10). SEC was used by 18% (n=19), and MTX by 42% (n=43, 25% sc, 13% oral, 4% unknown), respectively. Eight percent had a combination therapy of MTX and SEC. Twenty-seven percent used GC and/or other biologics/conventional disease modifying antirheumatic drugs (DMARDs). Mean glucocorticoid cumulative dose (GCCD) was 12.8±22.0g. Patients with SEC showed a significantly longer disease duration (median: 24 years vs. 13 years) compared to MTX, but showed no other differences in baseline-characteristics or risk factors. T-Scores of both femora were significantly higher in the MTX versus the SEC group. We could not find significant differences between these groups with regard to physical activity, back pain, movement restriction, fracture rates or GCCD. Twenty-five percent of the MTX users and 27% of the patients in the SEC group additionally had GC while; in contrast to no patient in the combination group.

Conclusion: The prevalence of osteoporosis in patients with PSO or PSOA was found to be as high as in the normal population. However, there was a high frequency of peripheral fragility, but not vertebral fractures. Patients with PSO or PSOA patients treated with SEC had a longer disease duration and lower hip BMD, but showed no differences in back pain, physical activity or movement restrictions compared to MTX users.

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FRI0434 THE RELATIONSHIP BETWEEN SYMPTOMS OF AUTONOMIC DYSFUNCTION AND CARDIOVASCULAR INVOLVEMENT IN PSORIATIC ARTHRITIS

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Background: The incidence of cardiovascular disease (CVD), diabetes mellitus, metabolic syndrome and subclinical atherosclerosis is markedly increased in patients with psoriatic arthritis (PsA). The autonomic nervous system is the visceral nervous system of the body consisting of two parts; sympathetic and parasympathetic. Patients with PsA have predominantly parasympathetic involvement autonomic nervous system.

Objectives: The aim of this study was to evaluate the symptoms of autonomic dysfunction and their relationship with cardiovascular involvement and other clinic parameters in patients with PsA.

Methods: The study included patients diagnosed with PsA according to the CASPAR criteria. For evaluation of cardiovascular involvement, body mass index (BMI), abdominal obesity, hypertension (HT), diabetes mellitus (DM), hyperlipidemia (HL), metabolic syndrome, fasting glucose levels, lipid levels, systolic and diastolic blood pressures (SBP-DBP) were assessed. DAPSA (Disease Activity in Psoriatic Arthritis), BASDAI (Bath Ankylosing Spondylitis Disease Activity Index), Leeds enthesitis index, Psoriasis Area Severity Index (PASI), Psoriatic Arthritis Quality of Life (PsAQoL) and Health Assessment Questionnaire (HAQ) were used to assess patients clinical situations. The Composite Autonomic Symptom Score (COMPASS-31) (range:0-100) consisting of 6 subdivisions including orthostatic, vasomotor, secretomotor, gastrointestinal (GIS), bladder and pupillomotor was used for the symptoms of autonomic dysfunction. The Mann-Whitney U-test, student's t-test and Spearman's correlation coefficient were used for statistical analysis. P<0.05 was considered statistically significant.

Results: A total of 64 subjects (43 female, 21 male) with a mean of age 49 years (SD:12.3) and disease duration of 59 months (SD:71.3) were recruited into the study. The patients had HT (23.4%), DM (17.2%), abdominal obesity (62.5%), metabolic syndrome (45.3%) and dyslipidemia (42.2%). The mean total COMPASS-31 score was 19.7 (SD:8.3). There was no significant difference in COMPASS-31 scores in patients with or

without HT, DM, dyslipidemia. Bladder scores were significantly higher in patients with abdominal obesity and metabolic syndrome (p<0.05). GIS and pupillomotor scores were significantly higher in patients with enthesitis (p<0.05). Significant correlations were found between: LDL and bladder (r=0.392) scores; enthesitis and GIS (r= 0.303), pupillomotor (r=0.365) scores; DAPSA and total COMPASS-31 (r=0.310), secretomotor (r=0.359) scores; BASDAI and total COMPASS-31 (r=0.483), GIS (r=0.327), secretomotor (r=0.309), pupillomotor (r=0.302) scores; fatigue and total COMPASS-31 (r=0.503), GIS (r=0.377), pupillomotor (r=0.302) scores; HAQ and total COMPASS-31 (r=0.476), orthostatic (r=0.388), bladder (r=0.371) scores; PsAQoL and total COMPASS-31 (r=0.601), orthostatic (r=0.549), secretomotor (r=0.414), pupillomotor (r=0.380) scores.

Conclusion: The total score of COMPASS-31 and its subdivisions were high in PsA patients as compared with literature data on healthy subjects. The symptoms of autonomic dysfunction were increased in PsA patients. Disease activity, functional impairment, fatigue, LDL and quality of life are associated with autonomic dysfunction. Autonomic symptoms improve with disease control.

