group. In contrast, $TC^{hi}BMI^{lo}$patients had high prevalence of cases with unmeasurable UCP1 expression and higher levels of serum adiponectin (p=0.053) and HDL (p<10–5).
Measurable expression of UCP1 was found in 79%. In total cohort, the patients with measurable UCP1 had higher inflammation and RA activity presented by IL-6 (p=0.0001), IL1b (p=0.037) and DAS28 (p=0.0086), compared to those with no UCP1 expression. $TC^{lo}BMI^{hi}$patients had an overall increase in fat expression of UCP1 (p=0.047) and lowest prevalence of cases with no UCP1 expression (6.2%).
Conclusions: The study shows that UCP-1 expression in subcutaneous fat may be a CV protective mechanism in RA patients. The inflammation seems to be the driving force of UCP1 expression in RA.

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FRI0098 HEPATIC SAFETY IN PATIENTS WITH RHEUMATOID ARTHRITIS WHO RECEIVED ISONIAZID FOR LATENT TUBERCULOSIS: POST-HOC ANALYSIS FROM PHASE 3 BARICITINIB STUDIES

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Background: Baricitinib (BARI) is an oral selective Janus kinase (JAK 1/2)[1] inhibitor approved in the EU, Japan, and other countries for treatment (tx) of moderately to severely active rheumatoid arthritis (RA) in adults. RA therapies may increase risk of tuberculosis (TB).[2] The use of isoniazid (INH) plays a vital role to control TB. However, INH may result in hepatic adverse events (AEs)[3]. Limited data exist on hepatic safety in TB patients (pts) with RA treated with JAK inhibitors and INH.
Objectives: To evaluate the hepatic safety in pts with RA, who were receiving INH for latent TB (LTBI) in BARI phase 3 trials.
Methods: This is a descriptive post-hoc analysis of three phase 3 studies: RA-BEAM, RA-BUILD, and RA-BEACON. All pts were screened for LTBI prior to randomisation. Pts with untreated LTBI and without documentation of prior completed tx, received INH at least for 4 weeks (wk) prior to randomisation and during the clinical trial period. Changes in ALT levels (≥1X,≥3X,≥5X, and ≥10X of ULN) from baseline up to 24 wk were analysed by tx groups (BARI 4 mg, BARI 2 mg, adalimumab [ADA], and placebo [PBO]).
Results: In total, 2516 pts were included in this analysis. Of these, 891 pts were treated with BARI 4 mg, 403 with BARI 2 mg, 330 with ADA, and 892 with PBO. Background csDMARDs, mainly methotrexate (MTX) were continued. Overall, 246 pts reported LTBI at screening across all tx groups. Of these, 169 with confirmed lab data received INH as LTBI tx. At wk 24, ALT≥1X was reported in 24 (41.4%) pts receiving BARI 4-mg+INH. None of the pts in BARI 4-mg+INH reported ALT level of ≥3X,≥5X, and ≥10X ULN. For BARI 2-mg+INH, ALT≥1X reported in 9 (33.3%) pts, ALT≥3X in 2 (7.4%), ALT≥5X in 1 (3.7%), and ALT≥10X in 1 (3.7%) of the pts. Among pts treated with ADA +INH, ALT≥1X was reported in 12 (44.4%), ALT≥3X in 2 (7.4%), ALT≥5X in 1 (3.7%), and ALT≥10X in none of pts. Among pts treated with PBO+INH, ALT≥1X was reported in 21 (36.8%), ALT≥3X and ALT≥5X levels were reported in 2 (3.5%, for both) of the pts. None of the pts reported ALT≥10X. One pt receiving INH in RA-BEAM PBO arm had temporary interruption of tx due to abnormal hepatic lab results. No study tx interruption or discontinuation was reported in INH users in BARI or ADA groups due to abnormal hepatic lab results.

