18 month timepoints after correction for multiple testing. A similar pattern was observed for IgG ACPA antibody levels, although IgG ACPA levels did not vary significantly comparing baseline with 6, and 6 with 18 month timepoints. Antibody levels were not influenced by exposure to therapy. Patients seropositive for anti-CCP2 at baseline demonstrated less labile antibody levels, with the exception of sustained increases in IgG ACPA reactivity compared with seronegative individuals. **Abstract THU0104 – figure 1.** demonstrates bidirectional changes in anti-PTM antibody reactivity at the patient level.

Abstract THU0104 – Figure 1. Fold change in anti-PTM autoantibody levels between baseline and 18 months in patients with newly-presenting rheumatoid arthritis (n=130)

Conclusions: Median AAPA and, to a lesser extent, ACPA levels fell over time, regardless of therapy. This was most marked for the IgA isotype. Differential isotype effects may represent maturation of the autoantibody repertoire from mucosal IgA antibodies involved in the breach of tolerance. Lability of AAPA levels may reflect the relative reversibility of acetylation of a lysine amino acid residue by comparison with citrullination and carbamylation modifications. By contrast, and as observed previously³, IgG ACPA levels increased over time, perhaps reflecting the tendency of the human proteome to accrue carbamylation modifications due to ageing, and metabolic or inflammatory stress.

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THU0105

RELATIONSHIP BETWEEN DAS28 CATEGORIES AND RAID PATIENT REPORTED OUTCOME IN RHEUMATOID ARTHRITIS; SIGNIFICANT ADVANTAGE OF TARGETING DAS REMISSION

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Background: EULAR/ACR guidelines recommend remission or low DAS28 as the treat to target goal for patients with rheumatoid arthritis (RA). The DAS28 is a composite score derived from objective (swollen joint count and ESR/CRP) and subjective (tender joint count and patient global) measures of disease activity, restricted to 28 joints. It has been criticised as not being representative of the whole patient or completely allied to patient experience. Alternative patient reported outcomes (PROM) have been developed, including the rheumatoid arthritis impact of disease (RAID) which is a self-reported index which assesses seven domains by visual analogue scale: pain, disability, fatigue, sleep, coping,

physical and emotional well-being. Responses are weighted differently producing a final score from 0–10 and a score <2 is considered a patient-acceptable status.

Objectives: Given uncertainty over the necessity to aim for DAS remission (RDAS) as opposed to low DAS (LDAS) as a treatment target, we sought to explore the relation between DAS outcomes and RAID scores in routine clinical practice.

Methods: RA patients attending for routine review in the outpatient clinic at St George's Hospital were assessed by a physician associate between June 2016 and September 2017. DAS28 CRP and ESR scores were recorded and RAID questionnaires completed by patients and calculated using the on-line EULAR tool. Data were analysed on Excel for summary statistics and Spearman correlation coefficient and socscistatistics.com for Mann-Whitney U tests.

Results: 117 RA patients were assessed, 84% female, mean age 59.6 years, 77% RF positive and 85% ACPA positive. The prevalence of DAS28 ESR categories was RDAS (<2.6) n=57 (49%), LDAS (2.6–3.2) n=17 (14.5%), moderate (MDAS 3.21–5.1) n=35 (30%), high (HDAS ≥5.1) n=8 (6.5%). RAID scores correlated strongly with patient global (r=0.62), DAS28 CRP (r=0.58) and DAS28 ESR (r=0.54) but poorly with tender joint count (r=0.32), swollen joint count (r=0.10), ESR (r=0.13) and CRP (r=0.09). The mean RAID score in DAS28 ESR categories was RDAS 2.49, LDAS 3.77, M+HDAS 5.92, see figure 1, box and whisker plots. RAID scores were significantly different (Mann-Whitney U) between M+HDAS versus RDAS (p<0.0001), M+HDAS versus LDAS (p=0.0048) and also between RDAS versus LDAS (p 0.013). Similar significant differences in RAID scores were found with DAS28 CRP categories. There were no significant differences in RAID scores between RF positive versus negative or ACPA positive versus negative patients.

