Late-Breaking Abstracts

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LBA-1 Genetics of VPS13A disease (chorea-acanthocytosis) in Puerto Rico
C. Alicea-Malave, L. Surillo-Dahdah, A. Velayos-Baeza, S. Ayala-Peña, C. Serrano, M. Barret, S. Berman, A. Lasker, R. Walker *(Manati, PR, USA)*

LBA-2 Estrogens in women with PSP in Argentina and North America
B. Couto, O. Gershanik, I. Litvan, A. Lang, C. Tartaglia, S. Fox, M.E. Gonzalez Toledo, L. Brolese, M. Rossi, C. Marras *(Buenos Aires, Argentina)*

LBA-3 Cross-species discovery of a serum-to-CNS link showing the neural impact of moderate intensity aerobic exercise (mAE) on Parkinson's disease resilience and recovery
V. Nejtek, M. Salvatore, J. Richardson, G. Boehm, H. Alphonso *(Fort Worth, TX, USA)*

LBA-4 Pimavanserin in Parkinson's Disease Psychosis: An Exploratory Post Hoc Delayed-Start Analysis
W. Ondo, Z. Mari, P. Zhang, L. Chrones, G. Brunson *(Houston, TX, USA)*

LBA-5 Analyzing the Effect of Language Barriers on Patient Communication for Recruitment and Retention in Parkinson's Disease Clinical Trials
I. Saraf, M. Park, J. Sanderson, F. Farrokhi, E. Tamadonfar, E. Bayram, M. Bruno, C. Tanner, N. Chunga Iturry, S. Mantri, C. Vila, R. Amin, K. Williams, S. Aslam, R.P. Vasireddy, P. Auinger, M. Gross, M. Afshari, C. Branson, S. Amundsen-Huffmaster, R. Camicioli, I. Subramanian, J. Jimenez-Shahed, P. Agarwal *(Kirkland, WA, USA)*

LBA-6 Effect of CREXONT® (IPX203, ER CD-LD) on sleep in patients with Parkinson's disease and associated sleep disturbances
O. Vaou, S. Allard, G. Banisadr, A. Morris, S. Fisher, R. Hauser *(San Antonio, Texas, USA)*

LBA-7 Initiation and titration of continuous subcutaneous apomorphine infusion (CSAI) in the United States: Early data from the Clinical Nurse Navigator (CNN) Program
C. Happel, A. Formella, M. Grall *(Rockville, MD, USA)*

LBA-8 Task-Specific Tremor and Parkinson's Disease: A DaTscan and alpha-synuclein biopsy study
A. Achuthaprasad, W.G. Ondo *(Houston, TX, USA)*

LBA-9 Left-DLPFC overactivation reflects reduced neural efficiency during dual-task gait in Parkinson's disease
G. Venas Santos, P. Rodrigues da Silva, L. Bitarães Rodrigues Santos, L. de Mattos Aranha, J.R. Sato, F.A. dos Santos Mendes, M.E. Pimentel Piemonte *(São Paulo, Brazil)*

LBA-1 Genetics of VPS13A disease (chorea-acanthocytosis) in Puerto Rico

C. Alicea-Malave, L. Surillo-Dahdah, A. Velayos-Baeza, S. Ayala-Peña, C. Serrano, M. Barret, S. Berman, A. Lasker, R. Walker *(Manati, PR, USA)*

Objective: To describe the genetics of VPS13A disease (chorea-acanthocytosis) in Puerto Rico.

Background: VPS13A is a rare neurodegenerative disease caused by bi-allelic pathogenic variants in the VPS13A gene. Recurrent pathogenic variants are unusual, and typically have been observed only in specific populations, e.g. the "Ehime" mutation in Japan. This disorder has been reported worldwide, however, we observed an apparently higher prevalence of VPS13A disease among people from Puerto Rico.

Background: VPS13A is a rare neurodegenerative disease caused by bi-allelic pathogenic variants in the VPS13A gene. Recurrent pathogenic variants are unusual, and typically have been observed only in specific populations, e.g. the "Ehime" mutation in Japan. This disorder has been reported worldwide, however, we observed an apparently higher prevalence of VPS13A disease among people from Puerto Rico.

Method: Data were collected from all patients in Puerto Rico or with Puerto Rican ancestry with a diagnosis of VPS13A disease for whom genetic and/or molecular confirmation was available.

