department of Internal Medicine/Rheumatology, Ann Arbor, United States of America;²⁶VA Ann Arbor Healthcare System, Center for Clinical Management Research, Ann Arbor, United States of America;²⁷Royal Hallamshire Hospital, Rheumatology Department, Sheffield, United Kingdom;²⁸University of Queensland, UQ Diamantina Institute, Queensland, Australia;²⁹Crystal Run Health, Rheumatology Department, Middletown, United States of America;³⁰Irish Children's Arthritis Network, Arthritis Department, Tipperary, Ireland;³¹University of Otago, Rheumatology Department, Dunedin, New Zealand;³²Boston Children's Hospital, Program in Rheumatology, Boston, United States of America;³³Beth Israel Deaconess Medical Center (BIDMC), Division of Rheumatology and Clinical Immunology, Boston, United States of America;³⁴Boston University School of Medicine, Section of Rheumatology, Boston, United States of America;³⁵McMaster University, Department of Health Research Methods, Evidence, and Impact, Hamilton, Canada;³⁶Canadian Arthritis Patient Alliance, Arthritis Department, Toronto, Canada;³⁷Healthpartners, Rheumatology Department, St Paul, United States of America;³⁸University of Queensland, Faculty of Medicine, Brisbane, Australia;³⁹Metro North Hospital and Health Service, Faculty of Medicine, Queensland, Australia;⁴⁰University College London, Centre for Rheumatology & Department of Neuromuscular Diseases, London, United Kingdom;⁴¹University College London Hospitals National Health Service (NHS) Trust, National Institute for Health Research (NIHR) University College London Hospitals Biomedical Research Centre, London, United Kingdom;⁴²Northwick Park Hospital, Department of Rheumatology, London, United Kingdom

Background: Targeted DMARDs may dampen the inflammatory response in COVID-19, perhaps leading to a less severe clinical course. However, some DMARD targets may impair viral immune defenses. Due to sample size limitations, previous studies of DMARD use and COVID-19 outcomes have combined several heterogeneous rheumatic diseases and medications, investigating a single outcome (e.g., hospitalization).

Objectives: To investigate the associations of baseline use of biologic or targeted synthetic (b/ts) DMARDs with a range of poor COVID-19 outcomes in rheumatoid arthritis (RA).

Methods: We analyzed voluntarily reported cases of COVID-19 in patients with rheumatic diseases in the COVID-19 Global Rheumatology Alliance physician registry (March 12, 2020 - January 6, 2021). We investigated RA treated with b/tsDMARD at the clinical onset of COVID-19 (baseline): abatacept (ABA), rituximab (RTX), Janus kinase inhibitors (JAK), interleukin-6 inhibitors (IL6i), or tumor necrosis factor inhibitors (TNFi). The outcome was an ordinal scale (1-4) for COVID-19 severity: 1) no hospitalization, 2) hospitalization without oxygen need, 3) hospitalization with any oxygen need or ventilation, or 4) death. Baseline covariates including age, sex, smoking, obesity, comorbidities (e.g., cardiovascular disease, cancer, interstitial lung disease [ILD]), concomitant non-biologic DMARD use, glucocorticoid use/dose, RA disease activity, country, and calendar time were used to estimate propensity scores (PS) for b/tsDMARD. The primary analysis used PS matching to compare each drug class to TNFi. Ordinal logistic regression estimated ORs for the COVID-19 severity outcome. In a sensitivity analysis, we used traditional multivariable ordinal logistic regression adjusting for covariates without matching.

Results: Of the 1,673 patients with RA on b/tsDMARDs at the onset of COVID-19, (mean age 56.7 years, 79.6% female) there were n=154 on ABA, n=224 on RTX, n=306 on JAK, n=180 on IL6i, and n=809 on TNFi. Overall, 498 (34.3%) were hospitalized and 112 (6.7%) died. Among all patients, 353 (25.3%) were ever smokers, 197 (11.8%) were obese, 462 (27.6%) were on glucocorticoids, 1,002 (59.8%) were on concomitant DMARDs, and 299 (21.7%) had moderate/high RA disease activity. RTX users were more likely than TNFi users to have ILD (11.6% vs. 1.7%) and history of cancer (7.1% vs. 2.0%); JAK users were more likely than TNFi users to be obese (17.3% vs. 9.0%). After propensity score matching, RTX was strongly associated with greater odds of having a worse outcome compared to TNFi (OR 3.80, 95% CI 2.47, 5.85; Figure). Among RTX users, 42 (18.8%) died compared to 27 (3.3%) of TNFi users (Table). JAK use was also associated with greater odds of having a worse COVID-19 severity (OR 1.52, 95%CI 1.02, 2.28). ABA or IL6i use were not associated with COVID-19 severity compared to TNFi. Results were similar in the sensitivity analysis and after excluding cancer or ILD.

