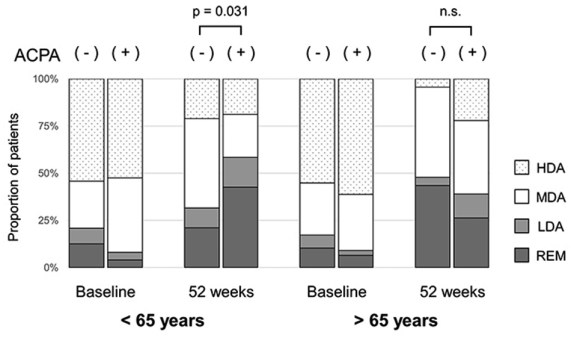
of low disease activity (LDA) at 52 weeks, separately in the young (<65 years, n=215) and the elderly (<65 years, n=248) group.

Results: Mean age was 55.3/74.0 years (young/elderly), disease duration was 10.2/12.8 years, DAS28-CRP was 4.34/4.40 at baseline, concomitant MTX was used in 55.4/37.1%, concomitant steroid was used in 51.0/49.6%, and proportion of bio-naïve was 49.3/63.3%. As shown in table 1, multivariate analysis revealed that no history of previous biologics and lower DAS28 score at baseline was the independent positive predictors in both young and elderly group. Interestingly, ACPA positivity was significant predictor only in the young group. The ACPA positive patients showed the significantly higher proportion of LDA achievement at 52 weeks, only in the young group (figure 1).



Conclusions: It has been reported that lower proportion of the elderly onset RA (EORA) patients has ACPA positivity, and some reports have previously demonstrated that the ACPA negative was negatively associated with good clinical outcomes of abatacept. Our current results suggested that the effect of ACPA positivity on the clinical results of abatacept treatment was different between ages. Abatacept would be a valuable treatment option in the ACPA negative elderly RA patients.

Disclosure of Interest: None declared

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Abstract THU0214 – Table 1. Logistic regression analysis to study predicitive factors for LDA achievement at 52 weeks.

	Univariate		Multivariate	
<65 years	OR (95% CI)	p-value	OR (95% CI)	p-value
Male	0.944 (0.438–2.036)	0.884		
Disease duration,<10 years	1.454 (0.771–2.742)	0.248		
ACPA positive	3.044 (1.070–8.656)	0.037	4.544 (1.049–19.689)	0.043
No previous biological DMARDs	2.458 (1.336–4.524)	0.004	3.386 (1.119–10.244)	0.031
Concomitant MTX	1.879 (1.022–3.453)	0.042		
Concomitant PSL	0.693 (0.380–1.265)	0.232		
mHAQ,<0.5	2.756 (1.373–5.531)	0.004		
DAS28-CRP at baseline	0.631 (0.489–0.814)	<0.001	0.457 (0.260–0.803)	0.006
>65 years	OR (95% CI)	p-value	OR (95% CI)	p-value
Male	1.205 (0.601–2.418)	0.599		
Disease duration,<10 years	2.154 (1.120–4.141)	0.021		
ACPA positive	0.697 (0.284–1.711)	0.431		
No previous biological DMARDs	3.040 (1.564–5.912)	0.001	3.678 (1.370–9.876)	0.010
Concomitant MTX	1.357 (0.746–2.467)	0.318		
Concomitant PSL	0.469 (0.256–0.857)	0.014		
mHAQ,<0.5	3.224 (1.670–6.224)	<0.001		
DAS28-CRP at baseline	0.571 (0.444–0.736)	<0.001	0.638 (0.433–0.942)	0.024

THU0215

EFFICACY OF SARILUMAB IN PATIENTS WITH RHEUMATOID ARTHRITIS WITH AND WITHOUT PREVIOUS RESPONSE TO TOCILIZUMAB

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Background: Sarilumab is an IL-6R inhibitor recently approved for the treatment of rheumatoid arthritis (RA). To help clinical decision making, we studied the response rate on open-label sarilumab treatment in patients previously treated with tocilizumab, the other IL-6R inhibitor approved for RA. In the ASCERTAIN trial (NCT01768572), patients were randomised 1:1:2 to 24 weeks of sarilumab 150 mg sc every 2 weeks (q2w; n=49), sarilumab 200 mg sc q2w (n=51), or tocilizumab 4 mg/kg iv q4w (n=102) increased to 8 mg/kg at investigator's discretion if clinically indicated as per the US label; each added to conventional synthetic disease-modifying antirheumatic drug (csDMARD) background therapy. Patients who completed ASCERTAIN were eligible to enrol in an open-label extension study of sarilumab 200 mg sc q2w (EXTEND, NCT01146652), with csDMARDs as specified in the previous study.