Of 30 patients with RAID <2, DAS28 ESR was <2.6 in 27 (90%) and <3.2 in 29 (97%). Of 74 patients with DAS28 ESR <3.2, RAID was >2 in 45 (61%) with fatigue followed by sleep being the worst scoring domains.

Conclusions: RAID scores strongly correlate with patient global and total DAS28 (ESR or CRP) scores, and are significantly different between all DAS categories, including RDAS versus LDAS. Patients with RAID <2 are almost all at LDAS target, but high numbers at LDAS have unacceptable RAID, largely driven by high fatigue and sleep scores. This suggests that achieving RDAS has significant benefit over LDAS from a PROM perspective, and attention should be paid to fatigue and sleep if RAID is unacceptable in LDAS patients.

Disclosure of Interest: None declared

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THU0106

CONVERTING PATIENT-REPORTED OUTCOME MEASURES OF FATIGUE AND PAIN TO PROMIS SCORES: DATA FROM PHASE 3 BARICITINIB RHEUMATOID ARTHRITIS TRIALS

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Background: Fatigue and pain in patients (pts) with RA are often measured with the Functional Assessment of Chronic Illness Therapy-Fatigue (FACIT-F) and the Medical Outcomes Study Short-Form-36 (SF-36). Patient-Reported Outcomes Measurement Information System (PROMIS) was developed using a population-calibrated T-score metric (Mean 50, SD 10). Crosswalk tables were developed linking legacy instruments to PROMIS instruments, including Fatigue and Pain

Interference (PI). Comparisons to the general population can be made from PROMIS scores.¹⁻²

Objectives: To convert FACIT-F and SF-36 Bodily Pain (BP) scores to PROMIS Fatigue and PI scores to determine how PROMIS performs in 2 phase 3 baricitinib (bari) RA trials.

Methods: In RA-BEAM, pts with inadequate response (IR) to MTX were randomised 3:3:2 to placebo (PBO) once daily (QD), bari-4 mg QD, or adalimumab (ADA) 40 mg biweekly.³ In RA-BEACON, pts with IR to bDMARDs were randomised 1:1:1 to receive PBO or bari 2 mg or 4 mg QD.⁴ FACIT-F assessed fatigue and SF-36 BP, pain. Pt-level PROMIS scores were converted from FACIT-F/SF-36 BP using validated crosswalk tables.¹⁻² Analysis of covariance was conducted on PROMIS score conversions to compare bari to all treatment arms.

Results: Pts had considerable baseline fatigue/pain; mean scores approached or exceeded 1 SD (10 points on the metric) from population norms. Bari was associated with clinically relevant improvements (exceeding 0.5 SD/5 points on the T-score metric) vs PBO for PROMIS fatigue/PI scores (table 1). PROMIS fatigue/PI scores in RA-BEAM reached population norms (<55) by week 12 for bari and ADA (table 1). Bari remained associated with significant improvements in PROMIS fatigue/PI vs PBO through 24 weeks in both studies and vs ADA for PI in RA-BEAM (figure 1).

Data are mean (SD)

^aHigher PROMIS scores mean more fatigue/pain

^bBEACON was a 24 week study

ADA=adalimumab; bari=baricitinib; PBO=placebo

Abstract THU0106 – Figure 1. Change from baseline for PROMIS Fatigue and Pain Interference in RA-BEAM and RA-BEACON

Conclusions: While PROMIS was not used in the studies directly, pt-level cross-walk tables provide an estimate of effect that may be demonstrated when using PROMIS in clinical trials. These results provide preliminary evidence of the ability of PROMIS to demonstrate treatment benefit.