Results: We have identified 17 patients from 14 families with a genetic and/or molecular diagnosis of VPS13A disease. Genetic data were available for 12 probands; 3 were homozygous, and 9 compound heterozygous. Three recurring variants were identified; c.1400-1401 del, which occurred in 4 alleles (3 patients); c. 3556_c.3557 dup, which occurred in 7 alleles (6 patients); and c.3889C>T, which occurred in 7 alleles (6 patients). At least one of the 3 alleles was present in each of the 12 patients. There were 4 other variants which occurred once, and one patient in whom a 2nd pathogenic variant has not yet been detected. (One additional pathogenic variant was from a non-Puerto Rican parent.)

Conclusion: The 3 recurrent variants can be considered as "founder mutations" and have likely been present in the population for many years. These variants have also been reported from patients in other countries, including in other Hispanic populations (Mexico, Spain). Of the 4 singly-occurring variants, 3 appear to be unique to Puerto Rico. The relatively high number of founder variants is likely a major driver of the large number of affected subjects from this small population. The population of Puerto Rico is 3.2 million, with an additional almost 6 million people of Puerto Rican ancestry living on the mainland US. We are aware of a number of additional patients previously diagnosed by clinical characteristics, thus the prevalence in this population may be several-fold higher than in other regions which is estimated at 1:1,000,000.

LBA-2 Estrogens in women with PSP in Argentina and North America

B. Couto, O. Gershanik, I. Litvan, A. Lang, C. Tartaglia, S. Fox, M.E. Gonzalez Toledo, L. Brolese, M. Rossi, C. Marras *(Buenos Aires, Argentina)*

Objective: To describe women's reproductive health factors in women with PSP from North America and Argentina

Background: The relation of estrogen exposure and Parkinson's disease has been inconclusive with inconsistent associations of PD risk and age at menopause or younger age at menarche. In women with progressive supranuclear palsy (WwPSP), only the ENGENE study reported a lower risk of PSP in women who used estrogen replacement therapy (ERT) during perimenopause.

Method: In a pooled sample of women from Argentina (n 16), United States (multicenter US-ENGENE study, n 150), and Canada (n 66) we describe the estrogen exposure composite (EEC), composed by the influence of endogenous estrogens: early menarche (younger than 10.5), late menopause (<52 \ if natural menopause, (<46 if surgical menopause), or presence of surgical menopause; and external estrogens: use of replacement therapy. Comparisons were done with 166 healthy women as controls.

Results: There were no differences in age, but women with PSP (WwPSP) had less formal education than controls. Neither endogenous nor exogenous estrogen variables were different between WwPSP and controls, neither was the EEC. Compared with the Argentinean WwPSP, in the North American cohort there was longer disease duration (t=17; p<.001) and less disease severity (t=-7; p<.001). The estrogen composite score (t=4.2; p<.001) and use of ERT (t=2.43; p<.01) were higher for North American WwPSP than Argentinean.

Conclusion: In this multi-cohort study, estrogen exposure of WwPSP does not differ from healthy controls. The differences in progression and disease course (i.e. severity and disease duration) between North American and Argentinean WwPSP may be related to a differential combined estrogen exposure throughout life. Those associations merit further study, and potential investigation of specific disease biomarkers.

LBA-3 Cross-species discovery of a serum-to-CNS link showing the neural impact of moderate intensity aerobic exercise (mAE) on Parkinson's disease resilience and recovery

V. Nejtek, M. Salvatore, J. Richardson, G. Boehm, H. Alphonso *(Fort Worth, TX, USA)*

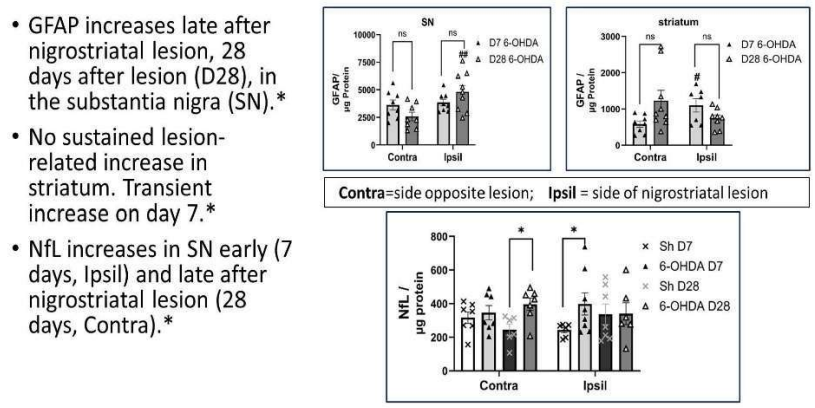
Objective: To illustrate that effective mAE is significantly related to neuroprotection in the substantia nigra that can be identified with GFAP, NfL, and UCH-L1 and validated with motor, and cognitive tests.

