

# SOCIAL PROBLEMS AND BRAIN STRUCTURE TRAJECTORIES FOLLOWING PEDIATRIC MILD TRAUMATIC BRAIN INJURY

Dégeilh F<sup>1,2</sup>, von Soest T<sup>3</sup>, Ferschmann L<sup>3</sup>, Beer JC<sup>4</sup>, Gaubert M<sup>1,2</sup>, Koerte IK<sup>2,5</sup>, Tamnes CK<sup>3,6,7</sup>

<sup>1</sup> University of Rennes 1, CNRS, Inria, Inserm, IRISA UMR 6074, EMPENN – ERL U 1228, Rennes, France; <sup>2</sup> Department of Child and Adolescent Psychiatry, Psychosomatics, and Psychotherapy, University Hospital, Ludwig-Maximilians-Universität, Munich, Germany; <sup>3</sup> PROMENTA Research Center, Department of Psychology, University of Oslo, Norway; <sup>4</sup> Department of Biostatistics, Epidemiology and Informatics, University of Pennsylvania, Philadelphia, PA, USA; <sup>5</sup> Psychiatric Neuroimaging Laboratory, Brigham and Women's Hospital, Harvard Medical School, Boston, MA, USA; <sup>6</sup> NORMENT, Institute of Clinical Medicine, University of Oslo, Norway; <sup>7</sup> Department of Psychiatric Research, Diakonhjemmet Hospital, Oslo, Norway

fanny.degeilh@inria.fr  
<https://team.inria.fr/empenn/fanny-degeilh/>

## INTRODUCTION

- **Pediatric mild traumatic brain injury (mTBI)**
  - Highly prevalent
  - ↑ risk of developing social problems (e.g., aggressive behaviors and social cognitive deficits) ⇔ ↓ the child's quality of life [1]
- **Social brain**
  - Network of cortical regions supporting social cognition and behavior [2]
  - Social brain maturation (↓ thickness) from childhood to adolescence [2, 3]
  - Particularly vulnerable to TBI [4] ⇔ mTBI may disrupt the expected developmental trajectory of the social brain structure

However, limited sample sizes and cross-sectional designs generally used in the neuroimaging study of pediatric TBI so far [5], prevented from exploring developmental trajectories following pediatric TBI

## OBJECTIVE

To examine the **longitudinal development** of parent-reported **social problems and cortical thickness** in social brain regions **following childhood mTBI** (sustained between age 0-10 years) using data from the large population-based cohort study Adolescent Brain Cognitive Development (ABCD) study [6].

## METHODS



### Participants

- 224 children (132 [59%] boys, 9.99±0.64 years) with a history of mTBI with loss of consciousness ≤30 min or memory loss
- 5,736 controls (2,985 [52%] boys, 9.97±0.62 years)



### Childhood (0-10 years of age) history of mTBI

- Parent's retrospective report: Parent Ohio State Traumatic Brain Injury Screen-Short Modified [7, 8]



### Social problems

- Baseline & 2-year follow-up
- Child Behavior Checklist-Parent (CBCL) social problem age-corrected t-scores [9]



