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Registered Report

From pre- to post-natal brain asymmetry: callosal contribution and relationships with cognitive and genetic factors

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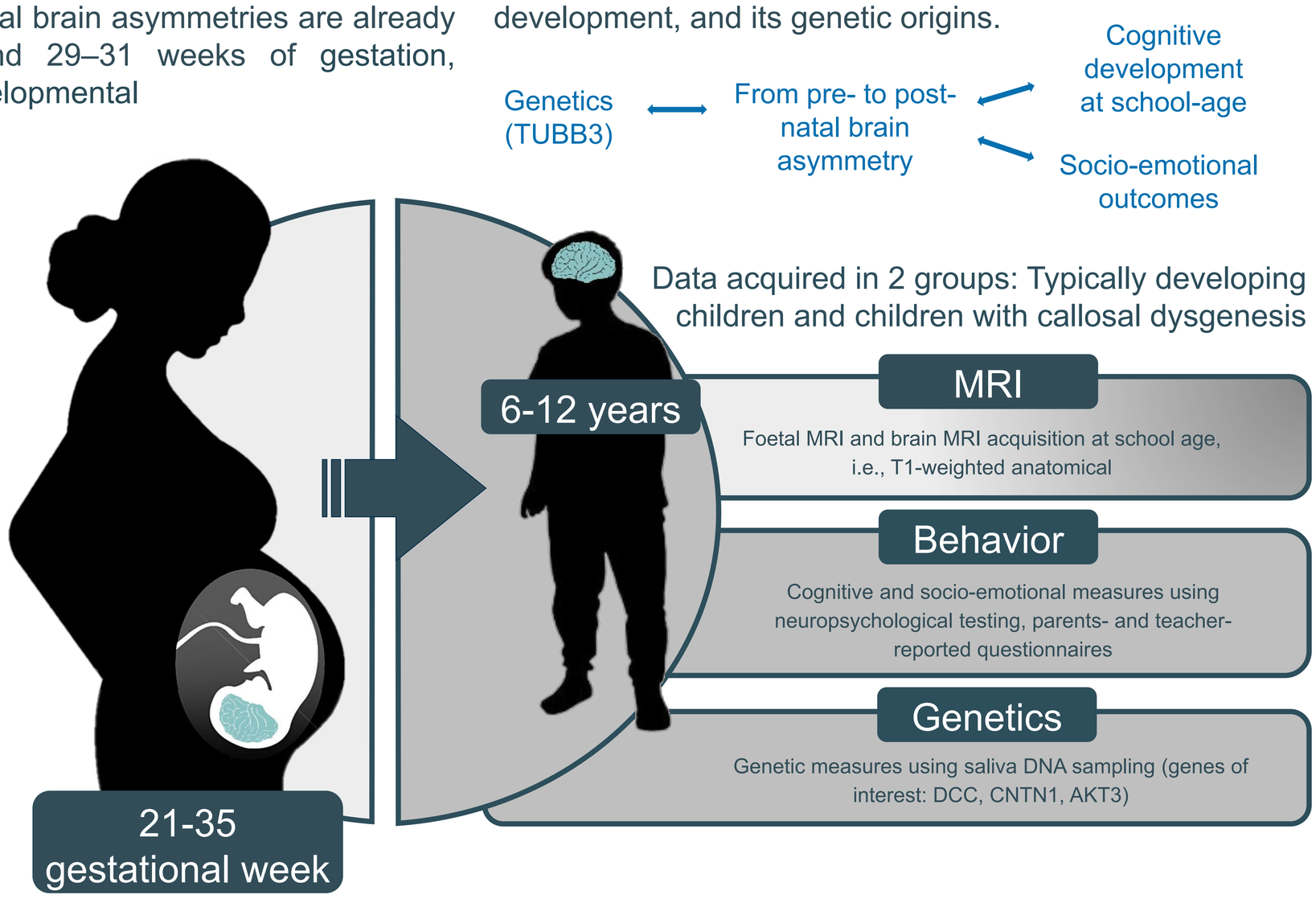
BACKGROUND

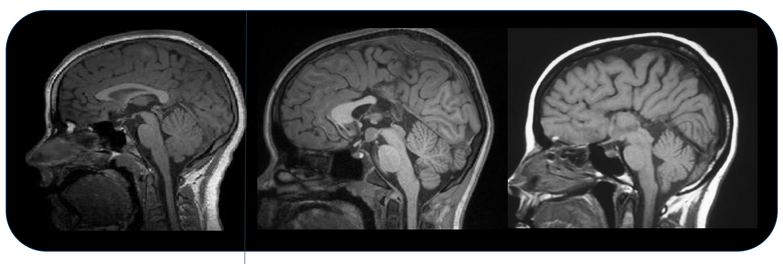
The human brain, despite its initial appearance of symmetry, harbours a variety of asymmetrical features that have captivated researchers for decades. Previous work in this area has established **strong associations between the emergence of brain asymmetry and the structure of the corpus callosum (CC)** [1,2]. Anatomical brain asymmetries are already apparent prenatally at around 29–31 weeks of gestation, reflecting an early genetic-developmental programme of left–right axis formation [3].

These asymmetrical features have implications for a wide range of cognitive and socio-emotional processes, and deviations in brain asymmetry have been associated with neurodevelopmental and psychiatric disorders [4]. However, the developmental trajectory of brain asymmetry spanning from prenatal origins to postnatal development lacks comprehensive understanding, including its interactions with corpus callosum integrity, its impact on cognitive and socio-emotional development, and its genetic underpinnings.

AIMS

To trace anatomical brain asymmetry from **pre- to post-natal period** in children with **typical development** and in children with **callosal dysgenesis**; to evaluate its relationship with callosal development, its role as a biological signature of cognitive and socio-emotional development, and its genetic origins.





60 typical brain | 30 CC dysgenesis / agenesis

Figure: T1-weighted mid-sagittal MR image of – from left to right – typically brain development, partial development (dysgenesis) of the corpus callosum and complete absence (agenesis) of the corpus callosum

METHODS

This study marks a pioneering effort in its scope and methodology. It involves pre- and post-natal longitudinal and multimodal data acquired in multiple center (CHUV and HUG). To tackle the research question, we will examine typical development of brain asymmetry from prenatal to postnatal period and compare it to children with callosal dysgenesis.

In both groups, we will investigate the **associations between brain asymmetry and cognitive** as well as **socio-emotional outcomes** using random effects models. Additionally, we will select specific genes known to play a role in the formation of the left-right axis and explore their associations with brain asymmetry (i.e., TUBB3 Single Nucleotide Polymorphism).

Translational Machine Learning Lab

Centre hospitalier universitaire vaudois

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References:
1.Gazzaniga, M. S. Cerebral specialization and interhemispheric communication: does the corpus callosum enable the human condition? Brain 123 (Pt 7), 1293–1326 (2000).
2.Karolis, V. R., Corbetta, M. & Thiebaut de Schotten, M. The architecture of functional lateralisation and its relationship to callosal connectivity in the human brain. Nat Commun 10, 1–9 (2019).
3.Sha, Z. et al. The genetic architecture of structural left–right asymmetry of the human brain. Nature Human Behaviour 2021 5:9 5, 1226–1239 (2021).
4.Toga, A. W. & Thompson, P. M. Mapping brain asymmetry. Nature Reviews Neuroscience 2003 4:1 4, 37–48 (2003).

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