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Abstract THU0106 – Table 1. PROMIS Patient-Reported Outcome scores^a in RA-BEAM and RA-BEACON

	PBO (n=488)	RA-BEAM Bari-4 mg (n=487)	ADA (n=330)	PBO (n=176)	RA-BEACON ^b Bari-2 mg (n=174)	Bari-4 mg (n=177)
PROMIS Fatigue converted from FACIT-F						
Baseline	58.1 (7.8)	58.6 (7.6)	59.0 (8.4)	62.8 (7.5)	62.4 (7.0)	61.9 (8.2)
Week 4	54.5 (7.6)	52.9 (7.5)	53.5 (7.9)	59.3 (8.0)	57.7 (8.5)	56.7 (8.5)
Week 12	53.2 (8.0)	51.4 (8.3)	51.7 (8.6)	58.7 (8.7)	56.0 (9.5)	55.3 (9.3)
Week 24	53.1 (8.5)	50.2 (8.7)	51.1 (9.0)	58.2 (8.7)	56.0 (9.4)	54.4 (10.3)
Week 52	–	50.1 (8.6)	51.0 (9.2)	–	–	–
PROMIS Pain Interference converted from SF-36 Bodily Pain score						
Baseline	61.3 (7.9)	61.9 (7.4)	61.9 (8.3)	65.2 (7.3)	63.7 (7.1)	64.8 (7.8)
Week 4	58.3 (7.8)	55.2 (7.4)	55.9 (7.9)	61.6 (7.1)	59.3 (8.3)	57.8 (7.9)
Week 12	57.5 (8.1)	53.7 (8.6)	54.8 (8.6)	60.2 (8.7)	57.6 (9.1)	57.2 (9.4)
Week 24	57.2 (8.4)	52.7 (8.9)	54.0 (8.8)	60.8 (8.3)	58.1 (8.8)	55.8 (9.8)
Week 52	–	52.0 (9.4)	53.1 (9.8)	–	–	–

THU0107

EVALUATION OF CARDIOVASCULAR RISK IN PATIENTS WITH RHEUMATOID ARTHRITIS AND ANTI-TNF-ALPHA THERAPY

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Background: Rheumatoid arthritis is a chronic inflammatory autoimmune disease that causes systemic inflammation associated with increased risk of coronary, cerebral and peripheral ischaemic accidents. Biological therapy, by decreasing systemic inflammation, implicitly reduces cardiovascular risk in patients with rheumatoid arthritis. The assessment of carotid atheromatosis in Doppler ultrasound examination can be a useful element in the diagnosis of patients with cardiovascular risk.

Objectives: We followed the cardiovascular risk as well as the development of the carotid artery plaque by carotid Doppler ultrasound, lipid profile and arterial hypertension in patients with rheumatoid arthritis who have undergone biological treatment with anti-TNF-alpha. We also studied the correlation of the intima-medium thickness index in the carotids with the activity of rheumatic disease (DAS28, CRP, VSH) and the evolution of these parameters under biological therapy.

Methods: Our study included 37 patients with rheumatoid arthritis in treatment with DMARD's (group 1) and 50 patients with rheumatoid arthritis in treatment with anti-TNF-alpha (etanercept, adalimumab, infliximab) biological therapy (group 2). The following parameters were assessed in all patients: DAS28, lipid profile (total cholesterol, LDL, HDL, triglycerides), which were followed at 0, 6, and 12 months. The Intima-Medium Thickness Index was measured by Mode B sonography at the level of the common carotid artery, bifurcation of both arteries and the external carotid artery. The value of the index above 0.7 mm was considered pathological.

Results: In lot 1, after the carotid Doppler ultrasound, of the 37 patients, 21 presented the carotid artery plaque, and in lot 2 of the 50 patients, 34 showed these changes. A full evaluation (DAS28, lipid profile, arterial tension, carotid ultrasound) of all patients was performed at the start of the study. At 6 months, inflammatory markers and DAS28 showed a decrease in baseline values in both groups, more significant in the biological therapy group. Reduction of the intima-medium thickness index and blood pressure was observed in both groups, with no significant differences. At 12 months, CRP and DAS28 in the DMARD-treated group corresponded to disease remission values in 33 patients (89%), and in the group treated with biological therapy, the same remission rates of the disease were recorded in 49 patients (97.7%). The intima-medium thickness index in the DMARD-treated group decreased from the 6 month values. In patients with biological therapy, the reduction in the mid-thickness index recorded a progressive decrease from the previous assessment.