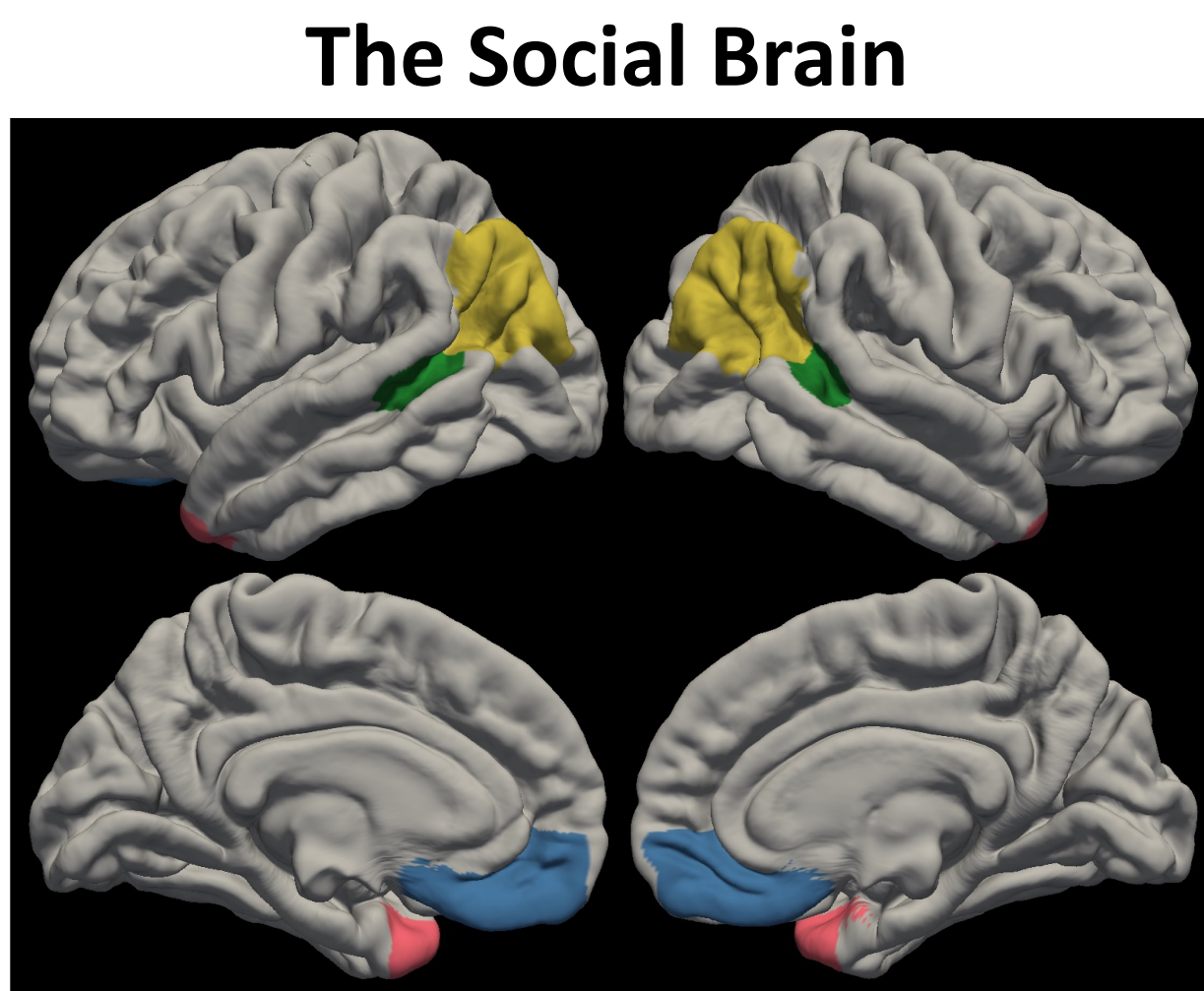
### Social brain cortical thickness

- Baseline & 2-year follow-up
- Pre-processed and quality checked cortical thickness from T1-weighted MRI data (FreeSurfer 5.3) [10]
- 8 regions of interest ⇔ core region of the social brain network [2] (Fig.1)
- Scanner effects (n = 26) corrected before statistical analyses using longitudinal ComBat [11] (Fig.2)

