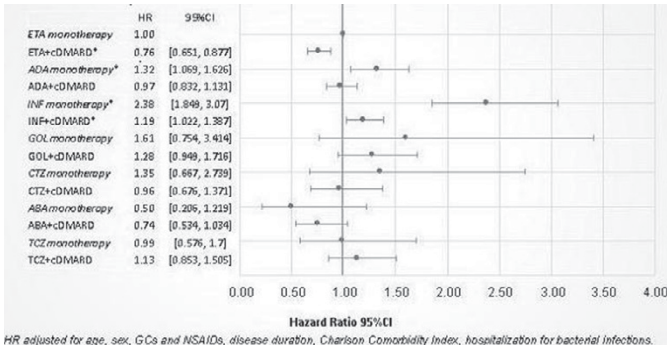
Compared to monotherapy, combination therapy was associated with a lower drug failure (crude HR 0.75 [95% CI 0.68–0.82]; adjusted HR 0.78 [95% CI 0.70–0.86]; $p<0.0001$).

In patients in monotherapy, considering ETA as reference and adjusting for the above mentioned clinical characteristics, the HR for bDMARD failure was 1.32 for ADA (95% CI 1.07–1.63) and 2.38 for INF (95% CI 1.85–3.07).



HR adjusted for age, sex, GCs and NSAIDs, disease duration, Charlson Comorbidity Index, hospitalization for bacterial infections.

Conclusions: Monotherapy with bDMARDs is consistenly associated with lower retention rate in first-line therapy for anti-TNF drugs. Comparing bDMARDs administered in monotherapy, INF and ADA show a higher risk of withdrawal than ETA. Real life data support the currently recommended use of bDMARDs in association to csDMARDs.

References:
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FRI0213 COMPARATIVE EFFECTIVENESS OF ABATACEPT, RITUXIMAB, TOCILIZUMAB AND ANTI-TNF BIOLOGICAL DMARDS IN RA: RESULTS FROM THE NATIONWIDE SWEDISH REGISTER

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Background: Many current guidelines rank abatacept (ABA), rituximab (RTX), tocilizumab (TOC), and the TNFi bDMARDs as equal in effectiveness for the treatment of RA, at least as second line therapies. This is mainly based on evidence from separate RCTs, with few direct comparisons and limited comparative effectiveness data from clinical practice.
Objectives: To describe outcomes in clinical practice among RA patients starting different bDMARDs as first bDMARD, and after switch from initial TNFi.
Methods: The Swedish Rheumatology Register was linked to nationwide registers with data on demographics and medical history. We included all patients with RA starting a first ever bDMARD, or switching to a new bDMARD after a TNFi as first bDMARD, in 2010 - 2014, with follow-up through 2015. Effectiveness was assessed at 1 year (±90 days) after starting therapy, and measured as 1) the proportion remaining on therapy, or the proportion remaining on therapy and with 2) Good EULAR response, 3) HAQ improvement >0.2, 4) no swollen or tender joints. Relative response was estimated with log-binomial regression adjusting for potential confounders.
Results: Patients starting non-TNFi were older than those starting a TNFi, had lower socioeconomic status, and more often a history of diseases including malignancy, serious infections, and diabetes. After switch from TNFi, those starting non-TNFi also had higher disease activity.
Non-TNFi were associated with better drug survival and higher proportion reaching response outcomes compared to TNFi as first bDMARD. After switch from TNFi, RTX and TOC, but not ABA, were associated with significantly better drug survival and response. Differences remained after adjusting for identified potential confounders.
Conclusions: Despite channeling of older and sicker individuals to non-TNFi-bDMARDs, treatment outcomes were in general better in these groups, particularly for TOC and RTX. In interpreting this, the risk of residual confounding should be remembered, and that we did not include safety or long term outcomes.
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FRI0214 LONG-TERM EFFICACY AND SAFETY OF SIRUKUMAB IN PATIENTS WITH ACTIVE RHEUMATOID ARTHRITIS DESPITE ANTI-TUMOR NECROSIS FACTOR THERAPY: RESULTS OF THE RANDOMIZED, PHASE 3 SIRROUND-T STUDY

