BSR CONCURRENT ORAL PRESENTATION OF ABSTRACTS

Concurrent Oral 1 – Rheumatoid Arthritis: Treatment

OP4. INHIBITION OF RADIOGRAPHIC PROGRESSION AND IMPROVEMENTS IN PHYSICAL FUNCTION AT 2 YEARS, WITH INCREASING CLINICAL EFFICACY OVER TIME, IN RHEUMATOID ARTHRITIS (RA) PATIENTS TREATED WITH TOCILIZUMAB (TCZ): THE LITHE STUDY

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Background: Patients with moderate to severe RA who remained on methotrexate (MTX) despite inadequate response were treated with TCZ in a double-blind, randomized, controlled phase 3 trial. Results of a 2-year planned analysis from this study are presented.
Methods: Patients were randomized to treatment with TCZ 4 mg/kg + MTX (TCZ4), TCZ 8 mg/kg + MTX (TCZ8) or placebo + MTX (CON) every 4 weeks. If patients failed to respond (<20% improvement in swollen and tender joint count; SJC and TJC) then stepwise rescue therapy could begin at week 16. Patients with ≥ 70% improvement in SJC and TJC could continue the blinded therapy at the end of year 1 to week 104. For all other patients, open-label TCZ8 was initiated at week 52. Change from baseline in Genant-modified total Sharp score (GmTSS) and physical function (AUC of change from baseline in HAQ-DI) were the primary 2-year end points. Linear extrapolation (GmTSS) or standardization (change in HAQ-DI) was used for missing data. The impact of 2 years of treatment was examined by assessing efficacy end points over time for patients randomized to TCZ8, with the last observation carried forward for SJC and TJC in patients who received rescue therapy or withdrew.
Results: The intention to treat population consisted of 398 TCZ8, 399 TCZ4 and 393 CON patients. At 2 years, exposure rates in patient-years (PY) were 1320.0, 521.9 and 284.8 in TCZ8, TCZ4 and CON patients, respectively. At year 2, patients in the TCZ8 group had 81% less radiographic progression *vs* CON patients (based on linear extrapolation of mean change in GmTSS). Significantly more TCZ8 patients had no radiographic progression *vs* CON patients ($P \leq 0.0001$). AUC of change from baseline in HAQ-DI showed significant improvement in physical function in TCZ4 and in TCZ8 *vs* CON patients ($P \leq 0.0025$). In patients initially randomized to TCZ8, a low disease activity score (LDAS; DAS28 < 3.2) was seen in > 60% of patients and the DAS28 remission (DAS28 < 2.6) rate was 48% at week 52 and continued to increase to week 104. By week 52, patients treated with TCZ8 had clinically significant improvements in SJC that were maintained through week 104. Rates per 100 PY for adverse events (AEs) were higher in TCZ8 and TCZ4 (263.6, 275.4) *vs* CON patients (251.4) while rates for serious AEs were comparable (11.4, 12.1, 10.9, respectively). Rates per 100 PY of AEs leading to withdrawal (7.4, 32.5, 4.8) and treatment modification (8.4, 30.7, 20.4) were higher in TCZ8 and TCZ4 *vs* CON patients, respectively and death rates were comparable (0.6, 0.2, 0.4).
Conclusions: Treatment with TCZ + MTX inhibits radiographic progression over 2 years and improves physical function as shown by DAS28 remission, LDAS and low SJC, with a manageable safety profile.

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OP5. BENEFIT OF CONCOMITANT DMARD USE ON THE PERSISTENCE WITH ANTI-TNF THERAPIES IN RHEUMATOID ARTHRITIS: RESULTS FROM THE BRITISH SOCIETY OF RHEUMATOLOGY BIOLOGICS REGISTER (BSRBR)