Abstract FRI0098 – Table 1. Changes in ALT level from baseline to week 24 in patients receiving BARI, ADA, and PBO

	BARI 4-mg (RA-BEAM, RA-BUILD, RA-BEACON) N=891		BARI 2-mg (RA-BUILD, RA-BEACON) N=403		ADA (RA-BEAM) N=330		PBO (RA-BEAM, RA-BUILD, RA-BEACON) N=892	
	INH (n=58)	No INH (n=833)	INH (n=27)	No INH (n=376)	INH (n=27)	No INH (n=303)	INH (n=57)	No INH (n=835)
ALT ≥1X ULN	24 (41.4%)	260 (31.2%)	9 (33.3%)	82 (21.8%)	12 (44.4%)	91 (30.0%)	21 (36.8%)	183 (21.9%)
ALT ≥3X ULN	0	13 (1.6%)	2 (7.4%)	6 (1.6%)	2 (7.4%)	9 (3.0%)	2 (3.5%)	13 (1.6%)
ALT ≥5X ULN	0	5 (0.6%)	1 (3.7%)	1 (0.3%)	1 (3.7%)	3 (1.0%)	2 (3.5%)	3 (0.4%)
ALT ≥10X ULN	0	2 (0.2%)	1 (3.7%)	0	0	1 (0.3%)	0	0

ALT=alanine transaminase; ULN=upper limit of normal; INH=isoniazid.
All pts were on concomitant use of csDMARDs, mainly MTX.

Conclusions: The percentage of pts with ≥1X ULN ALT was numerically higher in INH group vs no INH and was consistent across PBO, BARI and ADA tx groups. The data do not suggest an increased hepatic safety risk in pts treated with BARI who were receiving concomitant INH.

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FRI0099 LIVER ENZYME ABNORMALITIES AFTER TOFACITINIB TREATMENT IN PATIENTS WITH HEPATIC STEATOSIS FROM THE RHEUMATOID ARTHRITIS, PSORIATIC ARTHRITIS AND PSORIASIS CLINICAL PROGRAMMES

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Background: Non-alcoholic fatty liver disease, characterised by hepatic steatosis (HS), is a major cause of chronic liver disease in many countries. Limited data are available on liver enzyme elevation in patients (pts) with HS who are receiving medications for inflammatory conditions, such as rheumatoid arthritis (RA), psoriatic arthritis (PsA) and psoriasis (PsO). Tofacitinib is an oral Janus kinase inhibitor for the treatment of RA and PsA, and has also been studied in PsO.
Objectives: To describe baseline characteristics and liver enzyme abnormalities in pts from the tofacitinib RA, PsA and PsO clinical programmes with/without HS at baseline.
Methods: Pts randomised to the tofacitinib (5 or 10 mg twice daily; doses pooled) and placebo arms of 25 studies in the RA, PsA and PsO programmes were

included in this pooled post hoc analysis. Most studies allowed or mandated concomitant treatment with disease-modifying antirheumatic drugs. HS was determined by the investigator and captured per the Medical Dictionary for Regulatory Activities term at baseline. Baseline characteristics, incidence of elevated total bilirubin, aspartate aminotransferase (AST) and alanine aminotransferase (ALT) >1 x and >3 x the upper limit of normal (ULN) up to Month (M) 3, and change from baseline in C-reactive protein (CRP) at M3 – all by HS at baseline – are reported.
Results: A total of 10 212 pts were included in the analysis. The prevalence of HS was 1.6% across indications (RA: 87/6729 [1.3%]; PsA: 27/710 [3.8%]; PsO: 45/2773 [1.6%]). Baseline characteristics were generally similar in pts with or without HS (table 1). However, baseline obesity, diabetes, triglycerides and liver enzymes were numerically higher, and CRP was numerically lower, in pts with HS than in those without HS (table 1). In both tofacitinib- and placebo-treated pts, incidence of elevated total bilirubin, AST and ALT>1 x ULN up to M3 was higher in pts with HS than in those without HS, across indications (table 1). Incidence of elevated total bilirubin, AST and ALT>3 x ULN up to M3 was low across indications, irrespective of HS (table 1). Among tofacitinib-treated pts, CRP was reduced at M3 in pts with or without HS, but to a lesser extent in those with HS, across indications. Among placebo-treated pts, changes in CRP were small, irrespective of HS (table 1).

Abstract FRI0099 – Table 1. Baseline characteristics and liver function up to Month 3, by HS at baseline