Background: Parkinson disease (PD) is diagnosed after ~80% of brain receptors affecting dopamine (DA) levels are lost requiring dopamine replacement therapy (DRT) with complex dosing and adverse side-effects.1-6 An alternative to DRT, we discovered the underlying neurobiological mechanisms of effective moderate intensity aerobic exercise (mAE) centered on the substantia nigra rather than striatum. Now we further discovered specific serum biomarkers of effective mAE in substantia nigra tissue collected at 7-days and 28-days illustrates disease resilience in aged PD model rats.

Methods: Glial fibrillary acidic protein (GFAP) and neurofilament lite (NfL) were examined in aged 6-OHDA lesioned rats randomized to receive mAE or No-exercise. As ~80% of neurons are lost in the striatum by the time a patient receives a PD diagnosis, it is clinically relevant to illustrate serum GFAP and NfL levels signify disease progression in the rat striatum and neuroprotection in the substantia nigra signifying resilience to progression and recovery of motor and cognitive function are possible in human PD.

Results: We found GFAP increases at day 28 post-lesion in the substantia nigra, but no such increase in the striatum (p = 0001). Furthermore, we found mAE resulted in significant motor recovery (e.g. further distance traveled [t=4.45, ***p<0.001, df =16], forepaw adjustment steps [F(3, 47) = 5.52, p = 0.003] than no-exercise (NE) rats which also coincided with lower glial fibrillary acidic protein (GFAP). We also found (1) NfL increased in substantia nigra at 7-days post-lesion with additional increases at day 28 without increases in the striatum (p = 001); (2) ubiquitin carboxy-terminal hydrolase L1 (UCH-L1) increases in substantia nigra at day 7 and increases more at day 28 while the dopamine-regulating enzyme tyrosine hydroxylase decreases (p = 009). UCH-L1 also correlates with dopamine tissue in substantia nigra (p = .0002). In this cross-species study, early-stage idiopathic PD human subjects participating in mAE (3x/wk for 30-45-mins, >3-mos) had lower GFAP: [F(3,75)=6.57, p=0.001], and higher UCH-L1 [F(3,74)=5.45, p=0.001] and walked further distance in the 6-minute walk test (6MWT) [F(1,79)= 9.33, p=.00], than No-exercisers (NE) [F(1,77)= 9.32, p=.00].

Conclusion: Effective mAE is significantly related to neuroprotection in the substantia nigra that can be identified with GFAP, NfL, and UCH-L1 and validated with motor, and cognitive tests.



LBA-4 Pimavanserin in Parkinson's Disease Psychosis: An Exploratory Post Hoc Delayed-Start Analysis
W. Ondo, Z. Mari, P. Zhang, L. Chrones, G. Brunson *(Houston, TX, USA)*

Objective: Investigate whether initiating pimavanserin for Parkinson's disease psychosis (PDP) earlier (PDP duration <6 mo) may slow the progression of psychotic symptoms.

Background: Determining the impact of pimavanserin on the progression of psychotic symptoms in PDP could inform timing of treatment initiation. A previous exploratory analysis suggested earlier initiation may be associated with increased response [1]; here we investigate the impact on disease progression.

Methods: This exploratory analysis of a double-blind-to-open-label rollover study using pivotal trial data included 171 patients with PDP receiving 34-mg/d pimavanserin for up to 10 weeks (early-start) or placebo for 6 weeks followed by 34-mg/d pimavanserin for 4 weeks (delayed-start). Participants were stratified by PDP duration (<6 or ≥6 mo) at treatment initiation (not powered to detect differences; P values nominal). The main exploratory endpoint defined a positive result as demonstration of superiority of early-start vs delayed-start in estimates of mean change in the total SAPS-PD score between baseline (week 0) and week 10. Secondary exploratory endpoints assessed symptom progression, defined as an increase in SAPS-PD score per week (rate of change, slope) between weeks 0-6 and weeks 6-10.

Results: In patients with PDP duration <6 mo (n=21), LS mean (SE) change in SAPS-PD scores from weeks 0-10 (main endpoint) was −8.25 (1.70) in early-start and −2.28 (2.22) in delayed-start groups (treatment difference, −5.97; 95% CI: −11.95, 0.01; P=0.0505). For PDP duration ≥6 mo (n=150), the treatment difference was 0.14 (95% CI: −2.22, 2.49; P=0.9080) [figure1][table1]. In PDP duration <6 mo, treatment differences between early-start and delayed-start groups in SAPS-PD slopes (secondary endpoints) were −0.17 (95% CI: −1.06, 0.72; P=0.6898) from weeks 0-6 and −0.47 (95% CI: −1.65, 0.71; P=0.4155) from weeks 6-10 [figure2][table1] (noninferior symptom progression).

