severity of depression by HRDS (p=0.0098), and the index of personal anxiety (P=0.009) in group 1 decreased. In the 2nd group above mentioned parameters have not changed. On the 1 group data of SF-36 evaluation: the physical health component has improved - the increase of RP and BP 57.4% and 37.8% from the baseline; vital activity and role functioning due to emotional state, have also increased by 35.6% and 43.5%, respectively. In the 2nd group (n=22) investigated parameters have not undergone significant changes in dynamics. In the 1 receiving melatonin group TJC and SJC have decreased by 15% and 22% (p=0.0079, p=0.0022, respectively) and their dynamics in the 2nd group was less significant (p=0.013 and p=0.017, respectively). Also, patients in group 1 have highly significant (p<0.001) reduction in the severity of morning stiffness and joint pain, and in the 2nd group the changes were less significant (respectively, p=0.043, p=0.016). Positive dynamics of CRP in group 1 was more significant (p=0.003), than it was in 2 patients' group (p=0.033).

Conclusions: In the group of patients treated with melatonin was noted improvement in general condition (a significant improvement in the parameters of the physical components of health, reduction of depressive and psycho-vegetative disorders) and also more significant decrease of the intensity of pain and of morning stiffness duration, of TJC and SJC, than in not treated with melatonin patients. Inclusion of Melatonin in the comprehensive PsA therapy promotes not only reduction of depression symptoms and sleep disorders, but also reduces the severity of the chronic pain manifestations and, consequently, improves the quality of life of patients with this disease

Disclosure of Interest: None declared

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FRI0496 COMPARING TOFACITINIB SAFETY PROFILE IN PATIENTS WITH PSORIATIC ARTHRITIS IN CLINICAL STUDIES WITH REAL-WORLD DATA

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Background: Tofacitinib is an oral Janus kinase inhibitor under investigation for the treatment of psoriatic arthritis (PsA). Two Phase 3 studies have been completed (NCT01877668; NCT01882439) and a long-term extension (LTE) study is ongoing (database not locked; NCT01976364).

Objectives: To compare incidence rates (IR) for adverse events (AEs) of special interest in a tofacitinib cohort from the Phase 3 PsA trials with real-world experience in a comparison cohort from the US Truven MarketScan database.

Methods: The tofacitinib cohort included adult patients (pts) from 2 Phase 3 studies with ≥6 months PsA diagnosis who met ClASsification of Psoriatic ARthritis (CASPAR) criteria, had active plaque psoriasis, and active arthritis (≥3 swollen and ≥3 tender/painful joints) and who were treated with tofacitinib. Pts were grouped by those who received tofacitinib 5 (N=238) or 10 mg (N=236) twice daily (BID) in the 2 Phase 3 studies, and all pts who received ≥1 dose of tofacitinib in the 2 Phase 3 studies or the LTE (tofacitinib all doses, N=783). The comparison cohort (N=5799) comprised pts with moderate to severe PsA, defined by ≥1 inpatient or ≥2 outpatient 696.0 diagnosis codes on 2 unique calendar days (≥1 by a rheumatologist) between Oct 2010 and Sep 2015, initiating therapy with a systemic agent for PsA. Key Phase 3 study exclusion criteria were applied to the comparison cohort. IRs for serious infection events (SIEs), herpes zoster (HZ), malignancies (excluding non-melanoma skin cancer [NMSC]), NMSC and major adverse cardiovascular events (MACE) were compared.

Results: Mean age, gender and diabetes history were generally similar between the tofacitinib and comparison cohorts (48.7–49.5 years, 42.4–49.2% male, 12.2–15.7% with diabetes history). Overall more pts treated with tofacitinib had prior experience with corticosteroids (15.7–28.2%), conventional synthetic disease-modifying antirheumatic drugs (100%) and tumour necrosis factor inhibitors (48.1–55.9%) vs the comparison cohort (11.9%, 46.6% and 36.6%, respectively). IRs for SIEs were lower for the tofacitinib vs the comparison cohort (Table 1). The tofacitinib cohort had a higher rate of HZ vs the comparison cohort (Table 1). IRs for malignancies and MACE were similar between cohorts (Table 1).

Conclusions: IRs of AEs of special interest reported in tofacitinib PsA Phase 3 studies were generally comparable to those in a general PsA population comprising pts receiving a range of biologic agents, except HZ, which was higher for pts treated with tofacitinb but similar to the incidence observed with tofacitinib treatment in other indications.

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Table 1. Incidence rates (95% CI)[a] [PY exposure] for adverse events of special interest