Objectives: To examine outcomes for patients who switched from tocilizumab in ASCERTAIN to open-label sarilumab in EXTEND.

Methods: In this post-hoc analysis, patients were recorded as responders or non-responders at the end of ASCERTAIN and at Weeks 12 and 24 of EXTEND according to each of: clinical disease activity index (CDAI)≤2.8, CDAI≤10.0, disease activity score (DAS)−28 CRP <2.6, DAS-28 CRP <3.2, and American College of Rheumatology (ACR)20/50/70 response criteria.

Results: A total of 168 patients entered EXTEND from ASCERTAIN, of whom 93 had been in the tocilizumab group (last tocilizumab dose 4 mg/kg in 37 patients and 8 mg/kg in 56 patients). After switch to sarilumab, response was achieved in an additional number of patients who were non-responders on tocilizumab at the end of ASCERTAIN (table 1). The reverse, response loss in tocilizumab responders, was infrequent. Patients who switched from tocilizumab 4 mg/kg or 8 mg/kg achieved similar response rates.

Last tocilizumab dose, mg/kg	Week in EXTEND	CDAI		DAS28-CRP				
		≤2.8	≤10	<2.6	<3.2	ACR20	ACR50	ACR70
		Non-responders at the end of ASCERTAIN with response in EXTEND (%)[a,b]						
4	12	3/27[11]	7/18[39]	8/23[35]	8/18[44]	3/5 (60)	8/19[42]	7/25[28]
	24	4/25[16]	7/16[44]	9/22[41]	10/17 (59)	3/4 (75)	6/17[35]	5/22[23]
8	12	7/48[15]	9/31[29]	13/36[36]	8/21[38]	2/11[18]	14/30[47]	9/43[21]
	24	10/46[22]	13/29[45]	16/35[46]	7/20[35]	6/10 (60)	13/28[46]	13/41[32]
		Responders at the end of ASCERTAIN with response in EXTEND (%)[a,b]						
4	12	8/9 (89)	15/18 (83)	11/11 (100)	16/16 (100)	28/31 (90)	16/17 (94)	10/11 (91)
	24	6/8 (75)	15/17 (88)	9/10 (90)	14/15 (93)	27/29 (93)	14/16 (88)	9/10 (90)
8	12	4/7 (57)	21/24 (88)	13/17 (76)	26/32 (81)	38/42 (90)	19/23 (83)	7/11 (64)
	24	5/7 (71)	20/24 (83)	14/18 (78)	28/33 (85)	39/43 (91)	19/24 (79)	9/12 (75)

[a]responder status according to measure listed in column heading; [b]subjects with missing measurements are excluded from numerator and denominator

Conclusions: These results may indicate that clinical improvements can be attained in a relevant proportion of tocilizumab non-responders with switch to sarilumab, irrespective of previous tocilizumab dose, and the majority of patients responding to tocilizumab maintain response when switching to sarilumab.

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THU0216 SERUM CXCL16 LEVELS IN RF+/ACPA+RHEUMATOID ARTHRITIS PATIENTS BEFORE AND AFTER TREATMENT WITH DMARDS