Conclusion: At PDP duration <6 mo (but not ≥6 mo), SAPS-PD score changes from weeks 0-10 were numerically greater in early-start vs delayed-start groups, suggesting prompt treatment with pimavanserin may impact the progression of PDP symptoms. These preliminary signals should be interpreted cautiously but lay the groundwork for a future prospective study sufficiently powered to test the trajectory of PDP progression over a longer duration.

Table 1. Delayed-Start Analysis: Main and Secondary Exploratory Endpoints of SAPS-PD Scores Stratified by PDP Duration[a] Subgroup (Observed Cases, ITT Population)[b]

Outcome	PDP duration <6 months				PDP duration ≥6 months			
	Early-start (n=13)	Delayed-start (n=8)	Treatment difference, estimate (95% CI)[c]	*P* value[c]	Early-start (n=74)	Delayed-start (n=76)	Treatment difference, estimate (95% CI)[b]	*P* value[c]
Main exploratory endpoint: change from baseline to week 10 in SAPS-PD score								
BL, mean (SD)	11.46 (4.70)	13.88 (4.12)			16.82 (6.00)	14.42 (5.50)		
Week 10,[d] mean (SD)	3.67 (3.96)	8.86 (7.58)			10.13 (7.75)	8.03 (6.20)		
Difference in BL and week 10, LS mean (SE)	−8.25 (1.70)	−2.28 (2.22)	−5.97 (−11.95, 0.01)	0.0505	−6.38 (0.85)	−6.52 (0.83)	0.14 (−2.22, 2.49)	0.9080
Secondary exploratory endpoints: progression of symptoms as evaluated by an increase in SAPS-PD score per week								
Slope of BL to week 6,[d] LS mean (SE)	−1.10 (0.26)	−0.93 (0.33)	−0.17 (−1.06, 0.72)	0.6898	−1.03 (0.13)	−0.32 (0.13)	−0.71 (−1.06, −0.36)	0.0001
Slope of week 6 to week 10,[d] LS mean (SE)	−0.23 (0.34)	0.24 (0.45)	−0.47 (−1.65, 0.71)	0.4155	−0.10 (0.21)	−1.04 (0.20)	0.94 (0.36, 1.51)	0.0016

BL, baseline; ITT, intent to treat; LS, least squares; PDP, Parkinson's disease psychosis; SAPS-PD, Scale for Assessment of Positive Symptoms for Parkinson's Disease Psychosis.

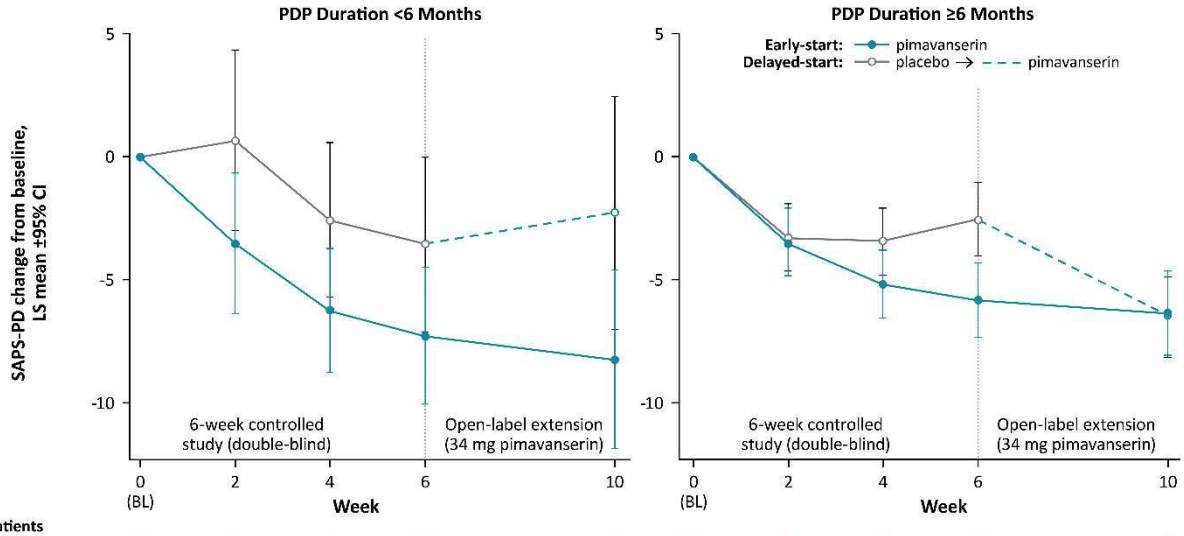
[a]PDP duration subgroup indicates time (<6 mo or ≥6 mo) between first onset of psychotic symptoms and treatment initiation (baseline).