Figure. Odds ratios for the ordinal COVID-19 severity outcome for patients with RA on biologic or targeted synthetic DMARDs (n=1673).

The effect size is the odds of being one level higher on the ordinal scale than the reference group.

n for propensity score (PS)-matched analyses: 98 ABA and 525 TNFi; 127 RTX and 520 TNFi; 198 JAK and 527 TNFi; 107 IL6i and 525 TNFi.

Multivariable covariates and propensity score included age, sex, region, season, smoking, obesity, RA disease activity, interstitial lung disease, cancer, comorbidity count, concomitant non-biologic DMARD use, and glucocorticoid use/dose.

Table 1. Frequencies for the ordinal COVID-19 severity outcome for patients with RA on biologic or targeted synthetic DMARDs (n=1673).

COVID-19 outcomes by severity scale (n, %)	ABA n=154	RTX n=224	JAK n=306	IL6i n=180	TNFi n=809
1)Not hospitalized	113 (73.3%)	121 (54.0%)	220 (71.9%)	150 (83.3%)	666 (82.3%)
2)Hospitalization without oxygenation	10 (6.5%)	14 (6.2%)	11 (3.6%)	9 (5.0%)	53 (6.5%)
3)Hospitalization with any oxygenation or ventilation	16 (10.4%)	47 (21.0%)	52 (17.0%)	16 (8.9%)	63 (7.8%)
4)Death	15 (9.7%)	42 (18.8%)	23 (7.5%)	5 (2.8%)	27 (3.3%)

Conclusion: In this large global registry of patients with RA and COVID-19, baseline use of RTX or JAK was associated with worse severity of COVID-19 compared to TNFi use. The very elevated odds for poor COVID-19 outcomes in RTX users highlights the urgent need for risk-mitigation strategies, such as the optimal timing of vaccination. The novel association of JAK with poor COVID-19 outcomes requires replication.

Acknowledgements: The views expressed here are those of the authors and participating members of the COVID-19 Global Rheumatology Alliance and do not necessarily represent the views of the ACR, EULAR, the UK National Health Service, the National Institute for Health Research, the UK Department of Health, or any other organization.

Disclosure of Interests: Jeffrey Sparks Consultant of: Bristol-Myers Squibb, Gilead, Inova, Janssen, and Optum, unrelated to this work, Grant/research support from: Amgen and Bristol-Myers Squibb, unrelated to this work, Zachary Wallace Consultant of: Viela Bio and MedPace, outside the submitted work., Grant/research support from: Bristol-Myers Squibb and Principia/Sanofi, Andrea Seet: None declared, Milena Gianfrancesco: None declared, Zara Izadi: None

