Table 1. Baseline data regarding patient characteristics, prior treatment and disease activity

patient characteristics		seronegative		seropositive	
		patients [n] (%)		patients [n] (%)	
prior RA treatment	patients [n] (%)	105 (22.2)		279 (59.0)	
	Sex female [n] (%)	81 (77.1)		207 (74.2)	
	Age mean [years] (SD)	56.6 (12.5)		59.6 (10.9)	
	BMI mean (SD) [kg/m ²]	29.2 (6.7)		28 (6.0)	
	Smoking history current/former [%]	22.1/11.5		25.5/20.4	
disease activity	Time since diagnosis of RA mean [years] (SD)	7.9 (7.6)		11.5 (9.9)	
	csDMARD [n] (%)	102 (97.1)		273 (97.8)	
	bDMARD (TNFi) [n] (%)	54 (51.4)		167 (59.9)	
	bDMARD (non-TNFi) [n] (%)	32 (30.5)		83 (29.7)	
	tsDMARD (JAKi) [n] (%)	25 (23.8)		57 (20.4)	
disease activity	CRP [mg/l] (SD)	12.9 (20.9)		14.2 (31.0)	
	ESR [mm/h] (SD)	23.7 (20.5)		25.3 (22.4)	
	SJC mean (SD)	5.3 (5.5)		4.6 (4.8)	
	TJC mean (SD)	10.2 (8.2)		7.2 (6.6)	
	HAQ-DI mean (SD)	1.4 (0.7)		1.2 (0.7)	
	DAS28-ESR mean (SD)	5.0 (1.5)		4.6 (1.5)	
	CDAI mean (SD)	27.7 (14.7)		23.0 (12.9)	
	Fatigue [VAS] (SD)	64.0 (25.5)		51.1 (28.1)

Fig. 1 only shows patients with post-baseline data available (full analysis set) and therefore differs slightly to baseline data in Tab. 1 (baseline analysis set)
BL=baseline

Figure 1. CDAI (A, B) and HAQ-DI (C) outcomes in seronegative and seropositive patients

Conclusion: Sarilumab treatment ameliorated CDAI in both seronegative (Δ CDAI -17.0 ± 16.5) and seropositive (Δ CDAI -15.4 ± 14.3) patients to the same extent over the course of 12 months, with a higher remission rate in seropositive patients. Functional capacity improved meaningfully in the seropositive cohort but had no significant impact in seronegative patients. The safety profile was consistent with data reported from controlled clinical trials.

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POS0592

BIOSIMILAR INFlixIMAB THERAPY IN RHEUMATOID ARTHRITIS, ANKYLOSING SPONDYLITIS AND PSORIATIC ARTHRITIS: A LONG-TERM FOLLOW-UP STUDY ON INFlixIMAB-NAÏVE PATIENTS AND PATIENTS SWITCHED FROM THE ORIGINATOR TO THE BIOSIMILAR CT-P13

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Objectives: The ReFLECT study was carried out to investigate real life use of CT-P13, the first monoclonal antibody biosimilar to infliximab originator.

Methods: ReFLECT is a multicentre, prospective, observational study conducted in France to determine the characteristics of patients (pts) receiving CT-P13, its effectiveness and safety in a real-life setting. Eligible were both pts who had been switched from infliximab originator (IFX_S) and infliximab-naïve pts started on CT-P13 (IFX_N). Intermediate results in pts with rheumatic diseases using descriptive statistical analyses from inclusion to a 24-month-follow-up period are described here.

