cognitive impairment in PD require further elucidation. FDG PET imaging data analyses have revealed distinct brain metabolic patterns associated with the cognitive features of PD. The PD cognition-related pattern (PDCP) and default mode network (DMN) are two overlapping, but topographically distinct, networks that may serve as biomarkers of cognitive decline in PD. Decreased activity of the resting-state DMN and increased expression of the PDCP are associated with cognitive impairment in PD. Studies have consistently demonstrated the association between neuropsychological memory test performance and PDCP expression. Thus, we examined whether memory performance could offer additional value in predicting PDCP expression in PD patients. We hypothesized that DMN and memory performance would predict greater variance in PDCP expression than the DMN alone.
Participants and Methods: Participants included 48 PD patients ages 46-80 (mean (SD) Age: 61.9 (8.1), Education: 15.0 (2.8), IQ: 112.5 (14.9), DRS total: 136.7 (5.8)). All participants completed the FDG PET and neuropsychological evaluation 8-12 hours after their last dose of Levodopa. Neuropsychological memory testing included the California Verbal Learning Test (CVLT) z score of sum of learning trials. PDCP and DMN values were z scores generated from normal controls in previous studies. Data were analyzed using linear regression analyses.
Results: A hierarchical regression was performed to predict PDCP as a function of DMN and CVLT learning performance. Variables were entered in two separate blocks. The first block included DMN as a predictor, and the overall regression was significant ($R^2 = 0.55$, $F(1, 39)= 47.0$, $p < 0.001$). As hypothesized, DMN significantly predicted PCDP expression ($\beta= -0.74$, $p < 0.001$). The second block of the regression included CVLT learning memory performance. Both DMN and CVLT performance explained a significant amount of variance in PDCP (R^2 change = 0.05, $F(2, 39)= 27.6$, $p < 0.001$). CVLT performance significantly predicted PDCP ($\beta= -0.22$, $p =0.048$). The final model accounted for 60.0% of the variance in PDCP.
Conclusions: Disruptions in functional connectivity within brain networks have become increasingly recognized as mechanisms responsible for cognitive impairment in patients. As demonstrated in previous studies, our results indicated that DMN loss is a strong predictor of PDCP expression, likely due to the networks' overlapping spatial regions. However, we found that the addition of memory performance to the model could explain a small amount of variance (5%) over and above DMN expression. Overall, the current findings demonstrate a functional (i.e., learning) distinction between population-specific (PDCP) and more general brain networks (DMN).

Categories: Movement and Movement Disorders
Keyword 1: Parkinson's disease
Keyword 2: neuroimaging: functional connectivity
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25 Delayed Cerebrovascular Response in Parkinson's Disease

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Objective: Cardiovascular risk factors and white matter hyperintensities predict the progression and severity of cognitive symptoms in PD. While controversial, emerging evidence suggests that cerebrovascular dysfunction is an etiological driver of protein aggregation in neurodegenerative conditions, highlighting a need to understand how cerebrovascular function impacts cognitive function in PD. MRI cerebrovascular reactivity (CVR) paradigms provide an opportunity to measure the ability of the cerebral vessels to dilate or constrict in response to challenges. The current study evaluates whether whole brain CVR measures, degree of response (fit) and delay differ in PD with normal cognition (PD-NC) and PD with mild cognitive impairment (PD-MCI) relative to healthy controls (HC). Additionally, we evaluate if these metrics are associated with cognitive performance.
Participants and Methods: 8 PD-NC, 11 PD-MCI and 11 age and sex-matched healthy

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controls (HC) participated in the study. PD participants were diagnosed with MCI based on the Movement Disorders Society Task force, Level II assessment (comprehensive assessment). Participants were asked to inhale gas enriched in CO_2 to elicit a vasodilatory response while undergoing bold oxygen level-dependent magnetic resonance image (MRI). Whole brain fit to an end-tidal CO_2 regressor and delay were used to quantify CVR in each participant. An analysis of covariance (ANCOVA) was used to evaluate group differences between HC, PD-NC, and PD-MCI in the whole brain fit and delay CVR measures accounting for age, sex, and education. Multiple regressions were conducted for each cognitive variable with whole brain fit and delay as the dependent variables adjusting for age, sex, and education.
Results: A significant main effect of group was observed for whole brain CVR latency ($F_{(2, 23)}$ = 4.227; p = 0.027). Post hoc tests were not significant, though indicated a trend that PD-NC (18.14 ±1.94) and PD-MCI (18.15 ± 1.55) patients exhibited longer delays relative to HC (15.84 ± 2.37). Regression results indicated limited relationships between CVR measures and cognitive functioning.
Conclusions: PD patients (PD-NC and PD-MCI) exhibited longer CVR delays relative to HC, suggesting a delayed vasodilatory response in PD. Examination of the association between CVR metrics and cognition were not significant, though these results should be interpreted with caution given the small sample size.

Categories: Movement and Movement Disorders
Keyword 1: Parkinson's disease
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Keyword 3: mild cognitive impairment
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26 Correlates of Neuropsychological Decline Following Deep Brain Stimulation in Patients with Parkinson's Disease

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Objective: Deep Brain Stimulation (DBS) is an FDA-approved treatment for Parkinson's Disease (PD), for which the medical workup includes routine pre- and post- operative neuropsychological assessment to determine potential surgical cognitive risk. Existing research suggests that cognitively normal individuals experience good cognitive outcome, whereas those with pre-existing cognitive deficits are prone to accelerated cognitive decline post-DBS. The goal of this study is to identify characteristics that determine which individuals with PD are at risk for accelerated post-DBS cognitive loss, and to characterize the nature of the decline in this population.
Participants and Methods: We conducted a retrospective chart review of PD- DBS patients who completed their DBS workup and surgery at Mount Sinai Hospital NYC between 2015 and 2022. Non-English speakers were excluded from this study due to small sample size and use of a neurocognitive battery different from that of English speakers. Using repeated measures t-tests, chi square, and regression analyses, we explored variables related to disease (e.g., duration, L-Dopa burden, DBS target), socio-demographic background (e.g., age onset, current age, education), assessment modality (telehealth vs in-office), neurocognitive performances (e.g., WMS-IV Logical Memory (LM), HVLT-R, WASI-II Matrix & Similarities, WAIS-IV Digit Span), and cognitive diagnosis (amnestic vs non-amnestic MCI) for all individuals in the sample. At the individual level, we utilized Reliable Change Indices (RCI) to identify clinically significant cognitive differences from pre- to post-DBS exam. We considered LM- Delayed Recall (LMDR) as a proxy for memory loss, as this cognitive function is expected to remain generally unchanged post PD-DBS. Therefore, decline on this measure in the first year after DBS could indicate a change in global memory function and possible evidence of accelerated postoperative decline.
Results: Of 65 charts reviewed, 44 patients were native English-speaking and included in our analyses. At the group level, there were no significant differences in disease characteristics, socio-demographic variables, or cognitive classification between those who declined versus those who did not decline on

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