declared, Kimme Hyrich Speakers bureau: Abbvie unrelated to this study, Grant/research support from: BMS, UCB, and Pfizer, all unrelated to this study, Anja Strangfeld Paid instructor for: AbbVie, MSD, Roche, BMS, Pfizer, outside the submitted work, Grant/research support from: grants from a consortium of 13 companies (among them AbbVie, BMS, Celltrion, Fresenius Kabi, Lilly, Mylan, Hexal, MSD, Pfizer, Roche, Samsung, Sanofi-Aventis, and UCB) supporting the German RABBIT register, outside the submitted work, Laure Gossec Consultant of: Abbvie, Biogen, Celgene, Janssen, Lilly, Novartis, Pfizer, Sanofi-Aventis, UCB, unrelated to this study, Grant/research support from: Lilly, Mylan, Pfizer, all unrelated to this study, Loreto Carmona: None declared, Elsa Mateus Grant/research support from: grants from Abbvie, Novartis, Janssen-Cilag, Lilly Portugal, Sanofi, Grünenthal S.A., MSD, Celgene, Medac, Pharmakern, GAfPA; grants and non-financial support from Pfizer, outside the submitted work, Saskia Lawson-Tovey: None declared, Laura Trupin: None declared, Stephanie Rush: None declared, Gabriela Schmajuk: None declared, Patti Katz: None declared, Lindsay Jacobsohn: None declared, Samar Al Emadi: None declared, Leanna Wise: None declared, Emily Gilbert: None declared, Ali Duarte-Garcia: None declared, Maria Valenzuela-Almada: None declared, Tiffany Hsu: None declared, Kristin D'Silva: None declared, Naomi Serling-Boyd: None declared, Philippe Dieudé Consultant of: Boehringer Ingelheim, Bristol-Myers Squibb, Lilly, Sanofi, Pfizer, Chugai, Roche, Janssen unrelated to this work, Grant/research support from: Bristol-Myers Squibb, Chugai, Pfizer, unrelated to this work, Elena Nikiphorou: None declared, Vanessa Kronzer: None declared, Namrata Singh: None declared, Manuel F. Ugarte-Gil Grant/research support from: Janssen and Pfizer, Beth Wallace: None declared, Akpabio Akpabio: None declared, Ran-jeny Thomas: None declared, Suleman Bhana Consultant of: AbbVie, Horizon, Novartis, and Pfizer (all <\$10,000) unrelated to this work, Wendy Costello: None declared, Rebecca Grainger Speakers bureau: Abbvie, Janssen, Novartis, Pfizer, Cornerstones, Jonathan Hausmann Consultant of: Novartis, Sobi, Biogen, all unrelated to this work (<\$10,000), Jean Liew Grant/research support from: Yes, I have received research funding from Pfizer outside the submitted work., Emily Sirotich Grant/research support from: Board Member of the Canadian Arthritis Patient Alliance, a patient run, volunteer based organization whose activities are largely supported by independent grants from pharmaceutical companies, Paul Sufka: None declared, Philip Robinson Speakers bureau: Abbvie, Eli Lilly, Janssen, Novartis, Pfizer and UCB (all < \$10,000), Consultant of: Abbvie, Eli Lilly, Janssen, Novartis, Pfizer and UCB (all < \$10,000), Pedro Machado Speakers bureau: Yes, I have received consulting/speaker's fees from Abbvie, BMS, Celgene, Eli Lilly, Janssen, MSD, Novartis, Pfizer, Roche and UCB, all unrelated to this study (all < \$10,000)., Consultant of: Yes, I have received consulting/speaker's fees from Abbvie, BMS, Celgene, Eli Lilly, Janssen, MSD, Novartis, Pfizer, Roche and UCB, all unrelated to this study (all < \$10,000)., Jinoos Yazdany Consultant of: Eli Lilly and AstraZeneca unrelated to this project

DOI: 10.1136/annrheumdis-2021-eular.1632

OP0007

MUC5B PROMOTER VARIANT AND LONG-TERM INCIDENCE OF INTERSTITIAL LUNG DISEASE IN PATIENTS WITH RHEUMATOID ARTHRITIS: A POPULATION BIOBANK STUDY OF 250,000 INDIVIDUALS

A. Palomäki¹, T. Laitinen², J. Koskela³, A. Palotie^{3,4,5}, N. Mars³ on behalf of FinnGen. ¹Turku University Hospital, Centre for Rheumatology and Clinical Immunology, Turku, Finland; ²Tampere University Hospital, Administration Center, Tampere, Finland; ³University of Helsinki, Institute for Molecular Medicine Finland, FIMM, HiLIFE, Helsinki, Finland; ⁴Massachusetts General Hospital, Analytic and Translational Genetics Unit, Department of Medicine, Boston, United States of America; ⁵Broad Institute of MIT and Harvard, Stanley Center for Psychiatric Research, Cambridge, MA, United States of America

Background: The promoter variant rs35705950 in *MUC5B* is the strongest known genetic risk factor for rheumatoid arthritis-associated interstitial lung disease (RA-ILD) [1]. There is, however, no large-scale data on the impact of *MUC5B* on the long-term incidence of RA-ILD.

Objectives: To describe long term risk of RA-ILD in RA patients carrying *MUC5B* variant compared to non-carriers with RA.

Methods: FinnGen is a collection of epidemiological cohorts and hospital biobank samples, linking genotypes with up to 46 years of follow-up within nationwide registries. Diagnoses of RA and ILD were identified from the Finnish national hospital discharge, medication reimbursement and cause-of-death registries. We estimated lifetime risks of ILD by age 80. *MUC5B* is a common variant and has an allele frequency of 0.1 in the Finnish population.

