

increased and decreased functional connectivity (FC) in the DMN in ASD and increased FC in ADHD. Additionally, little studies have investigated executive and attentional network dysfunction in the DMN for ASD and ADHD populations. To better understand the shared characteristics between ASD and ADHD, this study analyzed the DMN FC in children with ASD and ADHD.

Participants and Methods: Archival datasets from Autism Brain Imaging Data Exchange (ABIDE)-I and ADHD-200 datasets were used, with 33 ADHD, 35 ASD, and 32 typically developing (TD) males (ages = 7–17 years). After applying a standard pre-processing pipeline, 11 regions of interest (ROIs) from the Dosenbach-160 atlas were examined with 55 ROI pairs generated for the 100 subjects.

Results: Significant differences were noted between ASD–ADHD groups in attentional networks and executive functioning networks. Specifically, significant Group x VIQ interactions were noted for FC between the following pairs of regions: medial prefrontal cortex – ventromedial prefrontal cortex, anterior cingulate cortex – ventromedial prefrontal cortex, inferior temporal lobe – ventromedial prefrontal cortex, angular – ventromedial prefrontal cortex, angular – anterior cingulate cortex, inferior temporal lobe – ventrolateral prefrontal cortex, angular – superior frontal lobe, and intraparietal sulcus – superior frontal lobe. In the above FC pairs, FC in ADHD was negatively correlated with VIQ, with no correlation for ASD and positive correlation for TD. Previous literature has indicated that ADHD individuals demonstrate increased executive functioning deficits compared to ASD individuals. This study provides evidence at a neural level for these findings by demonstrating decreased FC trends in ADHD in attentional and executive functioning networks compared to ASD individuals. Group and VIQ main effects demonstrated mixed patterns across the three groups, as well as shared decreased FC in attention/executive networks for both ASD and ADHD groups.

Conclusions: In summary, this study found similar findings from previous studies regarding mixed connectivity patterns, as well as shared dysfunction between ASD and ADHD groups. These results help in solidifying the theory that ASD and ADHD share clinical and neural patterns which need to be examined further. Future directions include utilizing more ASD+ADHD comorbid individuals in studies comparing ASD and ADHD FC trends as well as

seeking to further understand the neuropsychological and neuroimaging profiles in ASD and ADHD.

Categories: Neuroimaging

Keyword 1: attention deficit hyperactivity disorder

Keyword 2: autism spectrum disorder

Keyword 3: neuroimaging: functional connectivity

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45 The Impact of Loneliness on Amyloid Burden, Cerebrovascular Disease, Neurodegeneration, and Memory Performance in a Community-Based Sample of Older Adults

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Objective: The current research framework recommends using biomarkers to further understand Alzheimer's disease (AD) pathogenesis, including other contributing factors like cerebrovascular disease. In longitudinal studies of people with neuropathological examination after death, baseline loneliness was associated with lower cognition, faster cognitive decline, and future AD risk, independent of AD pathology. Examination of memory impairment along with AD and cerebrovascular biomarkers, could aid risk reduction efforts earlier in the lifecourse and among populations with more exposure to loneliness. We hypothesized that loneliness is associated with amyloid, vascular, and neurodegeneration biomarkers; with worse memory; and that loneliness increases the susceptibility to biomarker-related memory impairment.

Participants and Methods: A subset of cognitively unimpaired older adults with available amyloid PET, vascular MRI (white matter hyperintensity volume, WMH), structural

MRI (cortical thickness in AD signature regions), neuropsychological testing (memory factor score), dichotomized loneliness data (one item from CES-D), and relevant medical data were drawn from the community-based Washington Heights-Inwood Columbia Aging Project (WHICAP; $n=169$; covariates included age= 81 ± 6 years; 63% women; 49/31/20% Non-Hispanic Black/Non-Hispanic White/Hispanic; education= 13 ± 4 years; 32% APOE- $\epsilon 4$ carriers). General linear models in the overall sample and stratified by race and ethnicity tested the association between loneliness and AD and cerebrovascular biomarkers, loneliness and memory, and the interaction of loneliness and biomarkers on memory, adjusting for covariates. **Results:** Loneliness was endorsed in 18% of participants, marginally associated with older age (2.1 [-0.2, 4.4], $p=0.08$), was more likely in those with untreated diabetes (13/0.1% lonely/not lonely, $p=0.001$), associated with lower cortical thickness (-0.05 [-0.09, -0.02], $p=0.01$), and associated with lower memory (-0.3 [-0.6, -0.001], $p=0.05$). In Non-Hispanic White participants, loneliness was associated with greater WMH volume (0.5 [0.07, 0.82], $p=0.03$), while in Hispanic participants, loneliness was associated with lower cortical thickness (-0.16 [-0.24, -0.08], $p=0.0006$). In Non-Hispanic Black participants, loneliness was associated with lower memory (-13 [-26, -0.5], $p=0.05$), and the association between lower cortical thickness and lower memory was stronger in those that endorse loneliness (5 [0.2, 10], $p=0.05$). In Hispanic participants, loneliness was associated with higher memory (13 [4, 22], $p=0.009$), but the association between higher amyloid burden and lower memory was stronger in those that endorse loneliness (-12 [-20, -4], $p=0.006$); further, loneliness was marginally associated with lower memory (-0.7 [-1.4, 0.1], $p=0.09$), independently of WMH.

Conclusions: Associations between loneliness and biomarkers may relate to health seeking behavior, reported as treatment status for diabetes, for cerebrovascular burden and general neurodegeneration, but might be more complex for amyloid. The degree to which loneliness increased the susceptibility to amyloid and neurodegeneration-related, but not cerebrovascular-related, memory impairment, specifically, may suggest that domains beyond memory should be considered. Future work should be longitudinal to disentangle the effects of loneliness from related constructs like depression and anxiety, incorporate other AD

biomarkers such as hyperphosphorylated tau, and incorporate biological mechanisms (e.g., stress, inflammation) into models of loneliness and AD pathogenesis. Older adults from all backgrounds may be more susceptible to loneliness, which was associated with lower memory; culturally-humble, social support-based interventions may reduce the risk of cognitive impairment.

Categories: Neuroimaging

Keyword 1: dementia - Alzheimer's disease

Keyword 2: emotional processes

Keyword 3: social processes

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46 Moderating Impact of Trauma on Brain Regions Underlying Social Cognition in Early Onset Psychosis

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Objective: Previous research has found that trauma is a risk factor for developing early-onset psychosis (EOP), both exhibiting widespread structural abnormalities and social cognitive dysfunction (Hoy et al., 2012; Nair et al., 2020; Rotiker et al., 2018). However, few studies have investigated the association between trauma, neural architecture, and social behaviors. The current study examines whether trauma exposure moderates the association between cortical volume and thickness and social cognition in EOP.

Participants and Methods: T1-weighted whole-brain magnetic resonance data were acquired on a 3T Siemens scanner for 23 adolescents with EOP aged 12-21 years ($M = 16.12$), and 20 age-matched controls ($M = 17.22$). Cortical volume and thickness were calculated using the Freesurfer software suite (v5.3; Reuter et al., 2012). Based on prior research, bilateral structures of interest included the rostral anterior cingulate cortex (rACC), insula, precuneus, and superior frontal cortex. Social measures included the WebCNP Emotion Recognition (KER40) and Emotion Differentiation Test