Social problems and brain structure trajectories following pediatric mild traumatic brain injury

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Introduction

Pediatric traumatic brain injury (pTBI), even mild (mTBI), is associated with risk of developing social problems, including aggressive behaviors and social cognitive deficits, which may profoundly impact the child's quality of life [Zamani et al., 2019]. The maturation of the neural bases of social behaviors and cognition, sometimes referred to as "the social brain", is protracted, continuing throughout childhood and adolescence, and correlates with increasing capacity for social information processing [Blakemore, 2012]. Therefore, pTBI may disrupt the expected developmental trajectory of the social brain structure that could manifest as development of social problems. However, limited sample sizes and cross-sectional designs generally used in the neuroimaging study of pTBI so far [Zamani et al., 2019], prevented from exploring developmental trajectories following pTBI. The present study aims to examine the longitudinal development of social problems and cortical thickness in social brain regions following pediatric mTBI using data from the large population-based cohort study Adolescent Brain Cognitive Development (ABCD) [Casey et al., 2018]).

Methods

Baseline and 2-year follow-up data from the ABCD 3.0 curated data release were used. Childhood history of mTBI was retrospectively assessed using the Parent Ohio State TBI Screen-Short Modified report [Bogner et al., 2017]. Age-corrected t-scores from the social problem subscale on the Child Behavior Checklist [Achenbach and Rescorla, 2000] were used to measure social problems. Cortical thickness was computed from T1-weighted images by the ABCD group using FreeSurfer 5.3 [Fischl, 2012]. Analyses focused on the following bilateral Desikan cortical regions of interest (ROIs) [Desikan et al., 2006] corresponding to core regions of the social brian [Blakemore, 2012]: medial orbitofrontal, temporal pole, inferior parietal, and bank of the superior temporal sulcus. Scanner (n=26) effects were removed before analyses using longitudinal ComBat [Beer et al., 2020]. Multigroup latent change score models were constructed with the lavaan 0.6-8 package [Rosseel, 2012] in R 4.1.0 to estimate latent difference scores between baseline and follow-up for social problems and social brain cortical thickness. Group differences in 4 parameters of interest (mean of the baseline score, rate of change over time, and variances of the baseline and of the change) were tested using chi-square tests [Kievit et al., 2018]. Child sex and parental education were included as covariates.

Results

The final sample included 5,736 controls (i.e., no history of TBI; 52% males, 9.97±0.62 years at baseline) and 224 mTBI (i.e., history of mTBI with loss of consciousness ≤ 30min or memory loss; 59% males, 9.99±0.64 years at baseline). Group differences were observed in both means and variances of the baseline values and rate of change in social problems (all $p \leq .002$). MTBI showed higher problems at baseline than the controls, and a decrease over time, but remained higher than the controls ($p = .03$), where problem scores stayed stable. Variances at baseline and in change were greater in mTBI than in controls. For all ROIs, no group differences were observed in either means or variances of the baseline values or rate of change in social brain cortical thickness (all $p \geq .17$). For all ROIs but the temporal pole, cortical thickness significantly decreased over the two years of the study in the two groups.

Conclusion

This longitudinal retrospective study revealed different developmental trajectories of social problems between adolescents with and without history of mTBI, but no difference in the trajectory of the social brain cortical thickness. Social problems decreased over time but remained high in mTBI, while stayed stable in controls. Cortical thickness decreased between age 10 and 12 years in both groups. Future studies should explore if changes in social brain function or other brain structure metrics may underly the changes in social problems after pTBI.

References

Achenbach TM, Rescorla LA (2000): Manual for the ASEBA preschool forms & profiles : an integrated system of multi-informant assessment. Burlington, VT: University of Vermont, Research Center for Children Youth, & Families.

Beer JC, Tustison NJ, Cook PA, Davatzikos C, Sheline YI, Shinohara RT, Linn KA (2020): Longitudinal ComBat: A method for harmonizing longitudinal multi-scanner imaging data. NeuroImage 220. https://pubmed.ncbi.nlm.nih.gov/32640273/.

Blakemore S-J (2012): Development of the social brain in adolescence. Journal of the Royal Society of Medicine 105:111–6. http://www.ncbi.nlm.nih.gov/pubmed/22434810.

Bogner JA, Whiteneck GG, MacDonald J, Juengst SB, Brown AW, Philippus AM, Marwitz JH, Lengenfelder J, Mellick D, Arenth P, Corrigan JD (2017): Test-Retest Reliability of Traumatic Brain Injury Outcome Measures: A Traumatic Brain Injury Model Systems Study. Journal of Head Trauma Rehabilitation.

Casey BJ, Cannonier T, Conley MI, Cohen AO, Barch DM, Heitzeg MM, Soules ME, Teslovich T, Dellarco D V., Garavan H, Orr CA, Wager TD, Banich MT, Speer NK, Sutherland MT, Riedel MC, Dick AS, Bjork JM, Thomas KM, Chaarani B, Mejia MH, Hagler DJ, Daniela Cornejo M, Sicat CS, Harms MP, Dosenbach NUF, Rosenberg M, Earl E, Bartsch H, Watts R, Polimeni JR, Kuperman JM, Fair DA, Dale AM (2018): The Adolescent Brain Cognitive Development (ABCD) study: Imaging acquisition across 21 sites. Developmental Cognitive Neuroscience 32:43–54.

Desikan RS, Ségonne F, Fischl B, Quinn BT, Dickerson BC, Blacker D, Buckner RL, Dale AM, Maguire RP, Hyman BT, Albert MS, Killiany RJ (2006): An automated labeling system for subdividing the human cerebral cortex on MRI scans into gyral based regions of interest. NeuroImage.

Fischl B (2012): FreeSurfer. NeuroImage 62:774–781. https://pubmed.ncbi.nlm.nih.gov/22248573/.

Kievit RA, Brandmaier AM, Ziegler G, van Harmelen AL, de Mooij SMM, Moutoussis M, Goodyer IM, Bullmore E, Jones PB, Fonagy P, Lindenberger U, Dolan RJ (2018): Developmental cognitive neuroscience using latent change score models: A tutorial and applications. Developmental Cognitive Neuroscience 33:99–117.

Rosseel Y (2012): **lavaan** : An *R* Package for Structural Equation Modeling. Journal of Statistical Software 48:1–36.

Zamani A, Mychasiuk R, Semple BD (2019): Determinants of social behavior deficits and recovery after pediatric traumatic brain injury. Experimental neurology 314:34–45. https://pubmed.ncbi.nlm.nih.gov/30653969/.