[b]This analysis includes patients from study ACP-103-020 (NCT01174004) who rolled over to study ACP-103-015 (NCT00550238).

[c]The Random Coefficient Model included baseline as a continuous covariate, with treatment as categorical fixed effect, visit as continuous fixed effect, and subject as a random effect. An unstructured covariance matrix was used to model within-subject correlation.

[d]Numbers of patients with available SAPS-PD scores at week 10 were as follows: for PDP duration <6 months, early-start (n=12), delayed-start (n=7); for PDP duration ≥6 months, early-start (n=62), delayed-start (n=67). At week 6, these were as follows: for PDP duration <6 months, early-start (n=13), delayed-start (n=8); for PDP duration ≥6 months, early-start (n=73), delayed-start (n=75).

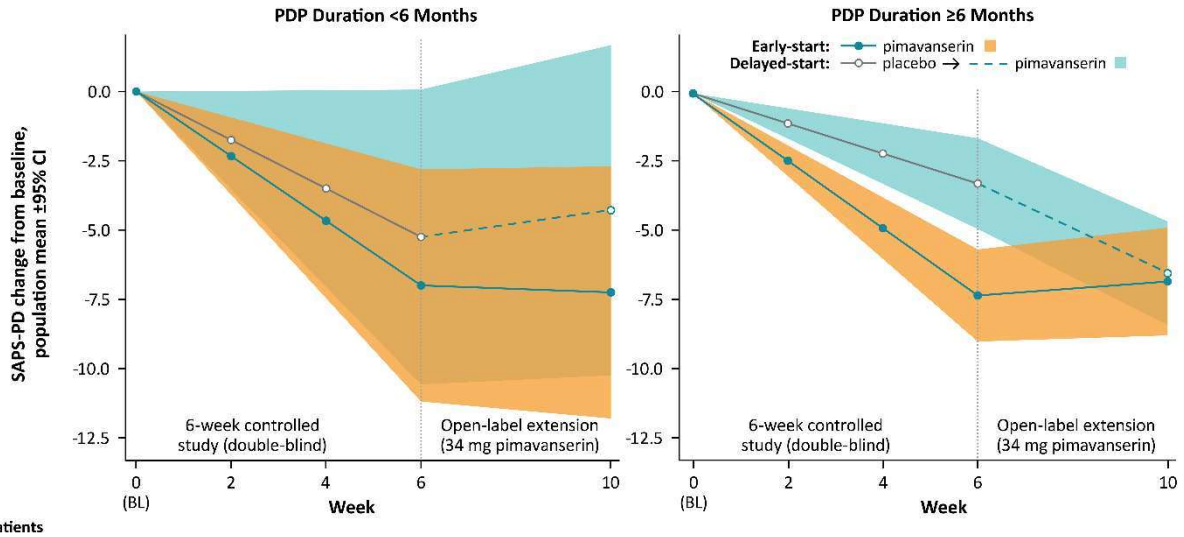
Figure 1. SAPS-PD Change From Baseline to Week 10 by Study Week by PDP Duration[a] Subgroup (ITT Population)[b]



No. of patients										
Pimavanserin	13	13	11	13	12	74	73	73	73	62
Placebo → Pimavanserin	8	8	8	8	7	76	76	76	75	67

BL, baseline; ITT, intent to treat; LS, least squares; MMRM, mixed-model repeated measures; PDP, Parkinson's disease psychosis; SAPS-PD, Scale for Assessment of Positive Symptoms for Parkinson's Disease Psychosis.
[a]PDP duration subgroup indicates time (<6 mo or ≥6 mo) between first onset of psychotic symptoms and treatment initiation (baseline).
[b]The MMRM included baseline as a continuous covariate, with treatment and visit as categorical fixed effects, treatment-by-visit interaction, and subject as a random effect. An unstructured covariance matrix was used to model within-subject correlation.

