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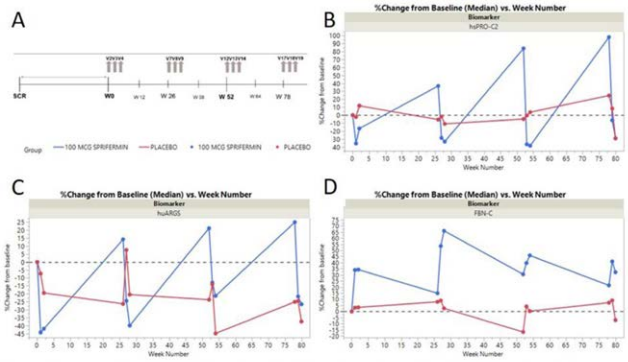

Fig. Assessment of cartilage markers in synovial fluid at the 4 cycles. A) Study overview. B) type II collagen formation, PRO-C2. C) Aggrecan degradation, huARGS. D) Fibronectin degradation, FBN-C. Data are shown as median of available samples at each timepoint.

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OP0190 UNDERSTANDING CURRENT PRESCRIPTION DRUG TREATMENT PARADIGMS FOR PATIENTS WITH OSTEOARTHRITIS IN EUROPE

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Background: Joint pain is the most prevalent symptom for sufferers of osteoarthritis (OA). Pharmacological management of OA is restricted by limited efficacy and considerable toxicity, with growing fears about opioid use.
Objectives: To understand the current real-world prescribed drug treatment paradigm related to OA disease severity for patients in 5 EU countries; France, Germany, Italy, Spain and the UK.
Methods: Data were drawn from the Adelphi OA Disease Specific Programme (2017-18), a point-in-time study of physicians and their patients. Physicians classified their patients as currently having mild, moderate or severe disease severity, and provided details on currently prescribed OA therapy and physician satisfaction with therapy, rated from very satisfied to very dissatisfied. Patients were excluded from these analyses if they suffered from back and neck OA only, and shoulder OA that had not been diagnosed by X-ray. Comparisons among disease severity groups were made using analysis of variance and chi-squared tests.
Results: The study included 489 physicians (primary care physicians, rheumatologists, orthopaedists) reporting on 3596 of their OA patients: 24% mild (n=874), 53% moderate (n=1904), and 23% severe (n=818). Overall, 73% patients were prescribed at least one drug for their OA (65% of mild; 76% of moderate; 77% of severe patients [<0.001]). Paracetamol (34%) was the most commonly prescribed OA treatment. NSAIDs (31%) and opioids (27%) were also frequently prescribed treatments, and worsening severity was associated with an increase in opioid use (11% of mild; 26% of moderate, 47% of severe patients [<0.001]), but not NSAID (Table 1). The mean number of prescription medications increased (0.9 for mild; 1.4 for moderate; 1.6 for severe patients [<0.001]) and physician satisfaction with treatment decreased (86% for mild; 70% for moderate; 41% for severe [<0.001]) with worsening OA disease severity.

Table 1. Prescribed treatment by physician-reported OA severity

	Mild (n=874)	Moderate (n=1904)	Severe (n=818)
Current class of medication prescribed for OA, n (%)			
Paracetamol	186 (21.3)	663 (34.8)	313 (38.3)
NSAIDs	267 (30.5)	605 (31.8)	237 (29.0)
Any opioid	93 (10.6)	501 (26.3)	386 (47.2)
Weak opioid	82 (9.4)	407 (21.4)	255 (31.2)
Strong opioid	11 (1.3)	99 (5.2)	146 (17.8)
Opioid + analgesic (combined)	6 (0.7)	15 (0.8)	7 (0.9)
Corticosteroid	31 (3.5)	150 (7.9)	92 (11.2)
Glycosaminoglycan	50 (5.7)	149 (7.8)	62 (7.6)
Viscosupplement	12 (1.4)	93 (4.9)	42 (5.1)
Number of currently prescribed drug classes, mean (SD)	0.9 (0.8)	1.4 (1.1)	1.6 (1.2)

Conclusion: Physicians reported decreasing satisfaction with treatment for their OA patients as disease severity increased, despite increasing use of opioids and numbers of classes of prescribed drugs.
Disclosure of Interests: Philip G Conaghan Consultant of: AbbVie, BMS, Eli Lilly, EMD Serono, Flexion Therapeutics, Galapagos, GSK, Novartis, Pfizer, Speakers bureau: AbbVie, Eli Lilly, Novartis, Pfizer, Lucy Abraham Shareholder of: Pfizer, Employee of: Pfizer, Peita Graham-Clarke Shareholder of: Eli Lilly and Co, Employee of: Eli Lilly and Co, Lars Viktrup Shareholder of: Eli Lilly and Company, Employee of: Eli Lilly and Company, Joseph C Cappelleri Shareholder of: Pfizer Inc, Employee of: Pfizer Inc, Craig Beck Shareholder of: Pfizer, Employee of: Pfizer, Andrew G Bushmakin Shareholder of: Pfizer Inc, Employee of: Pfizer Inc, Niall Hatchell: None declared, Emily Clayton: None declared, James Jackson: None declared
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OP0191 ASSOCIATION OF TRAMADOL WITH ALL-CAUSE MORTALITY, CARDIOVASCULAR DISEASE, VENOUS THROMBOEMBOLISM AND HIP FRACTURES AMONG PATIENTS WITH OSTEOARTHRITIS. A POPULATION-BASED STUDY

