Categories: Multiple Sclerosis/ALS/Demyelinating Disorders
Keyword 1: multiple sclerosis
Keyword 2: cognitive functioning
Keyword 3: fatigue
Correspondence: Tracy Lauren Fabri Department of Psychology, York University, Toronto, Canada tfabri@my.yorku.ca

46 Depression and Reward Responsiveness in Multiple Sclerosis

Valerie Humphreys, Fareshte Irani, Darshan Patel, Maria Schultheis, John Medaglia, Kathryn N Devlin
Drexel University, Philadelphia, PA, USA

Objective: Depression is common in persons with MS (PwMS), substantially contributing to morbidity and mortality. Depression can dually impact PwMS as both a psychosocial reaction to living with the disease and a neurological effect of it. Cardinal features of depression include reduced ability to seek and experience pleasure, often attributed to dysregulation of the brain's reward system. People with depression exhibit atypical reward processing, as do fatigued PwMS. However, it is unclear whether MS itself affects reward processing, and whether it interacts with depression. The current study explored the associations of depression, MS, and their interaction on reward responsiveness. We hypothesized that depression and MS would independently be associated with poorer reward responsiveness and that they would interact synergistically to impair reward responsiveness.
Participants and Methods: Forty PwMS and 40 healthy age- and education-matched healthy controls (HC) participated in a computerized switching task with high- and low-reward manipulations. The Chicago Multiscale Depression Inventory (CMDI) Mood subscale measured depressive symptoms. The Behavioral Inhibition/Activation Scales (BIS/BAS) measured self-reported reward responsiveness and behavioral inhibition. Switching task performance was measured as response time (RT) and accuracy. Performance differences between the high- and low-reward conditions represented performance-based reward responsiveness. Linear mixed effects models were used to estimate the associations of MS and depression with reward responsiveness, behavioral inhibition, and task performance.
Results: Depression, but not MS, was associated with higher BIS scores (p=.007). Neither depression nor MS was associated with BAS subscales. On the switching task, participants who reported lower depression responded to reward such that they were slightly faster in the high-reward condition compared to the low-reward condition (p=.07). By contrast, in participants who reported higher depression, there was no effect of reward on response time. Additionally, MS (p=.009) and depression (p=.018) were each associated with slower response times. Regarding accuracy, no effects of reward were observed; however, there was an interaction between MS and depression. Among HC participants, depression was not related to accuracy. In comparison, PwMS who reported higher depression were more accurate than PwMS who reported less depression (p=.043).
Conclusions: Consistent with hypotheses, higher depressive symptoms were associated with increased behavioral inhibition. Depression was not associated with self-reported reward responsiveness, but it was associated with reduced reward responsiveness on a cognitive task. Contrary to hypotheses, MS was not associated with reduced reward responsiveness. Additionally, higher depression and an MS diagnosis were related to slower response time, consistent with prior findings that psychomotor slowing is a hallmark feature of both disorders. Interestingly, we observed a unique behavioral trend in PwMS, such that PwMS with higher depressive symptoms were more accurate than PwMS with lower depressive symptoms, whereas this relationship was not present among HCs. Altogether, depression in both PwMS and cognitively healthy individuals may be associated with blunted reward responsiveness, but MS does not exacerbate this relationship. In fact, PwMS with depression may be more conscientious in their functioning and therefore perform better on cognitive task accuracy. Continued work should examine how reward processing and its underlying mechanisms may differ in depressed PwMS.

Categories: Multiple Sclerosis/ALS/Demyelinating Disorders
Keyword 1: multiple sclerosis
Keyword 2: depression
Keyword 3: motivation

https://doi.org/10.1017/S1355617723007178 Published online by Cambridge University Press

Correspondence: Valerie Humphreys, Drexel University, vh339@drexel.edu

47 Evolution of Brain Morphology and Cognitive Performance in Parkinson's Disease with Impulse Control Disorder

Adina Lorena Patru[1,2], Lucas Ronat[1,2], Alexandru Hanganu[1,2]
[1]University of Montreal, Montreal, Quebec, Canada. [2]Research Center of the University Institute of Geriatrics of Montreal, Montreal, Quebec, Canada

Objective: Parkinson's disease (PD) affects the person's quality of life, but the comorbidity of PD and impulsive control disorder (ICD), which has an average prevalence of 23%, can enhance the disruption of quality of life for the patients and their caregivers. The effects of ICD in PD on brain morphology and cognition have been little studied. Thus, this study proposes to investigate the differences in the evolution of cognitive performance and brain structures between PD patients with ICD (PD-ICD) vs. without ICD (PD-no-ICD).
Participants and Methods: Parkinson's Progression Markers Initiative (PPMI) data of 58 patients with idiopathic PD, including their MRI data at baseline and three years later, were analyzed. The MRIs were processed with FreeSurfer (7.1.1) to extract cortical volumes, areas, thicknesses, curvatures and folding index as well as volumes of subcortical segmentations. All participants underwent cognitive evaluations. The Questionnaire for Impulsive-Compulsive Disorders in Parkinson's Disease was used to differentiate those with at least one ICD from those without any ICD. 12 of the 58 patients had an ICD at their first visit and 19 had an ICD at their visit three years later. There was no significant difference between PD-ICD and PD-no-ICD with respect to sex, use of overall medication, age, age of onset, age at diagnosis, years of education and the Montreal cognitive assessment score. Two-way mixed ANOVAs were performed for each neuropsychological test and brain structure extracted from MRIs with the time of the visit as the repeated independent variable (within participants) and the presence or absence of an ICD as the other independent variable (between participants).
Results: The mixed ANOVA revealed that PD-ICD had their performance decline after three years, for the Hopkins Verbal Learning Test delayed recall and the Symbol Digit Modalities Test while PD-no-ICD saw their performance increase. A whole brain analysis showed that PD-ICD had a significant decrease after three years of the right cortex area total brain volume in comparison to PD-no-ICD. Specific brain structures also underwent significant changes over three years. Cortical changes in PD-ICD were: (1) increased surface area in the left temporal parahippocampus and (2) decreased surface areas of the right insula, right middle and superior temporal regions, left occipital lingual as well as left cingulate isthmus. Furthermore, in the subcortical nuclei, PD-ICD showed (1) increased volumes of the paratenial thalamic nucleus and whole right amygdala and (2) decreased volumes of the right amygdalian basal nucleus and thalamic ventromedial nucleus.
Conclusions: This study suggests that PD patients who also have ICD might be prone to develop over three years: (1) significant changes in cognitive performance (memory, attention), (2) morphological changes in the amygdala and thalamic nuclei and (3) significant atrophy and area shrinkage in the temporal and insula regions.

Categories: Neurodegenerative Disorders
Keyword 1: Parkinson's disease
Keyword 2: neuropsychiatry
Keyword 3: neuroimaging: structural
Correspondence: Adina Lorena Patru adina.lorena.patru@umontreal.ca University of Montreal Research Center of the University Institute of Geriatrics of Montreal

48 Longitudinal Study: Impact of Anxiety on the Evolution of Cognitive Performance and Brain Morphology in Patients with Parkinson's Disease

Adriana Cannizzaro[1,2], Adina Lorena Patru[1,2], Alexandru Hanganu[1,2]
[1]Centre de recherche de l'Institut universitaire de gériatrie de Montréal, Montréal, QC, Canada. [2]Université de Montréal, Montréal, QC, Canada

https://doi.org/10.1017/S1355617723007178 Published online by Cambridge University Press