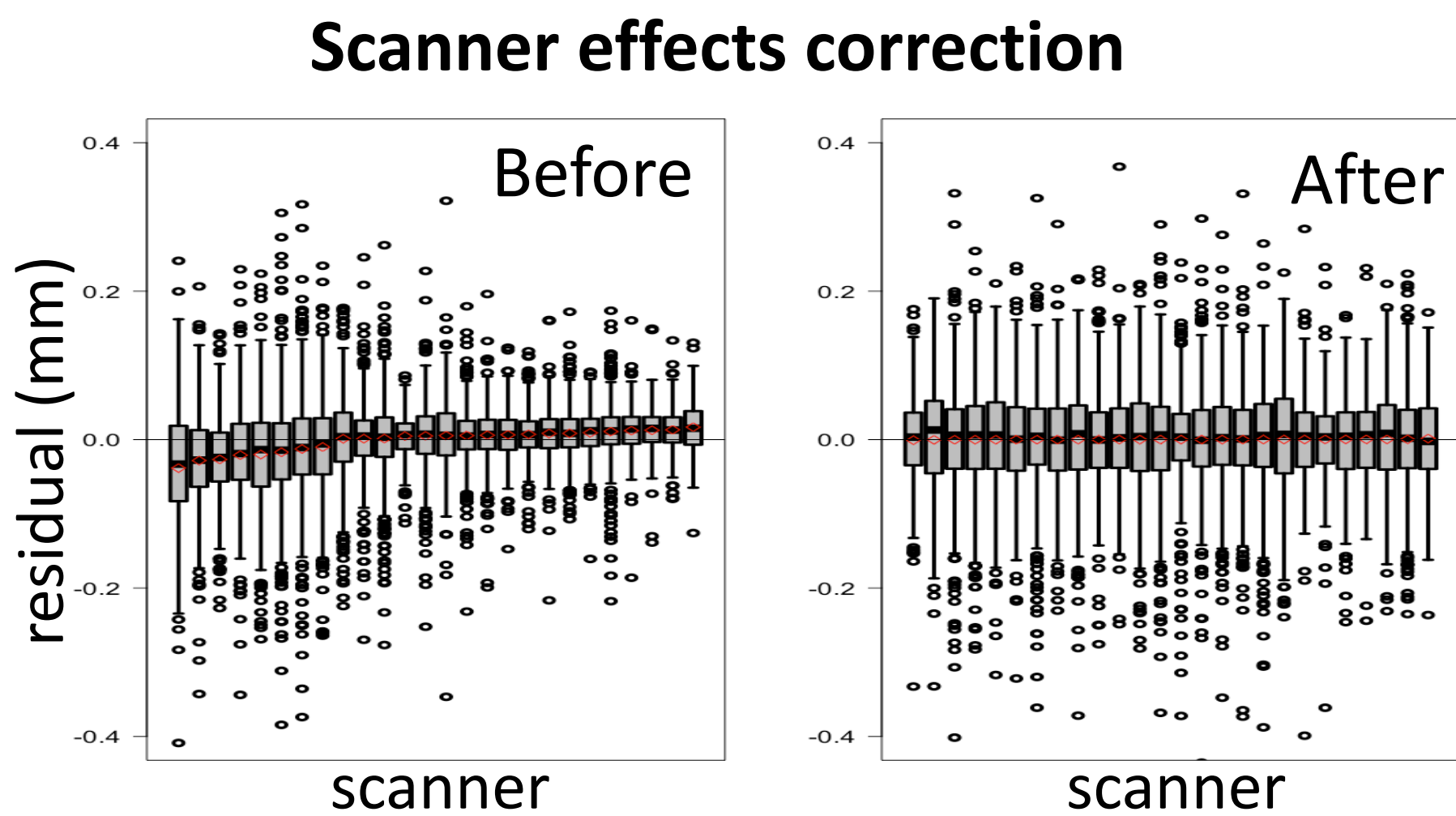


### Longitudinal changes modeling

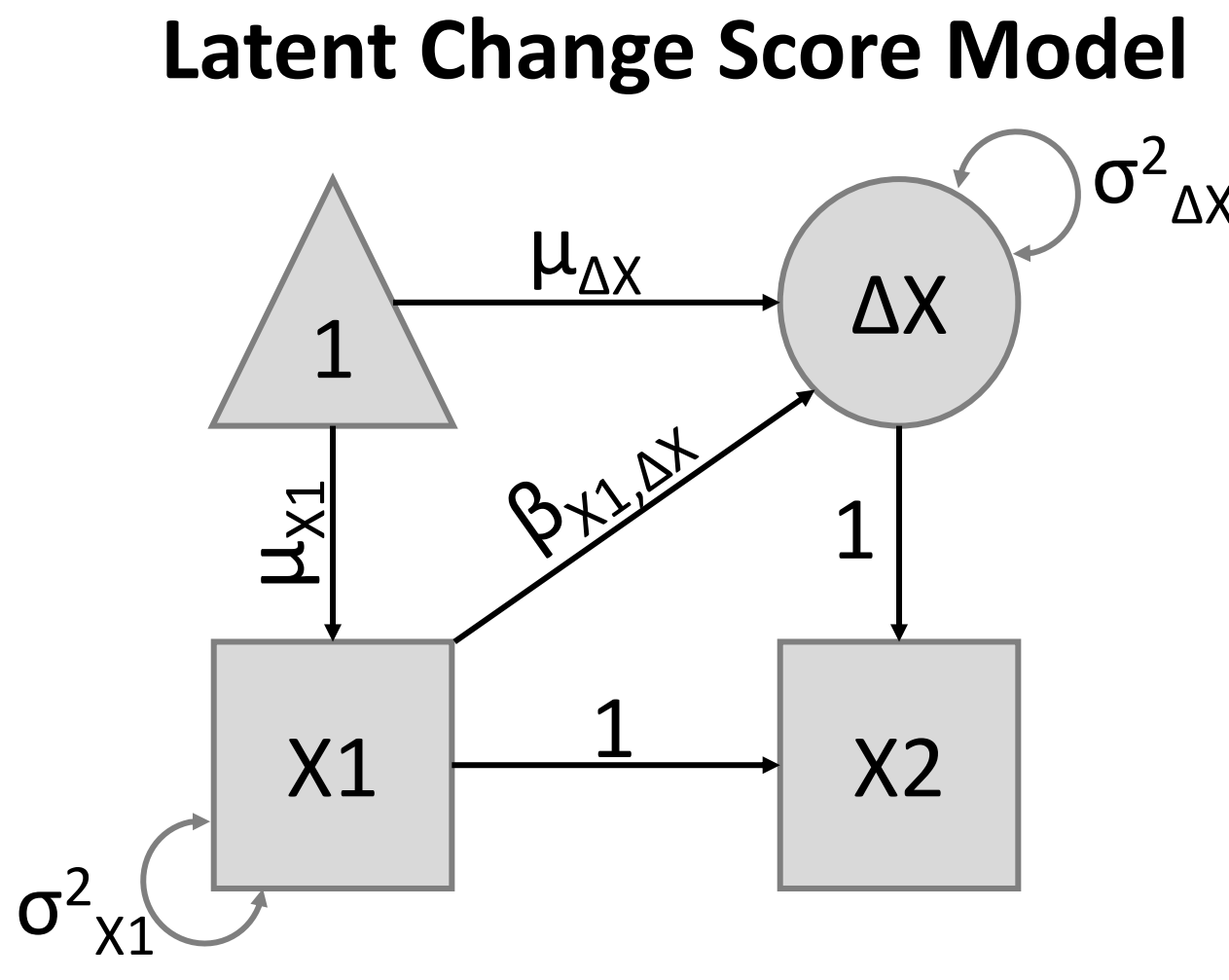
- Two-group Latent Change Score Model [12] (Fig.3) - lavaan 0.6-8 package [13], R version R4.1.0
- Chi-square tests ⇔ Group differences in mean and variance of the baseline and change scores
- Covariates: child sex and parental education



**Fig.1:** Social brain regions of interest. medial orbitofrontal (bleu); inferior parietal (yellow); superior temporal sulcus (green); temporal pole (pink)



**Fig.2:** Distribution of cortical thickness residuals and means (red) across scanner before and after ComBat. Model variables: child sex, parental education, group, time x group, and a subject-level random intercept.