		SIEs[b]	HZ	Malignancies[c]	NMSC	MACE
Tofacitinib cohort[d]	Tofacitinib 5 mg BID (N=238)	1.30 (0.16, 4.69) [154]	1.96 (0.41, 5.74) [153]	NR	NR	NR
	Tofacitinib 10 mg BID (N=236)	2.00 (0.41, 5.83) [150]	2.66 (0.73, 6.81) [150]	NR	NR	NR
	Tofacitinib all doses (N=783)	NR	NR	0.63 (0.21, 1.48) [791]	0.51 (0.14, 1.30) [789]	0.38 (0.08, 1.11) [791]
Comparison cohort	Any bDMARD (N=5075)	5.02 (4.19, 5.97) [2569]	1.26 (0.91, 1.70) [3343]	0.51 (0.34, 0.74) [5499]	1.40 (1.10, 1.75) [5488]	0.38 (0.22, 0.61) [4468]
	Any bDMARD + csDMARD (N=2542)	5.10 (3.83, 6.66) [1058]	1.53 (0.94, 2.37) [1303]	0.40 (0.16, 0.82) [1751]	1.79 (1.21, 2.53) [1736]	0.25 (0.07, 0.64) [1591]
	Any TNFi (N=4617)	5.13 (4.26, 6.11) [2419]	1.26 (0.90, 1.71) [3181]	0.51 (0.33, 0.74) [5144]	1.39 (1.09, 1.76) [5098]	0.41 (0.24, 0.65) [4183]
	Any TNFi + csDMARD (N=2383)	5.12 (3.83, 6.72) [1015]	1.51 (0.91, 2.36) [1257]	0.42 (0.17, 0.86) [1670]	1.75 (1.17, 2.52) [1656]	0.26 (0.07, 0.67) [1520]
	Adalimumab (N=1934)	4.16 (3.00, 5.63) [1009]	1.16 (0.65, 1.91) [1297]	0.48 (0.23, 0.88) [2095]	1.40 (0.94, 2.01) [2070]	0.41 (0.16, 0.84) [1724]
	Etanercept (N=1412)	4.82 (3.37, 6.67) [747]	1.10 (0.55, 1.97) [1000]	0.41 (0.16, 0.84) [1720]	1.46 (0.95, 2.16) [1709]	0.30 (0.08, 0.76) [1343]
	Infliximab (N=615)	8.91 (6.09, 12.57) [359]	1.94 (0.93, 3.57) [516]	1.21 (0.55, 2.30) [743]	1.35 (0.65, 2.48) [741]	0.47 (0.10, 1.37) [638]
	Golimumab (N=389)	3.49 (1.40, 7.19) [201]	1.16 (0.24, 3.39) [258]	0.00 (0.00, 0.90) [410]	0.99 (0.27, 2.53) [404]	0.91 (0.19, 2.67) [328]
	Certolizumab (N=267)	6.80 (2.74, 14.02) [103]	0.91 (0.02, 5.06) [110]	0.00 (0.00, 2.09) [176]	1.72 (0.35, 5.02) [175]	0.00 (0.00, 2.44) [151]
	Apremilast (N=617)	5.34 (3.56, 9.82) [187]	2.62 (0.85, 6.13) [191]	1.14 (0.24, 3.35) [262]	3.45 (1.58, 6.56) [261]	0.00 (0.00, 1.60) [231]

[a]Patients with event per 100 PY; [b]SIEs: any infection requiring parenteral antimicrobial therapy or hospitalisation or if the infection met criteria for a serious adverse event in the tofacitinib cohort; infections requiring parenteral antimicrobial treatment and hospitalisation in the comparison cohort; [c]All malignancies excluding non-melanoma skin cancer; [d]Tofacitinib 5 and 10 mg BID rows include patients randomised to these doses, respectively, in the two Phase 3 studies (12 or 6 months' duration), tofacitinib all doses row includes patients who received ≥1 dose of tofacitinib in the two Phase 3 studies or the LTE. IR estimates include events occurring ≤28 days after last dose of study drug (or to data cut-off in the LTE). Exposure (PY) is the total follow-up time to the day of the first event within the event-counting period for patients with events, or the last dose day plus a risk period of 28 days after the last dose (or to data cut-off for LTE) for patients without events. bDMARD, biologic disease-modifying antirheumatic drug; BID, twice daily; CI, confidence interval; csDMARD, conventional synthetic disease-modifying antirheumatic drug; HZ, herpes zoster; LTE, long-term extension study; MACE, major adverse cardiovascular event; NMSC, non-melanoma skin cancer; NR, not reported; PY, patient-years; SIE, serious infection event; TNFi, tumour necrosis factor inhibitor.

FRI0497 WHAT CHOICES DO RHEUMATOLOGIST MAKE IN ESCALATING DMARD THERAPY IN EARLY PSA?

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Background: Psoriatic arthritis (PsA) is a multifaceted disease.

Objectives: We aimed to evaluate change in medication over time guided by joints, skin, enthesis, low back pain and dactylitis in newly diagnosed PsA patients

Methods: Newly diagnosed PsA patients were included in the Dutch Early south-west Psoriatic Arthritis cohoRt (DEPAR) study between August 2013 and March 2016. Initial drug treatment and escalation of therapy were described for all patients. Drivers of treatment changes in the first year were evaluated by mixed

Table 1 Multivariable mixed effects ordered ordinal logistic regression of treatment change over time in early PsA

	Odds Ratio	95% CI	
Covariates			
Swollen joint count (per joint)	**1.14**	**1.06**	**1.24**
Tender joint count(per joint)	0.98	0.94	1.02
Tender entheses (per enthesis)	1.06	0.94	1.19
BASDAI (per point)	1.05	0.97	1.15
Dactylitis Index (per point)	**1.05**	**1.00**	**1.09**
timepoints (baseline reference)			
3 months	**1.55**	**1.01**	**2.37**
6 months	1.25	0.78	2.02
9 months	**0.45**	**0.25**	**0.81**
12 months	1.52	0.79	2.91
initial starting values (no dmard reference)			
other sdmards	0.05	-0.52	0.62
methotrexate oral	0.49	-0.08	1.07
methotrexate subcutaneous	4.67	3.95	5.38
leflunomide	5.32	4.57	6.08
biological dmards*	6.23	5.42	7.05
mixed effects parameters			
random slope	0.02	0.01	0.06
random intercept	8.30	6.12	11.25

in bold **p<0.05**; *biological dmards were TNF inhibitors