Table 1. Proportion of patients with a valid RA diagnosis[1] as a function of RF and aCCP laboratory results

Test Result	aCCP			
	Not Available (0)[2]	Negative (-)	Positive (+)	Total
Rheumatoid Factor	Not Available (0) (N = 1253)[3]	N 427 RA[4] 89.7% Poss[5] RA 5.9%	N 1,159 RA 98.2% Poss RA 0.0%	N 1,586 RA 93.5% Poss RA 3.2%
	Negative (-) N 810 RA 86.3% Poss RA 3.9%	N 5,308 RA 65.3% Poss RA 11.9%	N 2,005 RA 94.5% Poss RA 0.0%	N 8,123 RA 80.7% Poss RA 5.8%
	Positive (+) N 2,229 RA 94.6% Poss RA 0.0%	N 2,566 RA 78.0% Poss RA 8.5%	N 12,978 RA 95.8% Poss RA 2.1%	N 17,773 RA 87.6% Poss RA 4.3%
	Total N 3,039 RA 90.7% Poss RA 1.9%	N 8.301 RA 76.1% Poss RA 9.2%	N 16,142 RA 95.9% Poss RA 0.5%	N 27,482 RA 85.9% Poss RA 4.7%

[1]Diagnosis given by the treating rheumatologist.[2]No test results available or test results available but without normal range values.[3]1,253 patients without available or interpretable RF or aCCP excluded from initial cohort.[4]Percent of 553 charts reviewed confirmed as RA.[5]Poss RA = Possible RA. Patients met our inclusion criteria but the treating rheumatologist never made definitive diagnosis of RA or alternative diagnosis (from 553 charts reviewed).

The percentage of RA-confirmed patients with one test not available, whose complementary test was negative (RF0/aCCP- or RF-/aCCP0), was greater than of patients for whom both tests were negative (RF-/aCCP-). This suggests our data extraction methods may be incomplete or that unidentified bias may be present and warrants further study.

Conclusion: Our methodology for constructing an RA database by selecting patients with ≥2 rheumatology clinic visits, ≥1 ICD-10 diagnosis of RA, and treatment with ≥1 DMARD, has high positive predictive value for RA. Positive RF and aCCP test results were strong predictors of rheumatologists' diagnostic certainty for an RA diagnosis. Thus, the VANRAD database and the associated EHR provide opportunity for a wide range of retrospective observational and prospective longitudinal studies based on 'real-world' patient care.

REFERENCES:

[1] Ng B, et al. *Arthritis Care Res* 2012;64:1490-6; Hanly JG, et al. *Open Access Rheumatol* 2015;7:69-75.

Acknowledgements: This study was sponsored by Bristol Myers Squibb.

Disclosure of Interests: Amy Joseph Speakers bureau: Speakers bureau in the distant past., Grant/research support from: Bristol Myers Squibb, Jodi Yanagida: None declared, Xinliang Huang: None declared, Prabha Ranganathan: None declared, Melissa Laurie Shareholder of: Bristol-Myers Squibb, Employee of: I am currently employed by Bristol Myers Squibb., Hong Xian Grant/research support from: Bristol Myers Squibb Co., Seth Eisen Grant/research support from: The effort presented in this Abstract was supported with funds provided by Bristol-Meyers Squibb.

DOI: 10.1136/annrheumdis-2021-eular.2519

AB0129 IL-6R GENETIC VARIANTS AS PREDICTORS OF CLINICAL RESPONSE TO TOCILIZUMAB IN RHEUMATOID ARTHRITIS PATIENTS

L. Sainz Comas[1], P. Riera[2], P. Moya[1], S. Bernal[3], A. Lasa[3], S. Jeria Navarro[1], D. Lobo Prat[1], H. Codes[1], I. Castellví[1], C. Díaz-Torné[1], A. Laiz[1], B. Magallares[1], A. M. Millán Arciniegas[1], H. Park[1], H. Corominas[1]. [1]*Hospital de la Santa Creu i Sant Pau, Rheumatology, Barcelona, Spain*; [2]*Hospital de la Santa Creu i Sant Pau, Pharmacy/Genetics, Barcelona, Spain*; [3]*Hospital de la Santa Creu i Sant Pau, Genetics, Barcelona, Spain*

Background: Rheumatoid arthritis (RA) is a chronic, systemic, inflammatory autoimmune disease of unknown etiology. Tocilizumab (TCZ) is a first-line biological disease-modifying anti-rheumatic drug (bDMARD) which inhibits Interleukin 6 (IL-6) pathway through blockade of its receptor. At present, there is a lack of evidence to recommend the treatment of one bDMARD over another.(1) Seeking for genetic biomarkers to predict response to treatment could be key towards a personalized treatment strategy in rheumatology.(2)

Objectives: We aimed to evaluate whether functional single nucleotide polymorphisms (SNPs) in the *IL6R* gene could predict response and/or toxicity to TZC in Caucasian patients diagnosed with RA.