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Background: Increasingly, one or more disease modifying anti-rheumatic drugs (DMARDs) other than methotrexate (MTX) are being used with anti-tumour necrosis factor (anti-TNF) therapies in rheumatoid arthritis (RA). The effect of these alternative concomitant therapies on anti-TNF treatment persistence remains relatively unknown. This study aimed to evaluate the effect of different concomitant DMARDs on the persistence with anti-TNF therapies in RA.
Methods: This analysis included 10396 patients with RA registered with the BSRBR who were starting their first anti-TNF therapy (etanercept ($n = 3605$), infliximab ($n = 3136$) or adalimumab ($n = 3655$)) and were receiving one of the following DMARD/combinations: No DMARD ($n = 3339$), MTX ($n = 4418$), leflunomide (LEF) ($n = 610$), sulphasalazine (SSZ) ($n = 308$), MTX + SSZ ($n = 902$), MTX + hydroxychloroquine (HCQ) ($n = 401$) or MTX + SSZ + HCQ ($n = 418$). Kaplan–Meier survival analysis was used to study the persistence with anti-TNF therapy in each DMARD subgroup out to 5 years. Patients were censored at the time their first anti-TNF was discontinued, at the last known follow-up, or death, whichever came first. Multivariate Cox proportional hazard models, adjusted for baseline age, gender, DAS28, HAQ, anti-TNF therapy and previous DMARD history, were used to compare treatment persistence overall and according to the reason of discontinuation (inefficacy or adverse events) between each of the DMARD subgroups, using MTX as the reference group.
Results: One-year drug survival (95% CIs) of the first anti-TNF therapy was 71% (71:72) but this dropped to only 42% (41:43) at 5 years. Compared with MTX, patients receiving no DMARD, LEF or SSZ were more likely to discontinue their first anti-TNF therapy while patients receiving MTX in combination with other DMARDs showed superior treatment persistence. These associations remained when the results were restricted to the reason for stopping (inefficacy or adverse events) and when stratified by anti-TNF therapy (Table).
Conclusions: The use of concomitant DMARDs with anti-TNF therapies in RA significantly improves treatment persistence, with the best drug survival seen in those receiving combination therapies. These results would support the continued use of background DMARD therapy combinations which include MTX. Consideration should be given to the discontinuation of LEF and SSZ monotherapy at the time anti-TNF therapies are commenced.

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Risk of Anti-TNF discontinuation

DMARD Co-therapy	Adjusted Hazard Ratios of the multivariate analysis (95% CIs) *$P < 0.05$					
	All Stops	Stopping for Inefficacy	Stopping for Adverse Events	All Stops - Etanercept	All Stops - Infliximab	All Stops - Adalimumab
MTX	Reference					
None	1.40* (1.30:1.51)	1.34* (1.20:1.51)	1.47* (1.30:1.65)	1.27* (1.12:1.45)	1.39* (1.18:1.63)	1.42* (1.25:1.61)
SSZ	1.23* (1.03:1.47)	1.34* (1.04:1.74)	1.21 (0.91:1.60)	0.89 (0.64:1.24)	1.42 (0.92:2.19)	1.41* (1.11:1.80)
LEF	1.41* (1.25:1.59)	1.58* (1.32:1.88)	1.34* (1.10:1.62)	1.27* (1.02:1.57)	1.79* (1.43:2.26)	1.31* (1.07:1.60)
MTX-SSZ	0.76* (0.67:0.86)	0.77* (0.64:0.92)	0.70* (0.57:0.86)	0.64* (0.49:0.85)	0.87 (0.73:1.04)	0.72* (0.57:0.90)
MTX-HCQ	0.81* (0.68:0.96)	0.83 (0.64:1.06)	0.85 (0.66:1.11)	0.97 (0.68:1.37)	0.76* (0.58:0.98)	0.77 (0.58:1.03)
MTX-SSZ-HCQ	0.80* (0.68:0.95)	0.75* (0.58:0.96)	0.87 (0.67:1.13)	0.93 (0.64:1.35)	0.75* (0.59:0.96)	0.79 (0.60:1.05)

Research Grant, Roche - Research Grant. All other authors have declared no conflicts of interest.

OP6. RITUXIMAB PLUS METHOTREXATE (MTX) INHIBITS JOINT DAMAGE AND IMPROVES CLINICAL OUTCOMES IN PATIENTS WITH EARLY, ACTIVE RHEUMATOID ARTHRITIS (RA) WHO ARE NAÏVE TO MTX

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Background: To assess the clinical and radiological benefit of addition of rituximab to MTX in patients with early, active RA not previously treated with MTX.
Methods: Patients with active RA (swollen and tender joint counts ≥ 8) and naïve to MTX were randomized to either placebo + MTX or rituximab (2×500 mg or 2×1000 mg) + MTX. In all groups, MTX was initiated at 7.5 mg/week and titrated to 20 mg/week by week 8. Rituximab was given by IV infusion on days 1 and 15. From week 24, patients received a second course of rituximab when DAS28 was ≥ 2.6. Radiographs were taken at screening and at weeks 24 and 52 and were centrally read using the Genant-modified Sharp method (mTSS; readers blinded to sequence and treatment). The primary endpoint was change from screening in the mTSS at week 52.
Results: Of the 755 randomized patients, 715 were radiographically evaluable. Groups were well matched at baseline with mean RA duration of 0.9 years and DAS28 > 7. Rituximab (2×1000 mg) + MTX resulted in a significantly lower change in mTSS and significantly higher proportion of patients with no joint progression compared with placebo + MTX at 52 weeks (Table). Both doses of rituximab improved clinical outcomes compared with MTX alone. Safety data were consistent with those previously reported. Serious adverse events occurred in 10% (placebo + MTX), 9% (rituximab 2×500 mg + MTX) and 10% (rituximab 2×1000 mg + MTX) of patients. The rate of serious infections was 6.09, 4.61 and 3.73 events/100 patient-years, respectively. Three deaths occurred (pneumonia n = 2 and cerebral infarct), which all occurred in the placebo + MTX group.
Conclusions: Rituximab (2×1000 mg) + MTX significantly improved clinical outcomes (including major clinical response) and inhibited joint damage, compared with MTX alone in patients with early, active RA. Rituximab (2×500 mg) improved clinical but not radiological outcomes.

