for determining hemispheric contributions. In PD, use of refined metrics for determining the extent of asymmetric dopaminergic degeneration (e.g., DAT scan) at the hemispheric level coupled with sensitive neuropsychological measures may provide clearer understanding of potential neural circuitry relationships.

Categories: Movement and Movement Disorders
Keyword 1: Parkinson's disease
Keyword 2: laterality
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21 Exploratory Factor Analysis of the Cognitive Change Index-20 in Individuals with Parkinson disease or Essential Tremor

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Objective: Subjective cognitive complaints are common in individuals with Parkinson's disease and Essential Tremor. One scale often used to capture the type and severity of subjective cognitive concerns is the Cognitive Change Index-20 (CCI-20). Created by Saykin et al (2006), the CCI-20 is a questionnaire that assesses perception of cognitive changes in memory, executive function, and language domains. Despite its multidomain structure, previous research has not empirically examined whether the CCI-20's underlying factor structure aligns with the cognitive domains proposed during its original development. Thus, the goal of the current study was to investigate the factor structure of the CCI-20 in individuals with movement disorders (Parkinson's disease, Essential Tremor) who are known to experience varying degrees of cognitive sequelae as part of their disease progression.
Participants and Methods: Participants included a convenience sample of 216 non-demented individuals with Parkinson disease (n=149) or Essential Tremor (n=67) who were seen at the University of Florida Fixel Institute Movement Disorders Center. All received the CCI-20 as part of a neuropsychological evaluation. The CCI-20 consists of 20 items, rated on a 5-point Likert scale, that ask questions about change in memory (12 items), executive function (5 items), and language (3 items) over the past 5 years. An exploratory factor analysis was conducted on CCI-20 scores using Promax rotation with factor extraction based on scree plot visual inspection and Kaiser's rule (eigenvalues >1.0). Cronbach's alpha was used to assess internal consistency reliability. Finally, Spearman correlations determined associations between factors and mood measures of depression (Beck Depression Inventory-II, BDI-II), apathy (Apathy Scale, AS), and anxiety (State-Trait Anxiety Inventory, STAI).
Results: Because the Parkinson's disease and Essential Tremor groups did not statistically differ in their CCI-20 total scores, they were combined into a single group for analyses. This resulted in 216 participants who were well-educated (m=15.01±2.92), in their mid-60's (m=67.72±9.33), predominantly male (63%), and non-Hispanic White (93.6%). The factor analysis resulted in 3 factors: factor 1 included 8 memory items (items 1-4, 6, 10-12; loadings from .524 to .920); factor 2 included all executive and language (items 13-20; loadings from .605 to .824), and factor 3 included four remaining memory items (items 5, 7-9; loadings from .628 to .810). Reliability of the 20 CCI items was good (α = .94), and reliability within each factor ranged from adequate (Factor 3, α = .78) to good (Factors 1 and 2, α = .90). All factors showed significant weak to moderate associations with BDI-II, AS, and STAI (state and trait) scores.
Conclusions: The CCI-20 revealed three distinct dimensions of subjective cognitive complaints that did not correspond to the memory, executive function, and language domains. Rather, the CCI-20 was decomposed into two different dimensions of memory complaints and one dimension of non-memory complaints. Mood symptoms played a significant role in driving all dimensions of subjective cognitive complaints. Future studies should confirm this triadic structure in a healthy older adult sample and explore the relationship between factors and objective cognitive performance beyond the contribution of mood.
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22 Cognitive Reserve's Relationship to Brain Burden in Parkinson's Disease Without Dementia

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Objective: Individuals with Parkinson's disease (PD) have varying trajectories of cognitive decline. One reason for this heterogeneity may be "cognitive reserve": where higher education/IQ/current mental engagement compensates for increasing brain burden (Stern et al., 2020). With few exceptions, most studies examining cognitive reserve in PD fail to include brain metrics. This study's goal was to examine whether cognitive reserve moderated the relationship between neuroimaging indices of brain burden (diffusion free water fraction and T2-weighted white matter changes) and two commonly impaired domains in PD: executive function and memory. We hypothesized cognitive reserve would mitigate the relationship between higher brain burden and worse cognitive performance.
Participants and Methods: Participants included 108 individuals with PD without dementia (age mean=67.9±6.3, education mean=16.6±2.5) who were prospectively recruited for two NIH-funded projects at the University of Florida. All received neuropsychological measures of executive function (Trails B, Stroop, Letter Fluency) and memory (delayed recall: Hopkin's Verbal Learning Test-Revised, WMS-III Logical Memory). Domain specific z-score composites were created using data from age/education matched non-PD peer controls (N=62). For the Cognitive Reserve (CR) proxy, a z-score composite included years of education, WASI-II Vocabulary, and Wechsler Test of Adult Reading. At the time of testing, participants completed multiple MRI scans (T1-weighted, diffusion, Fluid Attenuated Inversion Recovery) from which the following were extracted: 1) whole-brain free water within the white matter (a measure of microstructural integrity and neuroinflammation), 2) white matter hyperintensities/white matter total volume (WMH/WMV), and bilaterally-averaged edge weights of white matter connectivity between 3) dorsolateral prefrontal cortex and caudate and 4) entorhinal cortex and hippocampi. Separate linear regressions for each brain metric used executive function and memory composites as dependent variables; predictors were age, CR proxy, respective brain metric, and a residual centered interaction term (brain metric*CR proxy). Identical models were run in dichotomized short and long disease duration groups (median split=6 years).
Results: In all models, a lower CR proxy significantly predicted worse executive function (WMH/WMV: beta=0.49, free water: beta=0.54, frontal edge weight: beta=0.49, p's<0.001) and memory (WMH/WMV: beta=0.42, free water: beta=0.35, temporal edge weight: beta=0.39, p's <0.01). For neuroimaging metrics, higher free water significantly predicted worse executive function (beta=-0.39, p=0.002) but not memory. No other brain metrics were significant predictors of either domain. Accounting for PD duration, higher free water predicted worse executive function for those with both short (beta=-0.49, p=0.04) and long disease duration (beta=-0.48, p=0.02). Specifically in those with long disease duration, higher free water (beta=-0.57 p=0.02) and lower edge weights between entorhinal cortex and hippocampi (beta=0.30, p=0.03) predicted worse memory. Overall, no models contained significant interactions between the CR proxy and any brain metric.
Conclusions: Results replicate previous work showing that a cognitive reserve proxy relates to cognition. However, cognitive reserve did not moderate brain burden's relationship to cognition. Across the sample, greater neuroinflammation was associated with worse executive function. For those with longer disease duration, higher neuroinflammation and lower medial temporal white matter connectivity related to worse memory. Future work should examine other brain burden metrics to determine whether/how cognitive reserve influences the cognitive trajectory of PD.

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