	No HS at baseline, all indications (N=10 053)*	HS at baseline, all indications (N=159)*
Age, mean (SD), years	48.9 (13.7)	53.5 (11.0)
Female, n (%)	6723 (66.9)	91 (57.2)
BMI, mean (SD), kg/m²	27.9 (6.6)	32.0 (6.8)
BMI ≥30 kg/m², n (%)	3171 (31.6)	84 (53.2)
Alcohol use, n (%)	2913 (29.7)	52 (33.3)
Diabetes, n (%)	935 (9.3)	37 (23.3)
CRP, mean (SD), mg/L	13.2 (21.0)	10.7 (17.1)
Concomitant NSAID use, n (%)	5658 (56.1)	93 (57.9)
Corticosteroid use, n (%)	3910 (38.9)	54 (34.0)
LDL, mean (SD), mg/dL	113.7 (33.1)	117.1 (36.1)
HDL, mean (SD), mg/dL	56.5 (16.8)	52.4 (15.3)
Triglycerides, mean (SD), mg/dL	131.2 (81.7)	165.8 (96.4)
AST, mean (SD), IU/L	21.9 (13.1)	27.8 (15.0)
ALT, mean (SD), IU/L	23.0 (16.9)	34.0 (19.6)
Gamma-GT, mean (SD), IU/L	31.3 (35.0)	51.8 (77.7)
MTX use at baseline, n (%)	3752 (37.1)	67 (42.1)

Liver function up to Month 3, tofacitinib-treated patients (patients with a post-baseline visit for total bilirubin, AST or ALT)

Indication	RA (N=6490)	PsA (N=689)	PsO (N=2166)	All (N=8115)	RA (N=74)	PsA (N=13)	PsO (N=87)	All (N=124)
ΔCRP, mean (SD) mg/L[Δ]	-12.54 (21.92) (N=4747)	-9.09 (21.48) (N=634)	-4.44 (17.79) (N=701)	-11.15 (20.28) (N=5882)	-9.71 (19.94) (N=67)	-3.40 (9.16) (N=12)	-2.31 (4.75) (N=13)	-7.34 (17.70) (N=92)
Total bilirubin >1x ULN, n (%)	124 (2.3)	12 (2.5)	134 (6.2)	270 (3.3)	4 (5.4)	1 (7.7)	5 (13.5)	10 (8.1)
Total bilirubin >3x ULN, n (%)	5 (0.0)	0 (0.0)	1 (0.1)	1 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)
AST >1x ULN, n (%)	858 (15.8)	86 (18.7)	376 (17.5)	1334 (16.0)	36 (48.7)	5 (38.5)	13 (35.1)	54 (43.6)
AST >3x ULN, n (%)	36 (0.7)	4 (0.9)	10 (0.5)	50 (0.6)	1 (1.4)	0 (0.0)	0 (0.0)	1 (0.8)
ALT >1x ULN, n (%)	1070 (19.5)	120 (26.1)	507 (23.6)	1697 (20.9)	39 (52.7)	9 (69.2)	21 (56.8)	69 (55.7)
ALT >3x ULN, n (%)	73 (1.3)	2 (0.4)	26 (1.2)	101 (1.2)	2 (2.7)	1 (7.7)	0 (0.0)	3 (2.4)

Liver function up to Month 3, placebo-treated patients (patients with a post-baseline visit for total bilirubin, AST or ALT)

Indication	RA (N=1111)	PsA (N=221)	PsO (N=609)	All (N=1841)	RA (N=12)	PsA (N=13)	PsO (N=8)	All (N=34)
ΔCRP, mean (SD) mg/L[Δ]	2.09 (21.17) (N=781)	0.05 (10.93) (N=100)	-1.64 (7.42) (N=131)	1.33 (18.42) (N=1148)	4.43 (15.81) (N=9)	1.56 (5.29) (N=12)	10.79 (33.52) (N=8)	4.26 (15.70) (N=33)
Total bilirubin >1x ULN, n (%)	13 (1.2)	5 (2.3)	24 (4.7)	42 (2.3)	1 (7.7)	1 (7.7)	1 (12.5)	3 (8.8)
Total bilirubin >3x ULN, n (%)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)
AST >1x ULN, n (%)	113 (10.2)	16 (7.2)	48 (9.4)	177 (9.6)	2 (15.4)	2 (15.4)	2 (25.0)	6 (17.7)
AST >3x ULN, n (%)	7 (0.6)	1 (0.5)	1 (0.2)	9 (0.5)	0 (0.0)	1 (7.7)	0 (0.0)	1 (2.9)
ALT >1x ULN, n (%)	165 (14.9)	43 (19.5)	81 (15.9)	289 (15.7)	6 (46.2)	4 (30.8)	3 (37.5)	13 (38.2)
ALT >3x ULN, n (%)	12 (1.1)	1 (0.5)	2 (0.4)	15 (0.8)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)

