

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

(MED36) accuracy score (Gur et al., 2010), The Awareness of Social Inference Test Total Score (TASIT; McDonald et al., 2003), and Social Responsiveness Scale, 2nd Edition (SRS-2; Constantino & Gruber, 2012). Trauma exposure was assessed using the Structured Clinical Interview Diagnostic (controls $n = 5$; EOP $n = 9$; First et al., 2015). Pearson's correlations and independent t-tests were used to examine the relationship between cortical measurements and social cognition. Additionally, PROCESS macro (Hayes, 2018) was used to examine if trauma history statistically moderated the relationship between cortical measurements and social cognition performance.

Results: Significant group differences in SRS-2 scores were observed, as EOP participants scored 24.272 points higher than controls ($t = 20.724$, $p < .001$). Across both groups, there was a negative correlation between the SRS-2 score and precuneus volume ($r = -.438$, $p = .011$) and thickness ($r = -.383$, $p = .028$), TASIT total and superior frontal volume ($r = -.349$, $p = .023$), and KER40 and insular volume ($r = -.437$, $p = .20$). Further, the moderation analysis revealed that the relationships between precuneus volume and SRS-2 scores, precuneus thickness and MED36 scores, and rACC thickness and KER40 scores depended on experiencing trauma across both groups. Participants with trauma across groups had increased precuneus volume associated with higher SRS-2 scores ($p = .0442$). Experiencing trauma was also associated with lower precuneus cortical thickness and lower MED36 scores ($p = .0172$). Conversely, lack of trauma experience was associated with greater rACC thickness and higher KER40 scores ($p = .0119$).

Conclusions: Our findings indicate that past traumatic experiences may be a moderating factor in the relationship between atypical volume and thickness of social brain regions and social cognition. Overall, the significant interactions between trauma exposure and increased volume and thickness in both EOP and control participants were associated with increased impairment on social cognition measures. These findings emphasize the importance of accounting for the impact of early life adversities on brain development and how it may be relevant to social impairments, especially in individuals experiencing psychosis.

Categories: Neuroimaging

Keyword 1: neuroimaging; structural

Keyword 2: social cognition

Keyword 3: psychosis

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47 Exposure to Early Life Adversity is Related to Alterations in Neural Correlates of Inhibitory Control in Preadolescents: Findings from the ABCD Study Cohort

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Objective: Rapid neurodevelopment occurs during adolescence, which may increase the developing brain's susceptibility to environmental risk and resilience factors. Adverse childhood experiences (ACEs) may confer additional risk to the developing brain, where ACEs have been linked with alterations in BOLD signaling in brain regions underlying inhibitory control. Potential resiliency factors, like a positive family environment, may attenuate the risk associated with ACEs, but limited research has examined potential buffers to adversity's impact on the developing brain. The current study aimed to examine how ACEs relate to BOLD response during successful inhibition on the Stop Signal Task (SST) in regions underlying inhibitory control from late childhood to early adolescence and will assess whether aspects of the family environment moderate this relationship.

Participants and Methods: Participants ($N = 9,080$; $M_{age} = 10.7$, range = 9-13.8 years old; 48.5% female, 70.1% non-Hispanic White) were drawn from the larger Adolescent Brain Cognitive Development (ABCD) Study cohort. ACE risk scores were created (by EAS) using parent and child reports of youth's exposure to adverse experiences collected at baseline to 2-year follow-up. For family environment, levels of family conflict were assessed based on youth reports on the Family Environment Scale at baseline and 2-year follow-up. The SST, a task-based fMRI paradigm, was used to measure inhibitory control (contrast: correct stop > correct go); the task was administered at baseline and 2-year follow-up. Participants were excluded if