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 ($\geq 1X$, $\geq 3X$, $\geq 5X$, and $\geq 10X$ 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 $\geq 1X$ 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 $\geq 3X$, $\geq 5X$, and $\geq 10X$ ULN. For BARI 2-mg+INH, ALT $\geq 1X$ reported in 9 (33.3%) pts, ALT $\geq 3X$ in 2 (7.4%), ALT $\geq 5X$ in 1 (3.7%), and ALT $\geq 10X$ in 1 (3.7%) of the pts. Among pts treated with ADA+INH, ALT $\geq 1X$ was reported in 12 (44.4%), ALT $\geq 3X$ in 2 (7.4%), ALT $\geq 5X$ in 1 (3.7%), and ALT $\geq 10X$ in none of pts. Among pts treated with PBO+INH, ALT $\geq 1X$ was reported in 21 (36.8%), ALT $\geq 3X$ and ALT $\geq 5X$ levels were reported in 2 (3.5%, for both) of the pts. None of the pts reported ALT $\geq 10X$. 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 $\geq 1X$ 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 $\geq 3X$ ULN	0	13 (1.6%)	2 (7.4%)	6 (1.6%)	2 (7.4%)	9 (3.0%)	2 (3.5%)	13 (1.6%)
ALT $\geq 5X$ ULN	0	5 (0.6%)	1 (3.7%)	1 (0.3%)	1 (3.7%)	3 (1.0%)	2 (3.5%)	3 (0.4%)
ALT $\geq 10X$ 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 $\geq 1X$ 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