*Includes patients initially randomised to tofacitinib or placebo; †Patients with both baseline and Month 3 CRP
Δ, change from baseline; ALT, alanine aminotransferase; AST, aspartate aminotransferase; BMI, body mass index; CRP, C-reactive protein; GT, glutamyl transferase;
HDL, high-density lipoprotein; HS, hepatic steatosis; LDL, low-density lipoprotein; MTX, methotrexate; N, number of patients analysed; n, number of patients meeting criteria ; NSAID, non-steroidal anti-inflammatory drug; PsA, psoriatic arthritis; PsO, psoriasis; RA, rheumatoid arthritis; SD, standard deviation; ULN, upper limit of normal

Conclusions: In this exploratory analysis, prevalence of HS at baseline was 1.6% across the tofacitinib RA, PsA and PsO programmes. After up to 3 months of tofacitinib treatment, incidence of mildly elevated liver enzymes was higher in pts with HS than in those without HS. Incidence of severely elevated liver enzymes was low overall, and similar in pts with or without HS. Further studies are needed to evaluate the effects of tofacitinib on CRP and liver enzymes, and the potential impact on clinical response, in pts with RA, PsA or PsO who have comorbid HS.
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Rheumatoid arthritis – biological DMARDs

FRI0100 MULTI-OMICS ANALYSIS IDENTIFIES A GENE SIGNATURE ASSOCIATED WITH THE CLINICAL RESPONSE TO ANTI-TNF THERAPY IN RHEUMATOID ARTHRITIS

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Background: Tumour Necrosis Factor (TNF) inhibitors have improved the management of many patients with rheumatoid arthritis (RA). However,~30% of anti-TNF treated patients do not show a significant clinical improvement. To date, little is known on the biological mechanisms underlying the differential response to anti-TNF agents.
Objectives: We sought to identify genetic variation associated with the anti-TNF response in RA using a sequential multi-omics approach.
Methods: First, we aimed to identify gene coexpression modules (GCMs) associated with anti-TNF response. For this objective, we extracted the RNA from synovial biopsies of 13 RA patients starting anti-TNF therapy and determined the expression profiles using Illumina microarrays. GCMs were identified using the WGCNA approach. The association between GCMs and anti-TNF response was performed using the eigengene of each GCM. Clinical response was defined using the EULAR criteria at week 14. To analyse the association of GCMs with anti-TNF response at the genetic level, we used 348 anti-TNF treated RA patients from Spain. The statistical analysis was performed using GWAS data and the set-based test in PLINK. The GCMs that were significantly associated with anti-TNF response were subsequently tested for validation in an independent cohort of 2706 anti-TNF treated RA patients. The functional implication of the validated GCMs was studied via pathway and cell type epigenetic enrichment analyses.
Results: We identified 148 GCMs in the RA synovium. From these, 15 GCMs were found to be associated with anti-TNF response ($p<0.05$). At the genetic level, we found two of the 15 GCMs to be associated with the adalimumab (ADL) and infliximab response ($p<0.05$) in the Spain cohort. In the independent cohort, we replicated the association of the GCM associated with ADL response ($p=0.01$). The validated GCM was found to be enriched in genes that participate in the nucleotides metabolism ($p=2.41e-5$). The epigenetic analysis revealed that ADL-associated variants are enriched in epigenetic marks from immune cell types like Tregs ($p=0.04$).
Conclusions: Our study shows the existence of a drug-specific genetic basis for the anti-TNF response. Therefore, this molecular diversity should be considered for biomarker research in RA.
Disclosure of Interest: None declared
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FRI0101 UNMET NEEDS IN THE TREATMENT OF RHEUMATOID ARTHRITIS. AN OBSERVATIONAL STUDY AND A REAL-LIFE EXPERIENCE FROM A SINGLE UNIVERSITY CENTRE

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Background: Despite the progress in the treatment of rheumatoid arthritis (RA), a significant number of patients does not achieve low disease activity (LDA).
Objectives: The purpose of this study was to estimate the size of unmet needs in the treatment of RA, using all the conventional synthetic disease modifying antirheumatic drugs (csDMARDs) and/or biological DMARDs (bDMARDs) in a long-term observational study.
Methods: Between January 2006 and December 2017, 538 patients with early RA were followed up in the outpatient rheumatology clinic. All patients fulfilled the 2010 ACR/EULAR classification criteria, had disease duration less than 1 year and were csDMARDs and bDMARDs naive. The patients were treated according to EULAR and ACR recommendations and strategies for RA. The following