within PADS that make it difficult to generalize findings. Future studies could prospectively follow patients over time to better understand the trajectory of recovery, identify predictors for relapse, and those at greatest risk of neurocognitive and behavioral deficits.

Categories: Multiple Sclerosis/ALS/Demyelinating Disorders
Keyword 1: demyelinating disorders
Keyword 2: pediatric neuropsychology
Keyword 3: cognitive functioning
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29 Associations Between Social Support and Cognitive Performance Among Persons with MS

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Objective: Social support is an emerging protective factor against cognitive decline. However, the relationship between social support and cognitive functioning in the multiple sclerosis (MS) population is not well understood. The present study aimed to investigate the associations between different aspects of social support and cognitive performance among persons with MS.
Participants and Methods: A volunteer sample of 63 persons with MS (% female = 88.9, mean age = 48.16) completed measures assessing perceived levels of social support measured by the Medical Outcomes Study Support Social Survey 5-item short form (MSSS-5), and social network (social network diversity and total size of social network) measured by the Social Network Index (SNI). Cognitive functioning was assessed by a brief virtual examiner-administered neuropsychological test battery (using a teleconferencing platform), including the Rey Auditory Verbal Learning Test, Controlled Oral Word Association Test, animal naming, and the Symbol Digit Modalities Test. Participants also completed brief, self-paced, virtual cognitive tests through the testmybrain.org platform, which consisted of digit span and the Trail-Making Test. A principal component analysis (PCA) was carried out to reduce the number of neuropsychological outcomes into fewer dimensions. Multiple linear regressions were conducted to examine the associations between social support measures and cognitive performance. Regression models were adjusted by the levels of depressive symptoms (operationalized by the Chicago Multiscale Depression Inventory or the Hospital Anxiety and Depression Scale) and premorbid functioning (measured by the Test of Premorbid Functioning).
Results: A PCA reduced neuropsychological outcomes into 3 components representing cognitive domains of 1) processing speed/executive functioning, 2) verbal memory, and 3) verbal fluency / simple attention. In the unadjusted models, both perceived social support (i.e., to what extent one receives assistance from their social network) as well as total size of social network (i.e., total number of people one regularly talks to) were significant predictors of the processing speed/executive functioning component score of moderate strength, where $F(1, 59) = 11.93$, $p = .001$, $\beta = 0.41$ and $F(1, 59) = 11.57$, $p = .001$, $\beta = 0.41$, respectively. These associations were maintained after adjusting for depressive symptoms and level of premorbid functioning ($F(4, 55) = 3.31$, $p = .003$ and $F(4, 55) = 3.31$, $p = .006$, respectively). On the other hand, social network diversity (i.e., number of different types of close social relationships one has) was not a significant predictor of the processing speed/executive functioning component score ($p > 0.05$). None of the social support measures were significantly associated with the verbal memory and verbal fluency/simple attention component scores.
Conclusions: Greater social support (specifically, perceived levels of assistance and total size of social network) is associated with better performance on processing speed/executive functioning measures among persons with MS, independent of effects from depressive symptoms and premorbid functioning. Maintaining a strong social support

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network may be an important factor in optimizing cognitive health in MS.

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30 Hippocampal Internal Architecture Subfield Volumes Associated with Systematic Inflammatory Biomarkers in Multiple Sclerosis

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Objective: Multiple Sclerosis (MS) affects up to 500,000 adults in the United States, with cognitive impairment present in 45%-65% of people. Studies showed hippocampal atrophy in MS, but the underlying mechanisms remain unknown. Inflammation has been proposed to play a significant role, and associations between systemic inflammatory biomarkers and hippocampal atrophy have been shown in other neurological conditions. However, research exploring serum biomarker and volumetric associations in MS are lacking. Given that conventional imaging methods lack resolution for hippocampal internal architecture (HIA), new protocols were developed. We used the High-Resolution Multiple Image Co-Registration and Averaging (HR-MICRA) method to visualize the HIA subfields. We investigated the relationship between subfield volumes generated from HR-MICRA scans and systemic serum biomarkers in MS.
Participants and Methods: Patients with MS were recruited (N= 34, mean age= 54.6, 35.3% Black) underwent Magnetic Resonance Imaging (MRI), and serum biomarkers were obtained, specifically chosen for their potential role in MS. Inflammatory biomarkers included; granulocyte colony stimulating factor (G-CSF), interleukin-10 (IL-10), matrix metalloproteinase-9 (MMP-9), tumor necrosis factor- α (TNF- α), and growth factors; vascular endothelial growth factor (VEGF); insulin-like growth factor-1 (IGF-1), and brain derived growth factor (BDNF). Imaging was performed in a Siemens Prisma 3T scanner with a 64-channel head coil using the HR-MICRA method. Hippocampal subfields were calculated using the Automated Segmentation of Hippocampal Subfields (ASHS) package. We used the Magdeburg Young Adult 7T Atlas for sub-hippocampal structures and Penn Temporal Lobe Epilepsy T1-MRI Whole Hippocampus ASHS Atlas for general hippocampal structure and segmentation. Pearson's product-moment analyses provided correlations between biomarkers and hippocampal subfield volumes for each cerebral hemisphere. A statistical significance level of $p < 0.05$ was used for all analyses.
Results: Correlations emerged between left hemisphere Cornu Ammonis (CA) 2 and G-CSF ($r = –.384$; $p = .025$); IL-10 ($r = –.342$; $p = .048$); VEGF ($r = –.371$; $p= .031$); and CA3 with IL-10 ($r = –.488$, $p = .003$); G-CSF ($r = –.386$; $p= .024$); VEGF ($r = –.352$; $p= .041$). Dentate gyrus correlated with MMP-9 ($r =.416$; $p=.014$); IL-10 ($r = –.365$; $p =.034$). BDNF was correlated with right hemisphere CA1 ($r = –.417$, $p = .014$), CA2 ($r = –.497$; $p= .003$) and CA3 ($r = –.451$; $p=.007$).
Conclusions: In our sample of persons with MS, left hemisphere hippocampal subfield volumes were negatively correlated with inflammatory biomarkers, supporting previous reports linking inflammation to reduced brain volumes in other neurological conditions. In the right hemisphere, we found negative correlations between HIA and BDNF, suggesting a neuroprotective function for BDNF in this neurodegenerative disease. These findings in a representative sample of patients with MS highlight the need for further research exploring

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