Disclosure statement: A.C., Genentech - Employment, Stocks or Stock options, Roche - Stocks or Stock options. E.H., Roche - Employment. C.P., Synarc Inc - Stock or stock options, Synarc Inc - Employment, Synarc Inc - Ownership or partnership. W.R., Genentech - Consulting fees, Speakers bureau, Biogen/IDEC - Consulting fees, Speakers bureau. A.R., Roche - Consulting fees, Abbott - Consulting fees, UCB - Consulting fees, Essex - Consulting fees, BMS - Consulting fees, Chugai - Consulting fees, Wyeth - Consulting fees. T.S., Roche - Stocks or Stock options, Employment. P.P.T., Roche - Research grants, Consulting fees, Merck-Serono - Research grants Genentech - Consulting fees. H.T., Roche - Employment. R.F.vV., Roche - Consulting fees, Research grants, Abbott - Research grants, Schering Plough - Research grants, UCB - Consulting fees, Wyeth - Consulting fees. All other authors have declared no conflicts of interest.

	Placebo + MTX	Rituximab (2×500 mg) + MTX	Rituximab (2×1000 mg) + MTX
Radiological outcome at 52 weeks	n = 232	n = 239	n = 244
Mean change in mTSS	1.079	0.646	0.359**
Patients with change in mTSS ≤ 0 (%)	53.4	57.7	63.5*
Clinical outcome at 52 weeks	n = 249	n = 249	n = 250
ACR20 (%)	64.3	76.7*	80.0***
ACR50 (%)	41.8	59.4***	64.8***
ACR70 (%)	24.9	42.2***	46.8***
ACR90 (%)	9.2	17.3*	16.4*
Major clinical response (%)	8.0	17.3*	18.4**
EULAR response (%)	71.1	82.3***	86.0***
DAS remission (%)	12.6	25.4**	30.5***
Mean change in DAS28	n = 244	n = 247	n = 248
	-2.06	-3.05***	-3.21***

*$P < 0.05$, **$P < 0.001$, ***$P < 0.0001$ compared with placebo + MTX

OP7. AN ASSESSMENT OF THE SERIOUS INFECTION RATE IN RITUXIMAB-TREATED RHEUMATOID ARTHRITIS (RA) PATIENTS WHO SUBSEQUENTLY RECEIVED OTHER BIOLOGIC THERAPIES: A FOLLOW-UP FROM RITUXIMAB CLINICAL TRIALS

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Background: As the pharmacodynamic effect of rituximab may be long lasting, this analysis was conducted to describe the rate of serious infection events (SIEs) in patients with RA previously treated with rituximab and subsequently receiving a biologic disease-modifying antirheumatic drug (DMARD) while peripherally B cell (CD20+) depleted.
Methods: Patients with moderately to severely active RA receiving rituximab plus methotrexate who withdrew or exited from clinical studies entered a safety follow-up (SFU) period. During SFU, peripheral B cell counts were monitored at regular intervals for ≥ 48 weeks and patients were permitted to receive other biologics. SIEs were recorded throughout the SFU.
Results: As of April 2008, 2579 RA patients had received ≤ 1 rituximab course, which amounted to 5964.3 patient-years (pt-yrs) of follow-up. Overall SIE rate was 4.26 (95% CI: 3.77, 4.92) per 100 pt-yrs. Of patients who entered the SFU, 216 were subsequently treated with another biologic; most ($n = 178$) received a TNF inhibitor (TNFi: etanercept, infliximab or adalimumab) after rituximab for 191.72 pt-yrs, whereas others received abatacept ($n = 31$, 19.25 pt-yrs) or anakinra ($n = 9$). Median follow-up after receipt of the subsequent biologic was 11 months (range: 0–45). For the majority of patients (86.1%), peripheral B cell CD19 levels were depleted below the lower limit of normal at the time of receiving their subsequent biologic. About 35% of patients received their biologic within 6 months of their last rituximab infusion. Median time to the SIE after initiating biologic therapy was 6 months (range: 0–23). In the 31 patients receiving abatacept and the nine patients receiving anakinra, there was one SIE before and one SIE after receiving each of these biologics. Serious infections were typical for RA patients with no opportunistic or fatal infections.
Conclusions: The use of other biologics in RA patients previously treated with rituximab was not associated with an increase in rate of SIEs in patients who received biologics or in the subgroup who received a TNFi.