Results: Out of the 248,400 individuals, 5534 patients have been diagnosed with RA, out of whom 178 (3.2%) developed ILD. *MUC5B* was a strong predictor of ILD in RA patients (HR 2.14, 95%CI 1.56-2.92). In patients with RA, *MUC5B* conferred a lifetime risk of 14.5% (95%CI 10.7-18.1%), compared to 5.2% (4.1-6.2%) in *MUC5B* non-carriers with RA (Figure). In the population, *MUC5B* carriers and *MUC5B* non-carriers had lifetime risks of 3.9% and 1.3%, respectively.

The risk difference started to emerge at age 65. The risk was highest in men with RA who are *MUC5B* carriers: 18.5% (11.1-25.2%) developed ILD, compared to 8.5% (6.1-10.9%) of *MUC5B* non-carriers with RA.

Conclusion: We report findings from a large longitudinal study, showing that *MUC5B* confers a considerable lifetime risk of RA-ILD, and contributes to increased morbidity. These findings have clinical implications for improving identification of RA patients at high risk of developing ILD.

REFERENCES:

- [1] Juge P-A, Lee JS, Ebstein E, et al. *MUC5B* Promoter Variant and Rheumatoid Arthritis with Interstitial Lung Disease. *N Engl J Med* 2018;379:2209–19

Disclosure of Interests: Antti Palomäki Speakers bureau: MSD, Pfizer, Sanofi, Consultant of: Pfizer, Abbvie, Tarja Laitinen: None declared, Jukka Koskela Speakers bureau: Pfizer, Aarno Palotie: None declared, Nina Mars: None declared

DOI: 10.1136/annrheumdis-2021-eular.619

OP0008

A RANDOMIZED, DOUBLE-BLIND, PLACEBO-CONTROLLED PHASE III TRIAL OF IVIG 10% IN PATIENTS WITH DERMATOMYOSITIS. THE PRODERM STUDY: RESULTS ON EFFICACY AND SAFETY

R. Aggarwal¹, C. Charles-Schoeman², J. Schessl³, Z. Bata-Csorgo⁴, M. Dimachkie⁵, Z. Griger⁶, S. Moiseev⁷, C. V. Oddis⁸, E. Schiopu⁹, J. Vencovsky¹⁰, I. Beckmann¹¹, T. Levine¹², E. Clodi¹³, A. T. Proderm Investigators¹⁴. ¹University of Pittsburgh, Rheumatology, Pittsburgh, United States of America; ²UCLA Health, Rheumatology, Los Angeles, United States of America; ³Friedrich Baur Institute, University of Munich, Neurology, Munich, Germany; ⁴University of Szeged, Faculty of Medicine, Dermatology, Szeged, Hungary; ⁵University of Kansas, Medical Center, Neurology, Kansas City, United States of America; ⁶University of Debrecen, Department of Internal Medicine, Debrecen, Hungary; ⁷First Moscow State Medical University, Department of Internal Medicine, Moscow, Russian Federation; ⁸University of Pittsburgh, Rheumatology, Pittsburgh, United States of America; ⁹Michigan Medicine, Rheumatology, Ann Arbor, United States of America; ¹⁰Charles University, Rheumatology, Prague, Czech Republic; ¹¹Octapharma PPG, Clinical R&D, Vienna, Austria; ¹²Phoenix Neurological Associates, Neurology, Phoenix, United States of America; ¹³Octapharma PPG, Global Medical & Scientific Affairs, Vienna, Austria; ¹⁴Hospitals in different countries worldwide, Rheumatology-Dermatology-Neurology, Vienna, Austria

Background: Dermatomyositis (DM) is a rare chronic systemic autoimmune disease with characteristic skin rash and progressive proximal muscle weakness. Current therapies encompass corticosteroids and other immunosuppressants and intravenous immunoglobulins (IVIg), however, none of these therapies are proven by randomized controlled phase 3 studies. There have been no large randomized clinical trials supporting the efficacy and safety of IVIg in DM.

Objectives: The ProDERM study aimed to evaluate the efficacy and safety/tolerability of IVIg in DM patients in a double-blind, randomized, placebo-controlled, international multi-center, phase III clinical trial.