Table 1. Mean (SD) outcome scores by treatment type

	Advanced therapies N=65	Other therapies N=274	Not treated N=608
SF-36 MCS	38.0 (11.0)	40.1 (11.6)	39.8 (10.5)
SF-36 PCS	36.0 (9.9)	37.6 (9.6)	43.9 (8.4)
PHQ-9 total score[a]	7.7 (7.0)	9.1 (6.8)	7.8 (7.1)
WPAI domain scores[b]			
% work missed	26.8 (29.7)	27.8 (35.4)	20.8 (27.8)
% impairment at work	55.8 (31.1)	42.8 (29.6)	44.5 (29.3)
% overall work impairment	61.4 (34.2)	49.0 (33.2)	51.1 (32.6)
% activity impairment	62.0 (26.1)	57.8 (26.5)	48.3 (28.9)

[a]N = 22, 45, 137 for advanced therapies, other therapies, not treated, respectively

[b]N for WPAI % work missed = 33, 113, 338, respectively for advanced therapies, other therapies, not treated; N for WPAI % impairment at work and overall work impairment = 33, 99, 328, respectively, for advanced therapies, other therapies and not treated

MCS, mental component summary; PCS, physical component summary; PHQ-9, Patient Health Questionnaire-9; SD, standard deviation; SF-36, Short Form-36 health survey; WPAI, Work Productivity and Activity Impairment questionnaire

treatment. Regardless of treatment group, pts reported >20% work loss and >45% work impairment. Among treated pts, >50% reported moderate or severe PsA, suggesting a need for overall better management of PsA to reduce the disease impact and improve quality of life. Our results are limited by self-reported PsA diagnosis, which may differ from physician-reported PsA diagnosis, and the survey being conducted in the EU only, which may differ from other parts of the world. Further statistical analyses are needed to determine differences between groups and correlation to other health indicators.

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FRI0485 IN PERIPHERAL PSORIATIC ARTHRITIS DKK-1 AND PTH ARE LOWER THAN IN RHEUMATOID ARTHRITIS AND HEALTHY CONTROLS

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Background: The recent characterization of the canonical WNT pathway in the regulation of bone modeling and remodeling provided important insights for our understanding of the pathophysiology of bone involvement in chronic arthritis [1]. Dkk-1 and sclerostin are the main regulators of WNT/b-catenin signaling, regulating both bone formation and resorption [2]. In a previous our study we showed that in patients with Rheumatoid Arthritis (RA) Dkk-1 is significantly increased and associated with the presence of typical erosions and lower BMD [3].

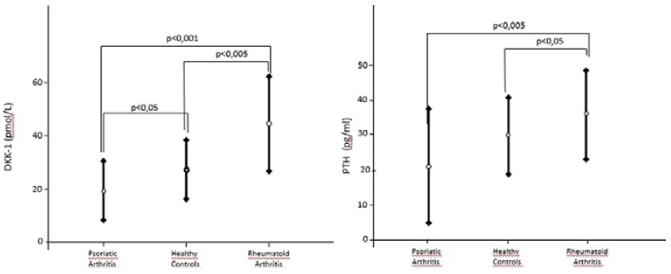
Objectives: we decided to perform this study in order to compare the serum levels of WNT-pathway regulators alongside bone turnover markers (BTM) and Parathyroid Hormone (PTH) between a group of female patients with PsA and healthy controls (HC) or patients with Rheumatoid Arthritis (RA).

Methods: this is a cross-sectional study including 18 patients with PsA classified with the CASPAR criteria, 35 HC, and 28 patients with RA classified with the ACR/EULAR 2010 criteria. Intact N-propeptide of type I collagen (PINP), C-terminal telopeptide of type I collagen (CTX-I), Dickkopf-related-protein 1 (Dkk-1), sclerostin, PTH and 25OH-Vitamin D serum levels were dosed. All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. Informed consent was obtained from all individual participants included in the study.