Figure 2. SAPS-PD Population Regression by PDP Duration[a] Subgroup: Piecewise Week 0-6 Slopes and Week 6-10 Slopes (ITT Population)[b]



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BL, baseline; ITT, intent to treat; PDP, Parkinson's disease psychosis; SAPS-PD, Scale for Assessment of Positive Symptoms for Parkinson's Disease Psychosis.
[a]PDP duration subgroup indicates time (<6 mo or ≥6 mo) between first onset of psychotic symptoms and treatment initiation (baseline).
[b]Week 0-6 results represent change from baseline; week 6-10 results represent change from week 6. The Random Coefficient Model was fitted without an intercept, with piecewise time (weeks 0-6 and weeks 6-10) and their interactions with treatment as fixed effects. Participant-level random slopes were included with independent variance components.

References: [1] Dashtipour K, Espay A, Tagliati M, et al. Duration of Illness and Response to Pimavanserin in Parkinson's Disease Psychosis: Post Hoc Analysis of Clinical Trial Data [abstract]. Mov Disord. 2025;40(suppl 1). https://www.mdsabstracts.org/abstract/duration-of-illness-and-response-to-pimavanserin-in-parkinsons-disease-psychosis-post-hoc-analysis-of-clinical-trial-data/. Accessed November 20, 2025.

LBA-5 Analyzing the Effect of Language Barriers on Patient Communication for Recruitment and Retention in Parkinson's Disease Clinical Trials

I. Saraf, M. Park, J. Sanderson, F. Farrokhi, E. Tamadonfar, E. Bayram, M. Bruno, C. Tanner, N. Chunga Iturry, S. Mantri, C. Vila, R. Amin, K. Williams, S. Aslam, R.P. Vasireddy, P. Auinger, M. Gross, M. Afshari, C. Branson, S. Amundsen-Huffmaster, R. Camicioli, I. Subramanian, J. Jimenez-Shahed, P. Agarwal *(Kirkland, WA, USA)*

Objective: To analyze differences in patient communication and to identify perceived effective strategies for patient recruitment and retention at Parkinson Study Group (PSG) sites.

Background: Many racial and ethnic groups remain underrepresented in Parkinson's disease (PD) clinical research, limiting the generalizability of observational and interventional data.

Method: Based on existing literature, we created a questionnaire assessing the study participants' race and ethnicities, surrounding community demographics, case studies to identify communication barriers, and strategies employed to increase recruitment and retention of underrepresented populations. This survey was distributed to research staff at North American PSG study sites. We used US Census race and ethnicity categorizations. We used responses to estimate inclusion of racial and ethnic groups in PSG study populations compared to each site's reported local demographics. We identified strategies that site staff found most helpful for increasing recruitment and retention of underrepresented groups. Additionally, we compared the case scenario responses to identify the effect of patient English fluency levels on recruitment to clinical trials.

Results: 36 staff at PSG sites across North America responded to the questionnaire. We calculated a disparity score for seven ethnic groups by dividing the research participant population percentage by the local population percentage. Black, Hispanic or Latino, and Asian patients showed the highest underrepresentation. Responders reported that provision of bilingual study materials and staff, and staff dedicated to recruitment and retention, were the most helpful strategies. While most responders reported that they would bring up clinical trials and explain them as frequently as the patient requires, including during future visits, to patients who speak fluent English, nearly half reported that they would not bring up clinical trials to those who did not speak English or Spanish.

Conclusion: These survey data, which capture the perceptions of research staff at PSG sites, highlight ongoing racial and ethnic disparities in PD research trial recruitment. However, responses also suggest effective strategies for reducing these disparities. Future systematic interrogation of these strategies and quantitative investigation of their impact will help reduce the racial and ethnic underrepresentation that continues to challenge PD research.

LBA-6 Effect of CREXONT® (IPX203, ER CD-LD) on sleep in patients with Parkinson's disease and associated sleep disturbances

O. Vaou, S. Allard, G. Banisadr, A. Morris, S. Fisher, R. Hauser *(San Antonio, Texas, USA)*

Objective: To assess the potential benefits of CREXONT® (IPX203) on sleep quality and nighttime motor states in Parkinson's disease (PD) patients with sleep disturbances.

Background: Sleep disturbances affect up to 80% of individuals with PD and are among the most disabling non-motor symptoms. CREXONT is a novel extended-release CD-LD formulation. In RISE-PD, CREXONT significantly increased “Good On” time per day and per dose vs IR CD-LD.