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FRI0435 INFLUENCE OF BASELINE DEMOGRAPHICS AND DISEASE CHARACTERISTICS ON EFFICACY OF AN ORAL, SELECTIVE TYK2 INHIBITOR, BMS-986165, IN PATIENTS WITH PLAQUE PSORIASIS IN A PHASE 2 TRIAL

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Background: BMS-986165 is an oral, selective inhibitor of tyrosine kinase 2 (TYK2), an enzyme that activates signal transducer and activator of transcription (STAT)-dependent cytokine signalling pathways involved in the pathophysiology of psoriasis (PsO). In a 12-week, Phase 2 trial of BMS-986165 in patients with moderate to severe plaque PsO, including those with baseline (BL) musculoskeletal symptoms, Psoriasis Area and Severity Index (PASI) 75 responses (primary endpoint) were highest at doses ≥3 mg twice daily (BID; 67–75%) vs placebo (7%; P<0.001), with a favourable safety profile.¹

Objectives: To evaluate the influence of BL demographics (weight, body mass index, age) and disease characteristics (age of onset, presence of musculoskeletal symptoms, disease duration, previous biologic use, PASI score, static Physician Global Assessment [sPGA] score, Dermatology Life Quality Index [DLQI] score) on Week 12 efficacy for the 3 most effective doses of BMS-986165 (3 mg BID, 6 mg BID, and 12 mg once daily [QD]) in the trial.

Methods: Adults with moderate to severe plaque PsO (body surface area [BSA] ≥10%, PASI score ≥12, sPGA score ≥3) were randomised equally to 1 of 5 oral doses of BMS-986165 (3 mg every other day, 3 mg QD, 3 mg BID, 6 mg BID, 12 mg QD) or placebo for 12 weeks.

Results: A total of 267 patients were treated; subgroup analyses based on BL characteristics are reported for patients treated with the most effective doses of BMS-986165 (≥3 mg BID; n=134). BMS-986165 showed no meaningful differences in efficacy among almost all of the 3 subgroups, including age of onset, presence of musculoskeletal symptoms, and disease duration (Table), with some variability across subgroups. Small patient numbers may underlie observed fluctuations in results. Similar consistency in responses was seen regardless of BL age (18–<45 years, n=66; ≥45 years, n=68), weight

(<90 kg, n=81; ≥90kg, n=53), PASI score (<20, n=96 vs ≥20, n=38), DLQI score (<10, n=52; 10–<20, n=62; ≥20, n=20), sPGA score (<4, n=89; ≥4, n=44), and BSA (<20, n=73; ≥20, n=61).

Conclusion: In patients with moderate to severe plaque PsO, BMS-986165 demonstrated consistent efficacy at doses ≥3 mg BID regardless of many BL characteristics, including presence of musculoskeletal symptoms, that may impact results. These observations will be evaluated further in ongoing Phase 3 studies (POETYK PSO Phase 3 programme; ClinicalTrials.gov identifiers: NCT03611751 and NCT03624127).

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Table 1. Efficacy of BMS-986165: endpoints at Week 12 by select baseline disease characteristics

