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].

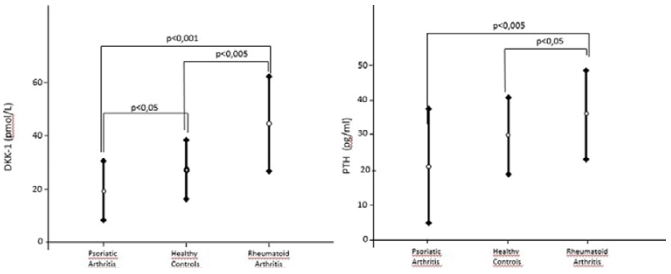
Objectives: we decided to perform this study in order to compare the serum levels of WNT-pathway regulators alongside bone turnover markers (BTM) and Parathyroid Hormone (PTH) between a group of female patients with PsA and healthy controls (HC) or patients with Rheumatoid Arthritis (RA).

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,

46.3% of GLM pts and 40.6% of PBO pts had ≥1 AE; 2.9% vs. 3.3% of pts, respectively, had ≥1 serious AE. Two deaths, 2 malignancies, and 1 demyelinating event were reported. The most common treatment-emergent type of AE was infection (20.0% of GLM pts vs. 13.8% of PBO pts). No opportunistic infection or tuberculosis was reported through wk24. The rate of infusion reactions was low at <2%; none was serious or severe.

Table. Clinical Response

	Placebo	Golimumab 2 mg/kg	P-values
Patients randomized, n	239	241	
Clinical efficacy at wk14			
ACR20/50/70, (%)	21.8/6.3/2.1	75.1/43.6/24.5	p<0.001
PASI 75, n (%)*	27/198 (13.6%)	116/196 (59.2%)	p<0.001
Change from BL in HAQ-DI (n) Mean (SD)	222 -0.12 (0.47)	233 -0.60 (0.53)	p<0.001
Change from BL in enthesitis** (n) Mean (SD)	173 -0.8 (1.98)	182 -1.87 (1.75)	p<0.001
Change from BL in dactylitis** (n) Mean (SD)	115 -2.8 (7.03)	130 -7.8 (8.57)	p<0.001
Minimal Disease Activity, n/N (%)	10/239 (4.2%)	65/241 (27.0%)	p<0.001
Number Needed to Treat (95% CI)		1.9 (1.64, 2.18)	
Clinical efficacy at Week 24			
ACR50, n (%)	15 (6.3%)	129 (53.5%)	
Imaging data at Week 24			
Change from BL in vdH-S score (N) Mean (SE)	237 1.95 (0.264)	237 -0.36 (0.144)	p<0.001
HRQoL at wk14			
Change from BL in SF-36 PCS score (n) Mean (SD)	222 2.69 (5.92)	233 8.65 (7.60)	p<0.001
Change from BL in SF-36 MCS score (n) Mean (SD)	222 0.97 (7.64)	233 5.33 (9.95)	p<0.001

*Among pts with ≥3% BSA involvement, **Among pts with finding at baseline, BL:baseline

Conclusions: In pts with active PsA, IV GLM demonstrated significant and clinically meaningful improvements of disease activity and physical function, skin psoriasis clearance, HRQoL, reduction in dactylitis and enthesitis, and inhibition of structural damage progression. GLM was well-tolerated through wk24; the safety profile was consistent with other anti-TNF therapies, including SC GLM.

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FRI0487 APREMILAST IS ASSOCIATED WITH LONG-TERM (4-YEAR) DAS-28 (CRP) REMISSION AND IMPROVEMENTS IN SKIN DISEASE: RESULTS FROM A PHASE III STUDY IN DMARD/BIOLOGIC-EXPERIENCED PATIENTS WITH ACTIVE PSORIATIC ARTHRITIS

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Background: Treatment goals for long-term control of skin and joint symptoms in active psoriatic arthritis (PsA) include clinically important changes in DAS-28 (CRP), achievement of remission in DAS-28 (CRP), reduction in swollen joint count (SJC), and decrease in skin disease.[1] PALACE 3 included PsA patients with active joint disease and an active skin lesion at the time of enrollment.

Objectives: Report the impact of apremilast (APR) on PsA manifestations over 4 years.

Methods: Patients were stratified by baseline (BL) DMARD use (yes/no) and psoriasis involvement of the body surface area (<3%/≥3%) and randomized (1:1:1) to placebo (PBO), APR 30 mg BID (APR30), or APR 20 mg BID (APR20). After the 24-week PBO-controlled phase, all patients were treated with APR30 or APR20 and could enroll in the long-term extension. Efficacy assessments were conducted through Week 208.