Results: Among the 1370 adult pts included between October 2016 and April 2019, data were analysed for 142 pts with rheumatoid arthritis (RA; 23.9% males; mean age: 61.5 ± 10.9 years; 80 IFX_N/61 IFX_S), 411 with ankylosing spondylitis (AS; 58.9% males; 48.1 ± 13.1 years; 228 IFX_N/179 IFX_S), and 96 with psoriatic arthritis (PsA; 41.7% males; 53.4 ± 14.1 years; 55 IFX_N/40 IFX_S) after a median of between 8.7 and 12.9 years since diagnosis. At inclusion, 66.9%, 24.3% and 50% had one concomitant treatment, mainly methotrexate. At the time of the first administration of CT-P13, disease had been active in 94.2%, 76.6% and 83.3% of the IFX_N pts compared with 45.3%, 24.0% and 32.0% of the IFX_S pts. After 24 months of follow-up, both CRP levels and disease activity remained stable compared to baseline in the IFX_S pts: median Δ CRP/RA_S = 1, Δ CRP/AS_S = 0, Δ CRP/PsA_S = 0 and median Δ DAS28/RA_S = 0, Δ BASDAI/AS_S = 0, Δ DAS28/PsA_S = 1. As expected, improvement was observed in the IFX_N pts: median Δ CRP/RA_N = -2, Δ CRP/AS_N = -2, Δ CRP/PsA_N = 0 and median Δ DAS28/RA_N = -1, Δ BASDAI/AS_N = -2, Δ DAS28/PsA_N = -1. In naïve pts, CT-P13 brought both CRP and disease activity down to levels comparable to those seen in patients switched from the IFX originator: median [Q1;Q3] CRP/RA_N = 5 [2;9] vs CRP/RA_S = 5 [2;8]; CRP/AS_N = 3 [2;9] vs CRP/AS_S = 3 [2;5]; CRP/PsA_N = 2 [1;4] vs CRP/PsA_S = 2 [1;3] and median [Q1;Q3] DAS28/RA_N = 3 [3;4] vs DAS28/RA_S = 2 [2;3]; BASDAI/AS_N = 3 [1;4] vs BASDAI/AS_S = 2 [1;3]; DAS28/PsA_N = 3 [2;3] vs DAS28/PsA_S = 3 [2;3]. (Figure 1). At 24 months, 62.7%, 74.9% and 75.0% remained on treatment with CT-P13 in RA, AS and PsA pts respectively. The reasons for treatment withdrawing and safety results are reported in Table 1.

Figure 1. Disease activity at the time of first administration of CT-P13 and after 24 months**Table 1. Reasons for treatment withdrawing and safety results**

Reasons for treatment withdrawing						
IFX pts, n (%)	RA _N , 37	RA _S , 15	AS _N , 64	AS _S , 38	PsA _N , 11	PsA _S , 12
Missing	1	0	0	0	0	1
Remission	0	2 (13.3)	2 (3.1)	0	0	0
Therapeutic failure *	25 (69.4)	8 (53.3)	41 (64.1)	23 (60.5)	5 (45.5)	6 (54.5)
Intolerance	5 (13.9)	1 (6.7)	5 (7.8)	2 (5.3)	1 (9.1)	1 (9.1)
Other	6 (16.7)	4 (26.7)	16 (25.0)	13 (34.2)	5 (45.5)	4 (36.4)
Safety data, n (%)						
RA, 142	AS, 411					
Pts with ≥ 1 adverse event (AE)	67 (47.2)	222 (54.0)				
Pts with ≥ 1 AE related to CT-P13	33 (23.2)	84 (20.4)				
Pts with ≥ 1 serious AE	19 (13.4)	42 (10.2)				
Pts with ≥ 1 allergic infusion reaction †	4 (2.8)	11 (2.7)				
Pts with ≥ 1 infection ‡	10 (7.0)	18 (4.4)				

* Encompassing primary failure in IFX_N pts and loss of efficacy in IFX_N and IFX_S pts † Including acute and delayed hypersensitivity reactions ‡ Including severe infections, tuberculosis, opportunistic infections, hepatitis

Conclusion: Year 2 follow-up data indicate that CT-P13 effectively induced improvement in disease activity in pts with RA, AS and PsA receiving infliximab for the first time and maintained stable disease activity in pts switched from

IFX originator to CT-P13. This real-life study did not highlight any new safety concerns.

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POS0593

THE REDUCTION OF RETINAL MICROVASCULAR ALTERATIONS AND THE DECREASE OF A POTENTIALLY RELATED T-CELL SUBSET IN PATIENTS WITH ACTIVE RHEUMATOID ARTHRITIS TREATED WITH ABACEPT

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Background: T-cells play a pivotal role in the pathogenesis of rheumatoid arthritis (RA) and in its cardiovascular (CV) comorbidities, acting at microvascular level [1]. Since small artery remodeling is the earliest form of target organ damage in hypertension, the evaluation of microvascular alterations might provide clinically useful information. The evaluation of retinal arterioles is a non-invasive technique to identify a precocious microvascular damage, which is related to an increase of the wall-to-lumen ratio (WLR) [2]. CD3+CD31+CXCR4+ T-cells may be involved in damaged endothelium repair and are increased in patients with morphological microvascular alterations [3]. In addition to its effect on disease activity, abatacept (ABA), a co-stimulator blocker which is approved for the treatment of RA, may have specific CV protective action, modulating the numbers of certain subtypes of lymphocytes [4].

Objectives: To non-invasively investigate morphological characteristics of retinal arterioles and to evaluate CD3+CD31+CXCR4+ T-cells in a cohort of RA patients treated with ABA.