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Background: Rheumatoid arthritis (RA) is characterised by profound mononuclear cell (MNC) recruitment into synovial tissue (ST). Studies have shown that chemokine CXCL16 is a premier MNC recruiter in RA. CXCL16 contributes to chronic inflammation, since it is highly expressed in RA synovial fluid (SF), is a potent chemoattractant for mononuclear cells (MNCs) *in vitro*, and is chemotactic for peripheral blood mononuclear cells (PBMCs) to RA ST *in vivo*. Hence treatment for RA will reflect change in serum CXCL16 levels
Objectives: The aim of this study was to analyse the change of serum chemokine level of CXCL16, in patients with either RF +or ACPA +rheumatoid arthritis (RA), by DMARDs treatment.
Methods: This was a prospective interventional study done in a tertiary care centre. 31 patients with RA were recruited for a period of 12 months. Serum CXCL16 levels were assayed in them along with other baseline investigations. The patients were treated with DMARDs. CXCL16 levels post treatment were measured after 6 months. For comparison another group of age and sex matched controls was taken (n=18) and their serum CXCL16 was also recorded. The serum CXCL16 levels were correlated with disease activity.
Results: After treatment with conventional DMARDs 26 patients showed lowering of mean serum CXCL16 levels from 56.07 pg/ml to 21.79 pg/ml (62% reduction) after 6 months. The patients who showed inadequate response to conventional DMARD treatment (n=5) underwent therapy with biological DMARDs(TNF-α blocker) which reduced their CXCL16 levels from 63.81 pg/ml to 12.36 pg/ml (80.6% reduction) in subsequent 6 months. There was a corresponding improvement in the disease activity of RA. Lowering of CXCL16 was found to correlate positively with improvement of symptoms and lowering of disease activity
Conclusions: DMARDs treatment significantly lowered the serum levels of CXCL16 in patients with RA. CXCL16 is one of the crucial chemokines regulated by DMARDs treatment

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Disclosure of Interest: None declared
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THU0217 SIMILAR EFFICACY AND SAFETY OF SARILUMAB 150 MG OR 200 MG Q2W REGARDLESS OF PRIMARY (1°) OR SECONDARY (2°) FAILURE WITH TNF INHIBITORS

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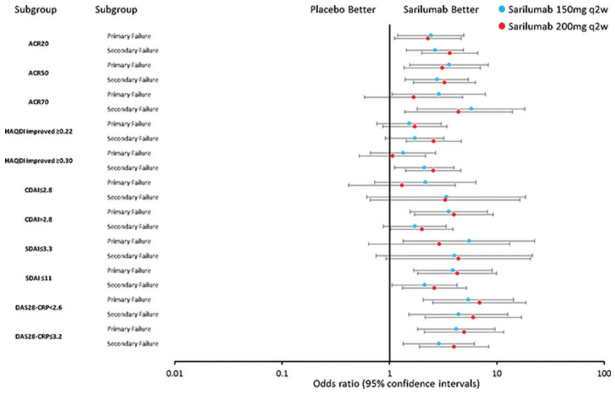
Background: Sarilumab (150 or 200 mg subcutaneously [SC] every 2 wks [q2w]) +csDMARDs demonstrated efficacy in adults with moderate–to-severely active rheumatoid arthritis (RA) with intolerance or inadequate response to prior TNFi treatment in TARGET (NCT01709578). Patients with an initial refractory response (1° failure) to TNFi may respond differently to subsequent treatment vs those who initially respond but lose TNFi effectiveness (2° failure).
Objectives: This post hoc analysis examined efficacy and safety of sarilumab +csDMARDS in patients who had previously demonstrated 1° vs 2° TNFi failure.
Methods: TNFi failure (1° vs 2°) was investigator-determined on enrolment to TARGET. Patients who experienced 1° or 2° failure were randomised to placebo (pbo; n=75 and n=99), sarilumab 150 mg (n=72; n=91), or sarilumab 200 mg q2w (n=64; n=103), respectively. Disease activity, physical function (HAQ-DI), and safety were assessed at Wk 24.
Results: By Wk 24, ACR20/50/70 response rates and improvements in LS mean HAQ-DI were similar in both sarilumab dose groups and superior to pbo, irrespective of 1° vs 2° TNFi failure (table 1). Odds ratios for the benefit of sarilumab over pbo according to ACR response rates, HAQ-DI, DAS28-CRP, CDAI and SDAI (figure 1) showed no differences between patients with 1° vs 2° failures. No significant treatment by subgroup (1° vs 2° failure) interactions were observed. In the 1° failure group, treatment emergent adverse events (TEAEs; table 1) occurred in 59.7%, 65.6% vs 45.3% (sarilumab 150, 200 mg vs pbo, respectively) of patients; and in 73.6% and 63.1% vs 52.5% with sarilumab 150, 200 mg vs pbo, respectively, in the 2° failure group. There was only one TEAE leading to death (pbo group) and one case each of venous thrombosis (200 mg sarilumab q2w, 2°) and pulmonary embolism (150 mg sarilumab q2w, 2°).



Conclusions: Key efficacy and safety measures were similar in patients treated with sarilumab +csDMARDs, regardless of previous 1° or 2° failure with TNFis.

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