Methods: Retrospective analytical preliminar study of a cohort of 31 patients diagnosed with RA (ACR/EULAR 2010 criteria) who received treatment with TCZ within the last 10 years. Epidemiological, clinical and laboratory data were collected. DNA was extracted from EDTA blood samples. Three SNPs in the IL-6 receptor gene (rs12083537, rs2228145, rs4329505) were genotyped by real-time PCR with TaqMan probes. The associations between polymorphisms and clinicopathological features were evaluated using parametric tests. Efficacy was assessed as the difference of DAS-28 CRP at 6 months. The toxicities recorded were hepatotoxicity, infections, hypersensibility, gastrointestinal, hematological and dyslipidemia.

Results: The 31 DNA samples from patients included were mainly female (83.9%) and had a mean age at diagnosis of 46.8 years. The mean duration of treatment was 51.3 months and, previously to initiate TCZ, they received a mean of 2,6 csDMARD and 1,7 bDMARD.

The more frequent adverse effects were hypertransaminasemia (22.6%) and neutropenia (32.3%). Most relevant epidemiologic and clinical data is shown in Table 1.

Table 1. ***Clinical characteristics. RA=Rheumatoid Arthritis. CCP= anti-Cyclic Citrullinated Peptides. RF=Rheumatoid factor. csDMARDs= conventional synthetic Disease-modifying antirheumatic drug. bDMARD= biological Disease-modifying antirheumatic drug. BMI=Body Mass Index. Sc=subcutaneous. Ev=endovenous. DAS28= Disease Activity Score in 28 joints***

Sex (n=31), n (% women/men)	26/5 (83,9%/16,1%)
Age at diagnosis (n=31), years +- SD	46,8+- 12,8
Erosive RA (n=31), n(%)	14 (45,2%)
Anti-CCP positive (n=31), n(%)	23 (74,2%)
UI+- SD	259,7 +- 137,3
RF positive (n=31), n (%)	21 (67,7%)
UI+-SD	189,4+- 114
Previous csDMARD (n=31), nº+-SD	2,6 +-1,3
Previous bDMARD (n=31), nº+- SD	1,7 +- 1,4
BMI (n=29), mean +- SD	29,3+- 5,1
Duration of treatment (n=31), months +-SD	51,3 +- 36,3
-Active treatment (n=12)	-80,9+- 18,3
-Finished treatment (n=19)	-32,6+- 32,2
Route of administration (n=31), n (%) sc/ev	11/20 (35,5/64,5)
Basal DAS28 (n=30), mean+- SD	5,3 +- 1,1
DAS28 reduction at 6 months (n=28), mean+-SD	2,9 +-1,1

The univariate analyses showed that the rs2228145 variant was statistically associated with differences in DAS28 reduction at 6 months (p=0.042). Regarding efficacy, we also found a trend with the SNP rs4329505 (p=0.173), which could achieve statistical significance with the projected inclusion of more patients. No associations were found regarding adverse effects.

Conclusion: The rs2228145 polymorphisms in the *IL6R* gene may be considered as a pharmacogenetic biomarker of TCZ response in RA patients. More studies are required in order to investigate the clinical use of pharmacogenetic biomarkers in rheumatic diseases.

REFERENCES:

[1] Smolen, Josef S., Robert B., et al. 2020. "EULAR Recommendations for the Management of Rheumatoid Arthritis with Synthetic and Biological Disease-Modifying Antirheumatic Drugs: 2019 Update." *Annals of the Rheumatic Diseases* 79 (6): 685–99.

[2] Tarnowski, Maciej, Agnieszka Paradowska-Gorycka, et al. 2016. "The Effect of Gene Polymorphisms on Patient Responses to Rheumatoid Arthritis Therapy." *Expert Opinion on Drug Metabolism & Toxicology* 12 (1): 41–55.

Disclosure of Interests: None declared

DOI: 10.1136/annrheumdis-2021-eular.2615

AB0130 QUESTIONING THE USEFULNESS OF CDAI AS A MEASURE OF DISEASE ACTIVITY IN A TREAT TO TARGET PROGRAMME

C. Lally[1], I. Ali[1], C. Silke[1], B. Whelan[1], M. O'sullivan[1]. [1]*Manorhamilton, Rheumatology, Manorhamilton, Ireland*

Background: Rheumatoid arthritis (RA) is a chronic autoimmune condition which if not treated can lead to joint destruction and long term disability. In RA, the concept of T2T is recommended as the appropriate method to manage early arthritis [1]. It has shown promising results to achieve clinical **remission** (CR) or **low disease activity** (LDA) [2].

Objectives: The objective of this study was to investigate the potential to achieve remission or LDA according to the Clinical Disease Activity Index (CDAI) for RA, during treatment with Disease-Modifying Anti-Rheumatic Drugs (DMARDs) and Biologics, and the factors that affect the remission/LDA outcome.

Methods: We performed an observational prospective study on patients' data available from our Early Arthritis Cohort. All patients with newly diagnosed RA who met the American College of Rheumatology (ACR) criteria were enrolled. Patients are managed by an Advanced Nurse Practitioner (ANP) with consultant supervision. To assess their response to treatment, we used the Clinical Disease Activity Index[3]. Analysis was performed using SPSS.

Results: Out of a total of 459 patients, 353 completed the programme. 217 patients (61.5%) were female and (136) 38.5 % were male. Mean age was 53.98 (SD 14.66). 195 patients were on monotherapy, 40 on combination DMARDs and