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	SIEs in patients receiving biologic DMARDs following rituximab treatment			
	Other biologic post rituximab		TNFi post rituximab	
	Before biologic	After biologic	Before TNFi	After TNFi
Exposure, pt-yrs	227.00	224.00	183.58	191.72
No. of SIEs	13	12	11	10
SIE rate (95% CI), per 100 pt-yrs	5.73 (3.33, 9.86)	5.36 (3.04, 9.43)	5.99 (3.32, 10.82)	5.22 (2.81, 9.69)

OP8. GENETIC POLYMORPHISMS IN KEY METHOTREXATE PATHWAY GENES ASSOCIATED WITH RESPONSE TO MTX TREATMENT IN RHEUMATOID ARTHRITIS

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Background: Methotrexate (MTX) is the cornerstone of treatment for rheumatoid arthritis (RA). However, up to a third of individuals will fail to respond to MTX treatment or suffer adverse events (AE). The main determinates of MTX response in RA remain unclear, although evidence suggests that part of this variability is genetic. Genes within the MTX metabolic pathway represent good candidates as predictors of response. We investigated SNPs spanning 11 MTX pathway genes on the efficacy and occurrence of AE in MTX treated patients with RA.
Methods: Subjects included 309 RA patients with a defined response to MTX. Patients were included if they were (i) good responders ($n = 147$) (with an ESR < 20 and/or normal CRP and on a stable dose of MTX for at least 6 months) (ii) inefficacy failures ($n = 101$) (physician statement and failure to reduce ESR/CRP by 20%) or (iii) AE failures ($n = 61$) (verified by medical record review). Tagging SNPs were selected for genes: *AMPD1, ATIC, DHFR, FPGS, GGH, ITPA, MTHFD1, MTHFR, SHMT1, SLC19A1 (RFC)* and *TYMS* using an r2 cutoff ≥ 0.8 and MAF ≥ 0.05 within 10kb up and down stream of each gene. Genotyping was performed using the Sequenom iPlex® MassARRAY platform. Three different analyses were conducted: 1) responders *vs* inefficacy failures 2) responders *vs* AE failures and 3) responders *vs* inefficacy and AE failures combined. Genotype frequencies were compared between the groups using the trend test implemented in PLINK and allelic odds ratios with 95 % CIs (CI) calculated in STATA 9.2.
Results: Of the 150 SNPs tested, nine were found to be significantly associated (p-trend ≤ 0.05) with MTX response and two with MTX related AE. Interestingly three of these (rs12995526, rs7563206 and rs16853834) were found in the 5-aminoimidazole-4-carboxamide ribonucleotide transformylase (ATIC) gene, which encodes an enzyme that is important in the de novo purine synthesis pathway. Individuals carrying these SNPs had an increased risk of poor response to MTX (OR 1.48, 95% CI (1.01-2.17), OR 1.47, 95% CI (1.00-2.17) and OR 1.47 95% CI (1.01-2.76). Other associations included 6 SNPs in the SLC19A1 gene (rs11702425, rs2838956, rs7499, rs2274808, rs9977268, rs7279445) all conferring an increased risk of poor response to MTX and two SNPs in the DHFR gene (rs12517451, rs10072026) were associated with AE. In addition there were a further two SNPs in the FPGS gene associated with AE under a recessive model (rs1054774, rs4451422) and one additional SNP in the *ATIC* gene approaching statistical significance (rs4673990) (p-trend < 0.1) warranting further investigation.
Conclusions: Genetic variations in a number of key MTX pathway genes have been found to be significantly associated with MTX response and AE in RA patients. Further studies will be required to validate these findings. If confirmed these results could contribute towards a better understanding of and ability to predict MTX response in RA.
Disclosure statement: All authors have declared no conflicts of interest.