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Background: Sirukumab, a selective, high-affinity human monoclonal antibody to the interleukin-6 (IL-6) cytokine, is under development for rheumatoid arthritis (RA) and other diseases.
Objectives: To evaluate long-term efficacy and safety of sirukumab in patients (pts) with RA refractory or intolerant to anti-tumor necrosis factor (TNF) agents.
Methods: This phase 3 study included pts ≥18 years with moderate to severe active RA, and a lack of benefit to ≥1 anti-TNF or intolerance to ≥2 anti-TNFs. Eligible pts were initially randomized 1:1:1 to sirukumab subcutaneous (SC) 50mg q4w, sirukumab SC 100mg q2w, or placebo SC q2w for 24 wks. Placebo-treated pts with <20% improvement in tender and swollen joints at Wk 18 (early escape [EE]) and those remaining on placebo at Wk 24 (crossover) were re-randomized to sirukumab through Wk 52. Efficacy endpoints included ACR response, HAQ-DI scores, DAS28 (CRP) remission rates, and SF-36 scores. Results are presented for these key endpoints at Week 52.
Results: 878 pts were initially randomized to placebo (n=294), sirukumab 50 mg q4w (n=292), or sirukumab 100 mg q2w (n=292). Of placebo-treated pts, 94 met EE criteria at Wk 18 and 158 crossed over at Wk 24 and were re-randomized to sirukumab. 60% of pts had received ≥2 prior biologics, including non-TNF–targeted biologics. RA signs and symptoms and patient-reported outcomes (PROs [SF-36 scores]) improved significantly with sirukumab versus placebo through Wk 24. Improvements were maintained through Wk 52 with no dose response (Table 1). Through Wk 52 in the combined sirukumab 50mg q4w and 100mg q2w groups, respectively, an adverse event (AE) was reported for 79.6% and 81.3% of pts and a serious AE was reported for 14.2% and 13.2% of pts; injection-site reactions and alanine aminotransferase increases were the most commonly reported AEs.
Conclusions: In this population intolerant or refractory to anti-TNFs/other biologics, sirukumab SC 50mg q4w and 100mg q2w were well tolerated and reduced signs and symptoms of RA and improved PROs through 52 wks of treatment, also among pts who switched from placebo to sirukumab.
Disclosure of Interest: Y. Tanaka Grant/research support from: Mitsubishi-Tanabe, Takeda, Daiichi-Sankyo, Chugai, Bristol-Myers, MSD, Astellas, Abbvie, and Eisai, Speakers bureau: Abbvie, Chugai, Daiichi-Sankyo, Bristol-Myers, Mitsubishi-Tanabe, Astellas, Takeda, Pfizer, Teijin, Asahi-kasei, YL Biologics, Sanofi, Janssen, Eli Lilly, and GlaxoSmithKline, D. Aletaha Grant/research support from: AbbVie, Pfizer, Grünenthal, Merck Medac, UCB, Mitsubishi/Tanabe, Janssen, and Roche, Consultant for: AbbVie, Pfizer, Grünenthal, Merck Medac, UCB, Mitsubishi/Tanabe, Janssen, and Roche, P. Agarwal Shareholder of:

Abstract FRI0213 – Table 1. Status at 12 months among all patients with RA initiating a biologic DMARD 2010–2014 in Sweden

	TNFi	RTX		TOC		ABA	
	%	%	RR†	%	RR†	%	RR†
First bDMARD	N=5568	N=654		N=202		N=240	
On drug	68.4	87.8	1.34 (1.27–1.41)	75.5	1.20 (1.09–1.31)	77.7	1.15 (1.05–1.27)
On drug + EULAR Good resp.	26.1	31.1	1.42 (1.19–1.69)	53.1	2.03 (1.70–2.42)	34.3	1.37 (1.10–1.72)
On drug + HAQ Improvement	26.7	39.2	1.64 (1.40–1.93)	45.0	1.54 (1.27–1.87)	36.8	1.37 (1.09–1.71)
On drug + 28 Joint count = 0	20.3	22.4	1.13 (0.89–1.43)	30.9	1.60 (1.21–2.11)	22.8	1.26 (0.91–1.74)
Switch from TNFi	N=1840	N=408		N=320		N=256	
On drug	57.7	80.2	1.48 (1.37–1.60)	73.0	1.36 (1.23–1.49)	65.1	1.11 (0.98–1.26)
On drug + EULAR Good resp.	11.4	24.0	1.87 (1.41–2.49)	36.8	3.06 (2.37–3.94)	14.6	1.16 (0.76–1.76)
On drug + HAQ Improvement	16.6	34.3	1.85 (1.49–2.30)	32.4	1.71 (1.33–2.19)	20.4	1.10 (0.78–1.53)
On drug + 28 Joint count = 0	12.3	20.8	1.96 (1.43–2.70)	19.9	2.12 (1.48–3.02)	11.2	0.86 (0.48–1.52)

†Adj. for region, sex, age, birth country, RF, dis. dur., HAQ, DAS28, co-medication, recent history of malignancy, infection, SSRI, and hospital days last 5 yrs.