Endpoint	Dose	Age at onset			Musculoskeletal symptoms		Disease duration	
		<18 years	18–45 years	≥45 years	No	Yes	<15 years	≥15 years
PASI 75	3 mg BID	n=13 75.9 (46.2–95.0)	n=21 71.4 (47.0–98.7)	n=11 54.5 (23.4–85.3)	n=34 64.7 (46.5–80.3)	n=11 61.8 (48.2–97.7)	n=25 72.0 (50.6–97.9)	n=20 60.0 (40.8–84.6)
	6 mg BID	n=8 70.0 (24.7–75.3)	n=5 66.7 (56.4–91.0)	n=3 67.6 (9.4–89.2)	n=34 67.6 (49.5–82.6)	n=11 63.6 (30.0–91.1)	n=23 65.2 (41.1–86.1)	n=23 65.2 (42.7–83.6)
	12 mg QD	n=9 77.8 (40.0–97.2)	n=29 75.9 (56.5–99.7)	n=29 66.7 (22.3–98.7)	n=29 75.9 (56.5–99.7)	n=15 73.3 (44.9–92.2)	n=13 61.5 (31.6–86.1)	n=31 80.6 (62.5–92.5)
	Combined*	n=38 65.9 (48.6–80.4)	n=76 75.0 (63.7–84.2)	n=20 60.0 (36.1–80.9)	n=97 69.1 (58.9–78.1)	n=37 73.0 (55.9–86.2)	n=69 69.3 (55.0–79.7)	n=74 71.6 (59.9–81.5)
	3 mg BID	n=13 59.8 (25.1–80.8)	n=21 62.9 (21.8–86.0)	n=11 36.4 (10.9–69.2)	n=34 47.1 (29.8–64.9)	n=11 36.4 (10.9–69.2)	n=25 48.0 (27.8–69.7)	n=20 40.0 (19.1–63.9)
PASI 90	3 mg BID	n=16 18.8 (4.0–45.6)	n=5 40.0 (40.0–79.8)	n=6 33.3 (0.8–90.6)	n=34 44.1 (27.2–62.1)	n=11 45.5 (16.7–76.6)	n=22 34.5 (32.2–76.6)	n=23 34.8 (16.4–57.3)
	6 mg BID	n=8 44.4 (13.2–79.8)	n=29 48.3 (29.4–87.5)	n=29 16.7 (0.8–64.1)	n=29 44.8 (26.4–64.1)	n=15 40.0 (16.3–67.7)	n=13 45.0 (5.0–53.8)	n=31 43.2 (33.1–69.8)
	12 mg QD	n=9 36.9 (13.2–79.8)	n=29 48.3 (29.4–87.5)	n=29 16.7 (0.8–64.1)	n=29 44.8 (26.4–64.1)	n=15 40.0 (16.3–67.7)	n=13 45.0 (5.0–53.8)	n=31 43.2 (33.1–69.8)
	Combined*	n=38 26.8 (13.2–79.8)	n=76 48.3 (29.4–87.5)	n=20 16.7 (0.8–64.1)	n=97 45.4 (26.4–64.1)	n=37 40.0 (16.3–67.7)	n=69 45.0 (32.2–76.6)	n=74 43.2 (33.1–69.8)
	3 mg BID	n=16 18.8 (4.0–45.6)	n=5 40.0 (40.0–79.8)	n=6 33.3 (0.8–90.6)	n=34 44.1 (27.2–62.1)	n=11 45.5 (16.7–76.6)	n=22 34.5 (32.2–76.6)	n=23 34.8 (16.4–57.3)
sPGA 0/1	3 mg BID	n=16 56.3 (29.9–80.2)	n=29 64.6 (47.0–82.7)	n=29 72.7 (39.0–94.0)	n=34 67.6 (49.5–82.6)	n=11 63.6 (30.0–91.1)	n=25 68.0 (46.5–95.1)	n=20 60.0 (40.8–84.6)
	6 mg BID	n=8 56.3 (29.9–80.2)	n=29 64.6 (47.0–82.7)	n=29 72.7 (39.0–94.0)	n=34 67.6 (49.5–82.6)	n=11 63.6 (30.0–91.1)	n=25 68.0 (46.5–95.1)	n=20 60.0 (40.8–84.6)
	12 mg QD	n=9 59.9 (40.0–97.2)	n=29 64.6 (47.0–82.7)	n=29 72.7 (39.0–94.0)	n=34 67.6 (49.5–82.6)	n=11 63.6 (30.0–91.1)	n=25 68.0 (46.5–95.1)	n=20 60.0 (40.8–84.6)
	Combined*	n=38 59.9 (40.0–97.2)	n=76 64.6 (47.0–82.7)	n=20 72.7 (39.0–94.0)	n=97 67.6 (49.5–82.6)	n=37 63.3 (44.9–92.2)	n=69 68.0 (55.0–79.7)	n=74 60.0 (40.8–84.6)
	3 mg BID	n=16 56.3 (29.9–80.2)	n=29 64.6 (47.0–82.7)	n=29 72.7 (39.0–94.0)	n=34 67.6 (49.5–82.6)	n=11 63.6 (30.0–91.1)	n=25 68.0 (46.5–95.1)	n=20 60.0 (40.8–84.6)

Data are shown as response rate, % (95% exact CI).
Data for the clinical aim are as follows:
Age at onset: <18 years (n=14); PASI 75, 0.0 (0.0–23.2); PASI 90, 0.0 (0.0–23.2); 18–45 years (n=72); PASI 75, 11.1 (2.4–29.2); PASI 90, 3.7 (0.1–16.0); sPGA 0/1, 11.1 (2.4–29.2); ≥45 years (n=41); PASI 75, 0.0 (0.0–40.2); PASI 90, 0.0 (0.0–40.2); sPGA 0/1, 0.0 (0.0–40.2).
Musculoskeletal symptoms: No (n=24); PASI 75, 12.5 (2.7–32.4); PASI 90, 4.2 (0.1–21.1); sPGA 0/1, 12.5 (2.7–32.4); Yes (n=21); PASI 75, 0.0 (0.0–16.1); PASI 90, 0.0 (0.0–16.1); sPGA 0/1, 0.0 (0.0–16.1).
Disease duration: <15 years (n=16); PASI 75, 6.3 (0.2–30.2); PASI 90, 0.0 (0.0–20.6); sPGA 0/1, 12.5 (1.6–38.3); ≥15 years (n=29); PASI 75, 6.9 (0.8–22.8); PASI 90, 3.4 (0.1–17.8); sPGA 0/1, 3.4 (0.1–17.8).
*BMS-986165 3 mg BID, 6 mg BID, and 12 mg QD combined.
BID=twice daily; QD=once daily; CI=confidence interval; PASI 75=75% improvement in Psoriasis Area and Severity index score; PASI 90=90% improvement in Psoriasis Area and Severity index score; QD=once daily; sPGA 0/1=static Physician Global Assessment score of 0 or 1.