Methods: RISE-PD was a 20-week phase 3 study with a 3-week open-label IR CD-LD dose-optimization period, 4-week open-label dose-conversion to CREXONT, and a 13-week randomized, double-blind maintenance phase with two arms: IR CD-LD and CREXONT. The current analysis included patients with clinically relevant sleep disturbances at study entry (PDSS-2 total score ≥18). Outcome measures were changes in PDSS-2 total and subscale scores, and nighttime and early morning ‘Off’ per Hauser diary data.

Results: In the prespecified subgroup of patients with clinically relevant sleep disturbances (n=199), total PDSS-2 scores significantly improved during the open-label period (mean change = –6.99, $p<0.001$). Domain-specific analyses showed significant improvements: Disturbed Sleep Score improved from 11.94 ± 0.23 to 9.06 ± 0.28 (–2.88, $p<0.001$); Motor Symptoms at Night Score from 7.73 ± 0.24 to 5.56 ± 0.25 (–2.17, $p<0.001$); and PD Symptoms at Night Score from 6.62 ± 0.24 to 4.69 ± 0.22 (–1.93, $p<0.001$). Compared with study entry (V1), CREXONT treatment reduced nighttime “Off”: 2.5% reported none at V1, 4.5% at V2, and 18.6% by the end of CREXONT dose conversion. Early morning motor function also improved: 6.5% reported never experiencing early morning “Off” at V1, rising to 8% at V2 and 20.1% after CREXONT conversion.

Conclusion: These findings suggest that in PD patients with clinically relevant sleep disturbances, IR CD-LD optimization followed by conversion to CREXONT improves patient-reported sleep quality. CREXONT may also enhance nighttime and early morning motor control.

LBA-7 Initiation and titration of continuous subcutaneous apomorphine infusion (CSAI) in the United States: Early data from the Clinical Nurse Navigator (CNN) Program

C. Happel, A. Formella, M. Grall *(Rockville, MD, USA)*

Objective: Analyze CSAI titration patterns for people with PD (PwPD) who opt-in to a Clinical Nurse Navigator (CNN) support program.

Background: The manufacturer-sponsored CNN program is staffed by registered nurses with PD expertise and is available to all PwPD initiating CSAI treatment in the U.S. CNNs provide office-based education for HCPs and deliver tailored education to PwPD and care partners via in-home or in-clinic visits, starting pre-CSAI initiation and continuing throughout therapy.

Methods: Using information recorded in the CNN database, we report early data on practices for CSAI initiation and titration to “first optimization dose,” as determined by the patient and HCP to provide best initial balance of efficacy and tolerability.

Results: Data analysis included 558 patient initiations through 03Nov2025; of these 530 (95.0%) had ≥1 follow-up visit, and 144 (25.8%) had reached a first optimization dose at time of analysis. CSAI therapy was typically initiated at home (91.8% of PwPD) at a dose of 1.0 mg/h (98.7% of PwPD), and without antiemetic pretreatment (based on 89.6% of PwPD having no prescription for antiemetic pre-treatment). For most PwPD (90.9%), the first CNN follow-up visit occurred the day following CSAI initiation, with the second (n=468) and third (n=372) follow-up visits occurring at approximately weekly

intervals. At these respective visits, 54.8%, 81.4%, and 82.8% of PwPD underwent CSAI dose increase—typically in 0.5 mg/h increments to 1.5, 2.0 and 2.5 mg/h, respectively—0.0%, 3.0%, and 4.6% underwent dose reduction, and 45.2%,15.6%, 12.6% had no dose change. The median first optimization dose was 3.0 mg/h (Q1, Q3: 2.0, 4.0; range 1.0-6.0 mg/h) and was reached after a median of 5 follow-up visits (Q1, Q3: 3.0, 7.0; range 1-12 visits). Of those reaching a first-optimization dose, 89.6% did not use antiemetic pretreatment.

Conclusion: This analysis of over 500 PwPD initiating CSAI therapy during the first 7 months of U.S. market availability, can aid in identifying effective titration practices in the U.S. The analysis suggests that, with a tailored, flexible approach, most PwPD can reach initial optimization doses without antiemetic pretreatment.

LBA-8 Task-Specific Tremor and Parkinson's Disease: A DaTscan and alpha-synuclein biopsy study
A. Achuthaprasad, W.G. Ondo *(Houston, TX, USA)*

Objective: To investigate the association between Task-specific tremor (TST) and Parkinson's disease (PD) with DaTscan and alpha-synuclein skin biopsy.