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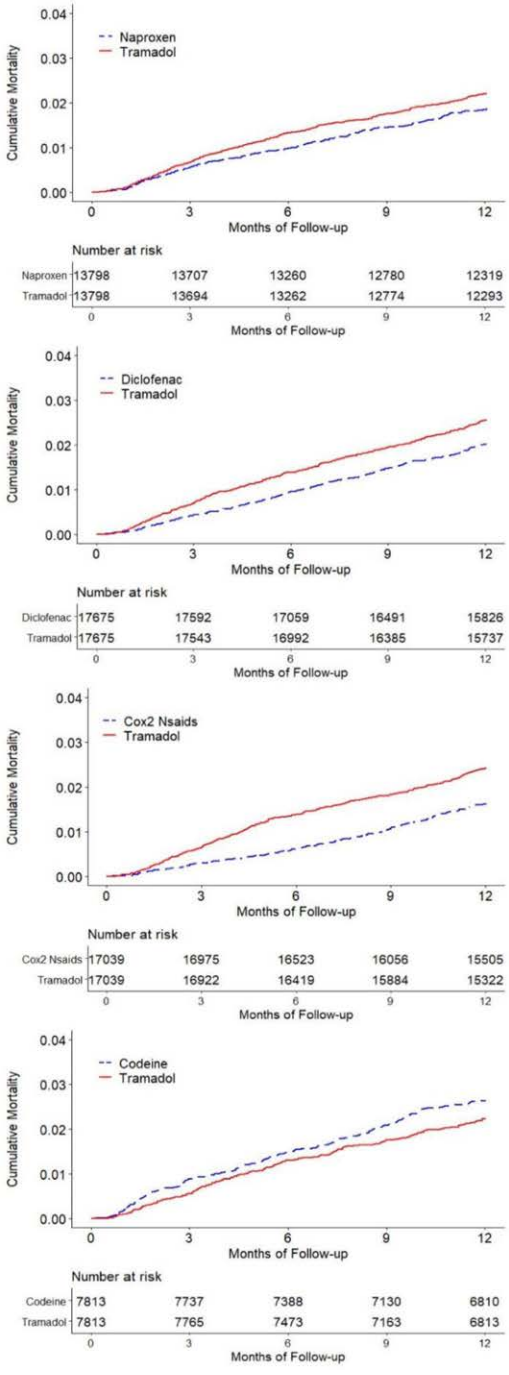
Background: Both tramadol (narcotic-like drug) and nonsteroidal anti-inflammatory drugs (NSAIDs) are prescribed for pain relief among osteoarthritis (OA) patients. Evidence comparing risks of adverse events between tramadol and NSAIDs users is inconclusive.
Objectives: To examine the association of tramadol with all-cause mortality, cardiovascular disease (CVD), venous thromboembolism (VTE) and hip fractures (HFx) compared with NSAIDs and codeine in OA.
Methods: Design: Sequential propensity score-matched cohort study. Sample: All patients with OA who received medical care from 2005 to 2014 in the entire province of British Columbia, Canada. Tramadol cohort: Initial prescription of tramadol (n=56325). Four comparator cohorts: the initiation of one of the following: naproxen (n=13798), diclofenac (n=17675), cyclooxygenase-2 [Cox-2] inhibitor (n= 17039), or codeine (a weak opioid) (n=7813). Patients required to be prescribed neither tramadol nor its comparators during the year before the initial prescription date (i.e., index date). Outcomes: 1) all-cause mortality; first ever 2) CVD, 3) VTE, 4) HFx within the 1st year after the initiation of tramadol or its comparators. Follow-up: from index date until the event occurred, disenrollment, or the end of a 1-year follow-up period. Statistical analysis: We created baseline covariates (demographics, comorbidities, medications and health resource utilization) from the year prior to the index date. Calendar years from 2005 to 2014 were divided into 10 blocks; propensity scores were calculated using logistic regression within each block. We used 1:1 greedy matching method. We estimated hazard ratios (HRs) using Cox proportional hazard models.
Results: After propensity score matching, 112650 patients with OA were included (mean age of 68 years, 62.8% were females). During the 1-year follow-up, 296 deaths (21.5/1000 person-years) occurred in the tramadol cohort and 246 (17.8/1000 person-years) in the naproxen cohort (Table 1). All-cause mortality was higher for tramadol compared with all NSAIDs cohorts, but not with the codeine cohort (Table 1, Figure 1). Tramadol initiators have also a higher risk of CVD and VTE compared with the diclofenac and Cox-2 inhibitor initiators with HRs ranging from 1.2 to 1.7. Furthermore, tramadol was also associated with a higher risk of HFx compared with all NSAIDs cohorts (HRs ranging from 1.4 to 1.5). No significant difference was found between tramadol and codeine (Table 1).
Conclusion: OA patients initiating tramadol have an increased risk of mortality, CVD, VTE, and HFx within 1 year compared with NSAIDs, but no statistically significant difference in the risk was observed between tramadol and codeine.

