trial performed significantly worse on non-memory cognitive domains pertaining to general fund of knowledge, working memory, and executive functioning. Additionally, the screen fail group reported greater levels of anxiety, but not depression nor endorsements on a measure of functional status.
Conclusions: Conclusions: Worse performance on non-memory neuropsychological domains was related to screen failure status for the EMERGE AD clinical trial. This finding may be explained by the traditional recruitment pathway from clinic to trials, which beyond the diagnosis of interest is up to the opinion of the physician to determine “fit” for a trial. Higher screen failure rates may result from physicians erroneously viewing more globally-impaired patients as being more appropriate for an AD clinical trial, resulting in greater tendencies towards recruiting patients who are too severe to meet inclusion criteria for a trial. Recruiting patients into clinical trials earlier in their disease course – when disease severity is less – may result in reduced screen failure rates in AD trials. That we could not detect a relationship between memory-related tasks and screen fail/pass status may be explained by either (1) the measures used in the EMERGE trial were not as sensitive to subtle changes in memory, or (2) that memory dysfunction is necessary for a diagnosis of AD but not sufficient to distinguish who will be successfully screened into an AD clinical trial. Overall, these findings have the potential to advance the field by reducing screen failure rates in AD clinical trials by using information already available to clinical trial teams, which will enhance trial-recruitment infrastructure and encourage greater engagement of older adults in AD research.

Categories: Dementia (Alzheimer's Disease)
Keyword 1: clinical trials
Keyword 2: dementia - Alzheimer's disease
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Paper Session 10: Pediatric topics

10:15 - 11:40am
Friday, 3rd February, 2023
Town & Country Ballroom C

Moderated by: Sakina Butt

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1 Social Brain Network Connectivity Relates to Social and Adaptive Outcomes Following Pediatric Traumatic Brain Injury

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Objective: Traumatic brain injury (TBI) is a prevalent cause of long-term morbidity in children and adolescents and can lead to persistent difficulties with social and behavioral function. TBI may impact brain structures that support social cognition, social perception, and day-to-day social interactions—termed the social brain network (SBN). We examined differences in links among the SBN and regions of interest from other neural networks thought to support social outcomes, i.e., the default mode network (DMN) and salience network (SN). Furthermore, we examined how differences in co-activation among the SBN and these other key networks were associated with ratings of social and day-to-day adaptive outcomes.
Participants and Methods: Participants included children and adolescents with moderate to severe TBI (msTBI; n=11, M_{age}=11.78, 6 male), complicated-mild TBI (cmTBI; n=12, M_{age}=12.59, 9 male), and orthopedic injury (OI; n=22, M_{age}=11.69, 15 male). Participants underwent resting-state functional MRI on a 3Tesla Siemens Prisma scanner. Parents rated their child’s social and adaptive function on the Child Behavior Checklist (CBCL) and Adaptive Behavior Assessment System-Third Edition (ABAS-3). Resting-state connectivity was assessed using the CONN Toolbox, including preprocessing, denoising, and alignment to the participants’ processed T1 MPRAGE sequence followed by

https://doi.org/10.1017/S1355617723006598 Published online by Cambridge University Press

seed-to-voxel analysis using a SBN mask and targeted regions of interest within the DMN and SN. Individual-level r-to-z correlations were extracted from resulting clusters of co-activation with the SBN mask and exported into SPSSv28.0 for integration with behavioral data.
Results: One-way ANOVAs used to examine group differences in social and adaptive outcome revealed significant group differences in CBCL Social Competence (F=4.49, p=.019) and all composite scores on the ABAS-3 (Fs=3.78 to 5.17, ps=.031 to .010). In each domain, children with msTBI were rated as having elevated difficulties relative to cmTBI or OI, whereas cmTBI and OI groups did not differ. Connectivity also differed significantly between groups, with children with OI demonstrating greater connectivity between the SBN and the anterior cingulate cortex of the SN (t=5.19, p(FDR)<.0001) and posterior cingulate cortex of the DMN (t=4.30, p(FDR)<.001) than children with msTBI. Children with cmTBI also showed greater connectivity between the SBN and left temporal pole of the DMN (t=7.45, p(FDR)<.000001) than children with msTBI. Degree of connectivity between the SBN and posterior cingulate was significantly positively correlated across all domains of adaptive function (rs=.451 to .504, ps=.010 to .003), whereas degree of connectivity between the SBN and left temporal pole was strongly positively related to Social Competence (r=.633, p=.006) and conceptual adaptive skills on the ABAS (r=.437, p=.037).
Conclusions: Our findings provide insights into the neural substrates of social and adaptive morbidity after pediatric TBI, particularly msTBI, by linking alterations in connectivity among the SBN, DMN, and SN with measures of social and adaptive outcome. While the posterior cingulate was broadly associated with adaptive outcome, the temporal pole was particularly strongly associated with social competence. This may reflect the diverse functions and high degree of interconnectivity of the posterior cingulate, which contributes to various cognitive and attentional processes, relative to the strong amygdala/limbic connections of the temporal pole.

Categories: Acquired Brain Injury (TBI/Cerebrovascular Injury & Disease - Child)
Keyword 1: neuroimaging: functional connectivity
Keyword 2: social processes
Keyword 3: pediatric neuropsychology
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2 Infant Imitation: Detecting Risk in the First Year with PediaTrac™

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Objective: Imitation has pervasive associations with social and communicative development. However, few methods have been developed to measure this construct in typically developing infants, and even less is available for at-risk populations, such as infants born preterm. Autism spectrum disorder (ASD), a particular risk of premature birth, is associated with atypical imitation and social communication. Although imitation emerges in infancy, most current screening and diagnostic tools for ASD cannot be utilized prior to 12 months. The present study aimed to develop and validate a caregiver-report measure of infant imitation, characterize imitation profiles at 4, 6, and 9 months in term and preterm infants, and explore the relationship between imitation and scores on an ASD screening questionnaire at 18 months.
Participants and Methods: Participants (N = 571) were recruited from a larger multi-site study of PediaTrac™ v3.0, a web-based tool for monitoring and tracking infant development, and were surveyed longitudinally at birth, 2, 4, 6, 9, 12, 15, and 18 months. Participants completed the online PediaTrac™ survey and several reliable and validated questionnaires via pen-and-paper format. For the purposes of this study, only the Ages and Stages Questionnaire (3rd ed.; ASQ-3), Communication and Symbolic Behavior Scales-Developmental Profile (CSBS-DP), Brief Infant Sleep Questionnaire (BISQ), and the Modified Checklist for Autism in Toddlers – Revised with Follow-Up (M-CHAT-R/F) were examined. The following hypotheses were tested: (1) proposed imitation items will represent a unitary latent construct, for which convergent and discriminant validity will be demonstrated, (2) there will be measurement

https://doi.org/10.1017/S1355617723006598 Published online by Cambridge University Press