Methods: Eleven RA patients [median (25th-75thpercentile) age=58 (50-65) years, baseline C-reactive protein (CRP)-DAS28=4.4 (3.8-4.6), body mass index (BMI)=23.4 (21.6-25.6) kg/m², rheumatoid factor (RF) positive:45%, anti-citrullinated peptide autoantibodies (ACPA) positive:73%] without known CV risk factors (arterial hypertension, diabetes, hypercholesterolemia, previous CV events, smoking) were evaluated by adaptive optics, a validated technique quantifying microvascular damage [5], before and every 6 months of therapy with ABA (T0, T6 and T12). Phenotypic analysis of peripheral blood T lymphocytes was made by flow-cytometry in 5 patients of the cohort at T0 and T6.

Results: A progressive significant reduction of the WLR of retinal arterioles was observed [T0=0.28 (0.25-0.30), T6=0.27 (0.24-0.31), T12=0.23 (0.23-0.26); p T0 vs T6=0.4; p T6 vs T12=0.01; p T0 vs T12=0.01] (Figure 1), without significant variations in the other parameters [internal diameter: T0=94.4 (84.1-104.0), T6=94.8 (84.6-107.7), T12=99.2 (89.1-109.1) µm; external diameter: T0=125.8 (111.1-131.0), T6=122.4 (109.1-134.5), T12=125.6 (113.9-134.4) µm; wall thickness: T0=13.2 (12.2-14.4), T6=13.4 (11.7-14.4), T12=12.5 (11.6-13.0) µm; wall cross-sectional area: T0=4581.0 (3788.7-5263.7), T6=4563.3 (3788.5-5295.2), T12=4099.7 (3899.0-5145.7) µm²]. In 5 patients evaluated also for T-cell immunophenotyping a negative correlation was observed between CD3+CD31+CXCR4+ T-cell number and the retinal wall thickness at baseline (R=0.871;p=0.05). After ABA therapy a trend for reduction of CD3+CD31+CXCR4+ T-cells [19.0 (13.8-38.3) vs 12.4 (5.2-18.0) % of CD3+], was observed as well as of significant reduction of retinal wall cross-sectional area [5123.3 (4385.0-5470.3) vs 4852.3 (4118.3-5228.0) µm²;p=0.04].

Conclusion: In a cohort of RA patients without known CV risk factors, a reduction in retinal microvascular alterations arterioles was demonstrated after treatment for 12 months with ABA. CD3+CD31+CXCR4+ T-cell number was inversely related to the possible presence of subclinical CV involvement. These results may suggest the possibility of microvascular abnormalities regression induced by the immune system modulation.

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Wall to lumen ratio (WLR)

Figure 1.

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POS0594

MEANINGFUL IMPROVEMENT AND WORSENING IN PATIENTS WHO DO NOT ACHIEVE LDA AND SWITCH THERAPY TO A NEW BIOLOGIC OR TARGETED THERAPY: RESULTS FROM THE CORRONA REGISTRY

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Background: Guidelines recommend adjusting therapy in patients with rheumatoid arthritis (RA) who fail to reach and sustain low disease activity (LDA) or remission (disease control). Many factors can affect the decision to change therapy, including the potential for improvement as well as the fear of potential worsening or loss of improvement already achieved. Although data exist on response to treatment in patients who switch therapy, data addressing the likelihood of worsening are limited.

Objectives: The aim of this analysis was to describe the demographic, clinical characteristics, and change in clinical outcomes in patients on biologic/targeted synthetic disease-modifying anti-rheumatic drugs (b/tsDMARDs) who had some improvement in clinical disease activity index (CDAI) but did not achieve LDA after ~ 6-12 months of treatment and then switched to a different b/tsDMARD.

Methods: This study included adult inadequately responding RA patients from the CORRONA registry who: (1) started a biologic or Janus kinase inhibitor (JAKi) between January 2010 to November 2020 (V1), (2) had any CDAI improvement (i.e., decrease ≥ 1 unit) but were not in LDA or remission at a subsequent visit (baseline [BL]) occurring 3 to 15 months after V1; (3) had a third visit (follow-up [F/U]) 6 (± 3) months after BL with a valid CDAI measure; (4) switched therapy at the BL or between BL and F/U, with the switch occurring at least 3 months prior to the F/U. CDAI >10 and ≤ 22 was defined as moderate disease activity (MDA) and CDAI >22 was defined as high disease activity (HDA). These thresholds of change in CDAI (≥ 6 and ≥ 12 units) were used to define meaningful improvement and meaningful worsening after the switch. If there was no meaningful improvement or meaningful worsening, this was considered as no meaningful change (-5 to $+5$ for 6 units change and -11 to $+11$ for 12 units change). These thresholds for meaningful change were set for all switchers regardless of their pre-switch CDAI value. Descriptive statistics were generated for demographic and clinical characteristics for the switchers at BL, and the change of clinical outcomes was evaluated from BL to F/U.