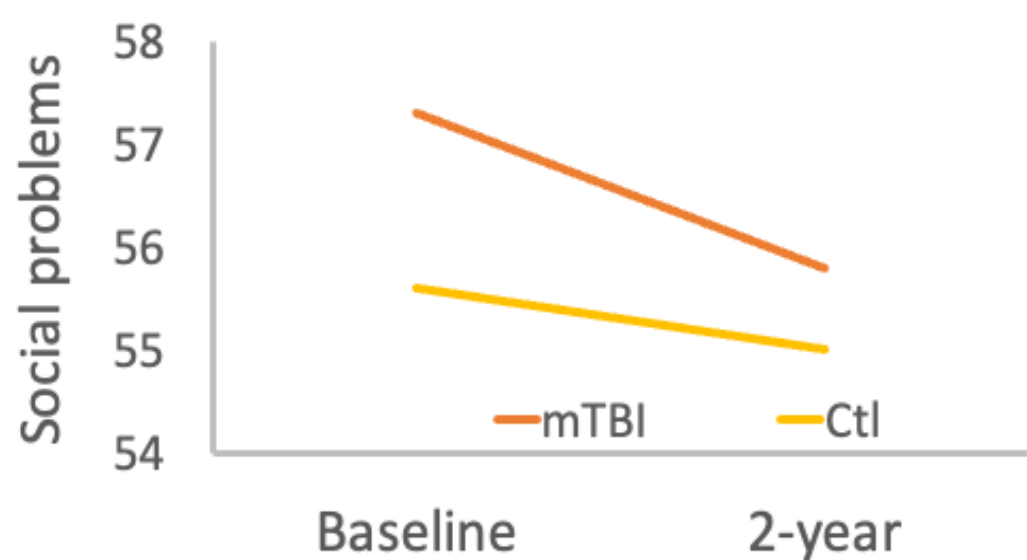


**Fig.3:** Latent Change Score Model. Social problems and cortical thickness measured at baseline (X1) and at 2-year (X2). Change (ΔX) is modeled as latent variable.

## RESULTS

### Social problems

- Group ≠ in both means and variances of the baseline & change scores (all  $ps \leq .002$ )



### Social brain cortical thickness

- No group ≠ in either means or variances of the baseline & change scores (all  $ps \geq .17$ )
- In both group and for all ROIs, but the temporal pole, ↓ cortical thickness by 0.05 to 0.11 mm over the 2 years of the study period (all  $ps \leq .001$ )

## DISCUSSION

Using an analytic method sensitive to longitudinal change at both individual and group level, this longitudinal retrospective study revealed :

- Different developmental changes in social problems between children with and without history of mTBI
- Higher levels of social problems at age 10 in children with mTBI than without history of mTBI
- ↓ social problems over time in children with history of mTBI, but remaining higher than in controls
- No difference in the trajectory of the social brain cortical thickness ⇔ ↓ between age 10 and 12 years in children with and without history of mTBI

## CONCLUSION

Prospective longitudinal studies with more detailed information on the TBI heterogeneity acute clinical profile (e.g., mechanisms of injury, acute symptoms, acute brain lesions) are needed to better understand the neural mechanisms underpinning social impairment following a childhood TBI.

## REFERENCES

- [1] Zamani et al., 2019; [2] Mills et al., 2014; [3] Tamnes et al. 2017; [4] Bigler, et al., 2013; [5] Zamani et al., 2019; [6] Casey et al. 2018; [7] Bogner et al., 2017; [8] Barch et al., 2018; [9] Achenbach and Rescorla, 2001; [10] Hagler et al., 2019; [11] Beer et al. 2020; [12] Kievit et al., 2018; [13] Rosseel, 2012.