Results: 505 patients were randomized and received ≥1 dose of study medication (PBO: n=169; APR30: n=167; APR20: n=169). A total of 91% (227/249) of patients starting the fourth year of APR therapy completed the Week 208 visit. Patients treated with APR30 demonstrated sustained decreases in disease activity at Week 208, as shown by mean change from BL in DAS-28 (CRP) of –1.66; 80.3% achieved good/moderate EULAR response and 50.4% achieved DAS-28 (CRP) remission. Sustained effect on inflammation at Week 208 was also demonstrated by mean/median percent changes in SJC, a marker of inflammatory activity, of –77.4%/–100.0% (Table); 64.8% of patients had an SJC of 0 or 1. Decreases in disability and maintenance of functionality were shown by sustained improvements in Health Assessment Questionnaire-Disability Index (HAQ-DI) scores (Table). A continued effect on skin disease was shown by decreases in skin involvement, as measured by the Psoriasis Area and Severity Index (PASI); 54.7% of APR30 patients had BL PASI >5 and 27.3% had BL PASI >10; at Week 208, 64.5% had PASI <3 and 77.4% had PASI ≤5. PASI-75 and PASI-50 response rates also demonstrated clinically significant relief (Table). In patients treated with APR20, similar findings were observed at Week 208. No new safety concerns were identified through 208 weeks of APR30 therapy. During Weeks >156 to ≤208 of APR30 exposure, the only adverse event (AE) occurring in ≥5% of patients was nasopharyngitis; most AEs were mild or moderate in severity. Serious AEs occurred in 7.2% of APR30 patients over Weeks >156 to ≤208, similar to rates in earlier study periods. Few discontinuations due to AEs (0.7%) occurred over Weeks >156 to ≤208. The APR20 safety profile was similar to that of APR30.

Outcomes at Week 208	
	APR30 n=129*
DAS-28 (CRP), mean change	−1.66
DAS-28 (CRP) <2.6, n/m (%)	64/127 (50.4)
SJC, mean/median % change	−77.4/−100.0
TJC, mean/median % change	−64.4/−86.6
HAQ-DI (0-3), mean change	−0.42
HAQ-DI MCID ≥0.30/≥0.35, n/m (%)	63/129 (48.8)
ACR20, n/m (%)	85/128 (66.4)
ACR50, n/m (%)	51/128 (39.8)
ACR70, n/m (%)	31/127 (24.4)
PASI-75, n/m (%)§	28/62 (45.2)
PASI-50, n/m (%)§	42/62 (67.7)

Data as observed. *The n reflects the number of patients treated with APR30, regardless of when APR was started (BL, Week 16, or Week 24) and who had data available at Week 208; actual number of patients available for each end point may vary. §Examined among patients with psoriasis involvement of the body surface area ≥3% at BL. APR30=apremilast 30 mg BID; DAS-28=28-joint count Disease Activity Score; CRP=C-reactive protein; SJC=swollen joint count; TJC=tender joint count; HAQ-DI=Health Assessment Questionnaire-Disability Index; MCID=minimal clinically important differences; ACR20/50/70=20%/50%/70% improvement in modified American College of Rheumatology response criteria; n/m=number of responders/number of patients with sufficient data for evaluation; PASI-75/50=≥75%/≥50% reduction from BL Psoriasis Area and Severity Index score; BL=baseline.

Conclusions: Over 208 weeks, APR demonstrated sustained and clinically important improvements in PsA signs and symptoms, including physical function and associated psoriasis, among patients continuing the study. APR was generally well tolerated with an acceptable safety profile.

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FRI0488 PSORIATIC ARTHRITIS IN THE UNITED STATES: INCREASING ALL-CAUSE HOSPITALIZATIONS 1993–2014

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Background: Psoriatic arthritis (PsA) is a rare disease, with an estimated prevalence of 0.02 – 0.42% in Europe and US (1). PsA is often regarded as a mild disease, but recent data suggest an increase in comorbidities and mortality, possibly related to systemic inflammation (1).

Objectives: To study all-cause hospitalizations in patients with PsA in the United States (US) from 1993 to 2014.

Methods: The Nationwide Inpatient Sample (NIS) is a stratified random sample of all US community hospitals. It is the only US national hospital database with information on all patients, regardless of payer, including persons covered by Medicare, Medicaid, private insurance, and the uninsured. We examined all inpatient hospitalizations in NIS from 1993 to 2014 with a primary or secondary diagnosis of PsA, and compared them to total all-cause US hospitalizations during the same period. US population estimates and projections for the resident US population were obtained from the US Census Bureau.

Results: There were 789.8 million all-cause hospitalizations in 6.4 billion person-years of observation from 1993 to 2014 (123.4 hospitalizations per 1,000 person-years). During this time-period, 332,496 hospitalizations occurred in patients with PsA (5.2 per 100,000 person-years). All-cause US hospitalizations increased from 33.7 million in 1993 to 35.4 million in 2014, an increase of 4.8% over 22 years (Figure, dotted blue line). All-cause hospitalizations in PsA patients have increased from 6,866 in 1993 (2.6 per 100,000 person-year) to 33,875 in 2014 (10.6 per 100,000 person-years, a dramatic increase of over 393% (p<0.0001, Figure solid red line). In 2014, hospitalizations in PsA patients accounted for 163,630 hospital days at a total national cost of over US$1.66 billion.

Conclusions: All-cause hospitalizations in patients with PsA in the US have significantly increased by 393% in the last 22 years, almost 80-fold of the 4.8% increase in US population all-cause hospitalization rate in the same time-period. This calls for an increase need for identification and management of serious co-morbid conditions in patients with PsA.