Results: the PsA group showed significantly lower Dkk-1 levels when compared to the HC and RA groups. Dkk-1 in the RA group was also significantly higher than in the HC group. A similar trend was documented also for PTH, however a statistically significant difference was observed only when we comparing the PsA vs RA group (table 1, figure 1). No other statistically significant differences in the other markers were found.

Table 1. Values of bone turnover markers (CTX-I, PINP), Dkk-1 and sclerostin of PsA, RA patients and control group (mean ± SD)

	PsA	RA	HC	P (ANOVA)
PINP ng/ml	42,80±16,670	39,19±21,38	42,49±11,52	NS
CTX-I ng/ml	0,21±0 ,17	0,32±0,21	0,28±0,10	NS
Dkk-1 pmol/l	19,45±11,30	44,51±17,81	27,29±11,48	<0,001
Sclerostin pmol/l	30,82±11,25	30,75±10,25	34,23±17,29	NS
PTH pg/ml	21,12±16,63	35,83±13,02	29,69±11,43	<0,005



Conclusions: this study demonstrated for the first time that Dkk-1 levels in PsA are lower than HC, in contrast with RA where they are higher. These results might contribute to explain the different bone involvement of the two different diseases.

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FRI0486 INTRAVENOUS GOLIMUMAB IN ADULT PATIENTS WITH ACTIVE PSORIATIC ARTHRITIS: EFFICACY AND SAFETY THROUGH WEEK 24

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Objectives: The GO-VIBRANT study was designed to evaluate the safety and efficacy of intravenous (IV) golimumab (GLM) in adult patients (pts) with active PsA (biologic-naïve).

Methods: GO-VIBRANT is a Phase 3, multicenter, randomized, double-blind, placebo (PBO)-controlled trial. Biologic-naïve active PsA pts were randomized (1:1) to IV GLM 2mg/kg at weeks (wk) 0, 4, and every 8 wks thereafter or PBO at wks 0, 4, 12, and 20 with crossover to GLM at wk24. The primary endpoint was ACR20 response at wk14. Multiplicity-controlled endpoints were ACR50, ACR70, PASI 75, change from baseline in HAQ-DI, enthesitis, dactylitis, SF-36 PCS/MCS scores at wk14; and ACR50 and change from baseline in total modified vdH-S (structural damage) score at wk24. Efficacy analyses were based on randomized treatment. Adverse events (AE) through wk24 are reported here. Investigators remain blinded through wk60.

Results: 480 pts were randomized (PBO: 239; GLM: 241). The study met its primary and all controlled secondary endpoints. At wk14, significantly greater proportions of GLM pts vs PBO achieved ACR20 (75.1% vs. 21.8%). Also, GLM treatment resulted in significant change from baseline HAQ-DI score (-0.60 vs. -0.12), ACR50 (43.6% vs. 6.3%), PASI 75 (59.2% vs. 13.6%), ACR70 (24.5% vs. 2.1%), change from baseline in enthesitis and dactylitis scores (-1.8 vs. -0.8 and -7.8 vs. -2.8, respectively), and change from baseline in SF-36 PCS and SF-36 MCS scores (8.65 vs. 2.69 and 5.33 vs. 0.97, respectively) (all p<0.001) at wk14. At wk24, significantly greater proportions of GLM pts vs. PBO pts achieved ACR 50 (53.5% vs. 6.3%, p<0.001). At wk24, there was significantly less progression of structural damage for GLM pts vs PBO as measured by change from baseline in total modified vdH-S score (-0.36 vs. 1.95; p<0.001). ACR20 was significantly higher with GLM than PBO as early as wk2 (45.6% vs. 7.5%; p<0.001). 27.0% of GLM pts (vs. 4.2% PBO) achieved Minimal Disease Activity by wk14. Due to the difference in response rates in GLM vs. PBO treated pts, the number needed to treat for ACR20 at wk14 was 1.9 in a post-hoc analysis (Table). Through wk24,