OP9. A COMPARISION OF TOCILIZUMAB (TCZ) AND METHOTREXATE (MTX) MONOTHERAPIES IN MTX- OR DMARD-NAÏVE PATIENTS WITH RHEUMATOID ARTHRITIS (RA)

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Background: In the AMBITION study, TCZ monotherapy demonstrated clinical superiority over MTX monotherapy in patients with active RA who had not previously failed MTX or biologic treatment, with a well-defined and manageable safety profile. The aim of this analysis was to compare the efficacy of TCZ and MTX monotherapies in MTX-naïve and disease-modifying anti-rheumatic drug (DMARD)-naïve patients with RA from AMBITION.
Methods: This study was a post hoc analysis from the AMBITION study, in which patients ($N = 570$, intent-to-treat [ITT] population) were randomized to one of two treatment arms for 24 weeks: intravenous TCZ 8 mg/kg every 4 weeks or an initial dose of MTX 7.5 mg/week titrated up to 20 mg/week within 8 weeks. Previous exposure to MTX was allowed in this study, but not within 6 months prior to study entry and patients could not have failed previous MTX, other DMARDs or biological therapy. For this analysis, MTX-naïve patients were defined as never having been exposed to MTX and DMARD-naïve patients as never having been exposed to MTX or any traditional DMARD; patients in these groups were not mutually exclusive. ACR20, ACR50 and ACR70 responses, DAS28 remission (DAS28 < 2.6) and EULAR good response efficacy parameters were assessed at 24 weeks for the ITT population and the MTX- and DMARD-naïve patients of the AMBITION study.
Results: In the ITT population, 67% of patients were MTX naïve and 43% were DMARD naïve. Baseline demographics and RA characteristics (rheumatoid factor positive, steroid use, DAS28) of MTX- and DMARD-naïve patients were well balanced between treatment groups and consistent with the ITT population; of note, RA duration was shorter in MTX- and DMARD-naïve *vs* the ITT population. Post hoc analysis showed that patients in the MTX- and DMARD-naïve populations achieved efficacy responses comparable with those in corresponding treatment groups of the ITT population at 24 weeks (Table), with a suggestion that these relatively treatment-naïve patients had higher response rates. In all populations, TCZ 8 mg/kg was superior to MTX (mean dose 15.5 mg/week) across all outcomes measured.
Conclusions: TCZ monotherapy was superior to MTX monotherapy after 24 weeks of treatment for RA regardless of the patient's previous exposure to MTX or DMARDs.
Disclosure statement: E.A., F. Hoffmann-La Roche - Employee. M.G., F. Hoffmann-La Roche - Honoraria, Research funding. E.G., F. Hoffmann-La Roche - Employee. G.J., F. Hoffmann-La Roche - Honoraria, Research Funding. All other authors have declared no conflicts of interest.

Efficacy parameters at 24 weeks for the ITT population and the MTX- and DMARD-naïve patients of the AMBITION study

Response rates at 24 weeks, n (%)	MTX (ITT) (n = 284)	TCZ (ITT) (n = 286)	MTX (MTX naïve) (n = 190)	TCZ (MTX naïve) (n = 191)	MTX (DMARD naïve) (n = 129)	TCZ (DMARD naïve) (n = 115)
ACR20	149 (52.5)	200 (69.9)*	102 (53.7)	131 (68.6)*	67 (51.9)	78 (67.8)*
ACR50	95 (33.5)	126 (44.1)*	63 (33.2)	86 (45.0)*	42 (32.6)	56 (48.7)*
ACR70	43 (15.1)	80 (28.0)*	27 (14.2)	52 (27.2)*	19 (14.7)	37 (32.2)*
EULAR good response	48 (16.9) (n = 248)†	115 (40.2) (n = 253)†	32 (16.8) (n = 166)†	84 (44.0)* (n = 170)†	23 (17.8) (n = 109)†	55 (47.8)* (n = 98)†
DAS28 remission	30 (12.1)	85 (33.6)	22 (13.3)	58 (34.1)*	16 (14.7)	39 (39.8)*

*$P < 0.05$ *vs* MTX; †No imputation for missing DAS28 remission was performed.

Concurrent Oral 2 – Case Reports

OP10. PERCUTANEOUS ENDOSCOPIC GASTROSTOMY (PEG) FEEDING: A LIFE-SAVING INTERVENTION IN SYSTEMIC SCLEROSIS-MYOSITIS OVERLAP WITH PHARYNGEAL DYSFUNCTION

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Background: Although dysphagia in patients with systemic sclerosis (SSc) is usually due to lower oesophageal dysmotility, oropharyngeal dysphagia can also occur, especially in patients with myositis overlap. Here we describe 3 patients with SSc and pharyngeal dysphagia, all of