Table 1

		Group1		Group2		Group3		Group4	
		Tramadol	Naproxen	Tramadol	Diclofenac	Tramadol	Cox-2 inhibitor	Tramadol	Codeine
All-cause Mortality	OA (n)	13798	13798	17675	17675	17039	17039	7813	7813
	Death (n)	296	246	439	345	402	267	168	199
	Rate (/1000 PY)	21.5	17.8	24.8	19.5	23.6	15.7	21.5	25.5
	HR (95% CI)	1.2 (1.0-1.4)	1.0	1.3 (1.1-1.5)	1.0	1.5 (1.3-1.8)	1.0	0.8 (0.7-1.0)	1.0
CVD	OA (n)	11708	11708	14924	14924	14779	14779	6809	6809
	CVD (n)	309	319	410	349	404	353	156	164
	Rate (/1000 PY)	26.4	27.3	27.5	23.4	27.3	23.9	22.9	24.1
	HR (95% CI)	1.0 (0.9-1.1)	1.0	1.2 (1.1-1.3)	1.0	1.2 (1.0-1.3)	1.0	0.9 (0.8-1.1)	1.0
VTE	OA (n)	13472	13472	17230	17230	16699	16699	7660	7660
	VTE (n)	41	37	60	40	70	40	28	30
	Rate (/1000 PY)	3.0	2.8	3.5	2.3	4.2	2.4	3.7	3.9
	HR (95% CI)	1.2 (0.9-1.6)	1.0	1.5 (1.1-1.9)	1.0	1.7 (1.3-2.3)	1.0	1.0 (0.7-1.4)	1.0
HFx	OA (n)	13378	13378	17216	17216	16670	16670	7593	7593
	HFx (n)	66	49	88	59	91	60	35	40
	Rate (/1000 PY)	5.0	3.7	5.1	3.4	5.5	3.6	4.6	5.3
	HR (95% CI)	1.4 (1.0-1.8)	1.0	1.5 (1.2-1.9)	1.0	1.5 (1.2-1.9)	1.0	0.9 (0.7-1.2)	1.0



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OP0192

PSORIATIC ARTHRITIS IS ASSOCIATED WITH A METABOLICALLY ADVERSE BODY COMPOSITION PROFILE PREDICTIVE OF GREATER CHD AND TYPE 2 DIABETES RISK – MRI FINDINGS FROM THE IMAPA AND UK BIOBANK STUDIES

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Background: Increased Body Mass Index (BMI) is associated with Psoriatic Arthritis (PsA) but with uncertain pathophysiological significance. BMI does not reflect body fat distribution, but fat storage site is important as increased ectopic fat including visceral adipose tissue (VAT), liver fat, and muscle fat infiltration (MFI), are associated with increased type 2 diabetes and coronary heart disease (CHD) risk[1]. To date no study has compared detailed body composition in PsA with the general population and other metabolic diseases.
Objectives: 1. To characterize the body composition profile of PsA compared to age, sex, and BMI-matched metabolic disease free (MDF) individuals, and type 2 diabetes. 2. To relate body composition to risk of type 2 diabetes and CHD in PsA versus MDF controls.
Methods: MRI body composition profiles were available for 29 PsA participants in the IMAPA study[2]. After excluding 3 participants with concomitant type 2 diabetes, body composition was compared in 26 PsA participants with 130 age, sex, and BMI-matched healthy MDF controls (matched 1:5) and 454 individuals with type 2 diabetes from UK Biobank, using Wilcoxon signed-rank test. Analyses were repeated adjusted for age, sex, and BMI. The propensity of PsA patients to develop CHD or type 2 diabetes based on their body composition profile was compared to that of matched MDF controls.
Results: PsA participants had significantly more ectopic fat including greater visceral adipose tissue (VAT) volume and liver fat percentage compared to MDF controls (table 1, figure 1A). This difference persisted after adjustment for age, sex, and BMI. Individuals with PsA shared a similar body composition to type 2 diabetes (table 1, figure 1B). Body composition-predicted propensity for CHD or type 2 diabetes was 1.3 and 1.8 times higher, respectively, for PsA compared to matched MDF controls.

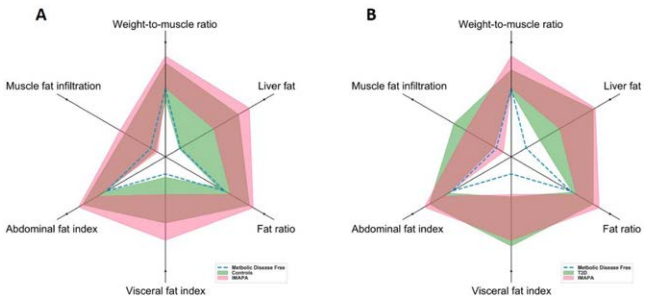

Figure 1. Body Composition Profiles of IMAPA PsA participants (pink) versus A. UK Biobank matched MDF controls (green), and B. type 2 diabetes (T2D) (green).