Background: Task-specific tremor (TST) is a form of action tremor that occurs predominantly while performing a specific activity. The clinical features, electromyographic findings, and response to medication are heterogeneous, and hence it is uncertain whether TST is an isolated tremor syndrome or more pathophysiologically related to essential tremor, dystonia, or PD. Previous reports suggest some TST patients develop DaTscan-positive tremor-dominant PD. Phosphorylated alpha-synuclein(P-syn) skin biopsies (Syn-One, CND Life Sciences) are very sensitive for diagnosing idiopathic PD. We identified patients with a TST and a Parkinsonian rest tremor with a positive DaTscan. These patients underwent a P-syn skin biopsy to further investigate their disease process.

Method: We identified patients at the Methodist Neurologic Institute Movement Disorder Clinic with TST and a Parkinsonian rest tremor, and a positive DaTscan. We subsequently performed P-syn skin biopsies. Descriptive statistics regarding their clinical features and results of the DaTscan and skin biopsies are provided.

Results: We identified 4 patients, three male and one female, with a mean age of tremor onset of 65.8 (SD 5.6, range 59-72). All the patients had resting tremor as the initial sign of Parkinson's disease. None of the patients had prominent non-motor features associated with Parkinson's disease. One patient had a history of Parkinsonism in the mother, and another patient had a possible history of essential tremor in a brother. The mean latency between the onset of TST and resting tremor was 6.2 years (SD 3.3, range 3-10). Three patients had a primary writing tremor (PWT), and another patient had a tremor while holding a microphone. The latter also had a concurrent tongue tremor. Despite the positive DaTscan, all 4 patients had a negative skin biopsy for phosphorylated alpha-synuclein. Two patients developed their initial rest tremor ipsilateral to the arm with the TST and two patients developed a contralateral rest-tremor that progressed to involve both arms. DaTscan revealed reduced uptake contralateral to the rest-tremor in 3 patients and bilateral reduced uptake in one patient. In all the patients, the TST remained refractory to levodopa, while the rest-tremor generally responded.

Conclusion: Our study advances the understanding of the pathophysiology of TST and its relationship to Parkinson's disease. The specific phenotype, including abnormal DaTscan, but lacking peripheral phosphorylated alpha-synuclein, suggests a unique heretofore unrecognized disease pathology.

LBA-9 Left-DLPFC overactivation reflects reduced neural efficiency during dual-task gait in Parkinson's disease

G. Venas Santos, P. Rodrigues da Silva, L. Bitarães Rodrigues Santos, L. de Mattos Aranha, J.R. Sato, F.A. dos Santos Mendes, M.E. Pimentel Piemonte *(São Paulo, Brazil)*

Objective: To compare DT-related gait adaptations between PD and healthy older adults and to examine the association between prefrontal activation and GAITRite®-derived gait parameters.

Background: Gait automaticity declines early in Parkinson's disease (PD), increasing reliance on attentional control and contributing to functional dependence and fall risk. These deficits become more apparent during dual-task (DT) walking, when cognitive and motor demands compete. Understanding cortical compensatory mechanisms, particularly prefrontal recruitment, is crucial for guiding the development of more effective rehabilitation strategies. Functional near-infrared spectroscopy (fNIRS) enables monitoring of these neural adjustments during gait.

Methods: We evaluated 36 individuals with PD (Hoehn & Yahr 1–3) and 37 healthy older adults. Groups did not differ in age, sex, socioeconomic status, education, depressive symptoms, or global cognition. PD participants were assessed in the ON-medication state. Gait was measured on the GAITRite® walkway during single-task (ST) and DT conditions while bilateral DLPFC activity was recorded with fNIRS. A 2×2 repeated-measures ANOVA (group×condition) examined gait effects. Correlations assessed associations between DLPFC activation and DT performance. The study was approved by the Research Ethics Committee of the Hospital das Clínicas, Faculty of Medicine, University of São Paulo (CAAE: 67388816.2.0000.0065; Approval number 6.913.344).

Results: Significant interactions were found for step time, single support, and stance time. Healthy adults showed typical compensatory modulation under DT, whereas PD participants showed lack of adaptive adjustments and significant reduction in stance time. PD participants also demonstrated significant increase in the DT-related prefrontal activation, predominantly in the left DLPFC, which was correlated with shorter step length and slower gait speed.

Conclusion: In PD, increased left-DLPFC activation under DT reflects reactive, non-compensatory recruitment and reduced neural efficiency. This prefrontal overactivation may serve as an early biomarker of declining gait automaticity. Longitudinal studies are needed to determine its predictive value for decline in gait automaticity in PD.