

From Early Adversity to Brain Health: The Blossom Programme

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Early adversity may contribute to almost 30% of lifetime mental disorders¹. Its influence can extend across the life-course, affecting mental and physical health, cognition and brain ageing. The Blossom programme integrates population-scale, multimodal and cross-cohort data to identify reproducible pathways of risk and resilience and inform earlier identification and intervention.

WHY BLOSSOM?

- Early adversity includes stressful or potentially traumatic experiences, such as abuse, neglect, parental illness or socioeconomic hardship, that require substantial adaptation and deviate from the expectable environment².
- Its effects vary** by type, timing, duration, intensity and social context.
- Outcomes also differ**: similar experiences can lead to different mental, physical and brain-health trajectories.
- Mechanisms remain unclear**, partly because differences in measurement, populations and methods limit reproducibility.
- Blossom integrates biological, psychological and social data from large cohorts through harmonised, cross-cohort analyses.
- Our goal** is to identify reproducible pathways of risk and resilience to inform earlier identification, prediction and intervention.

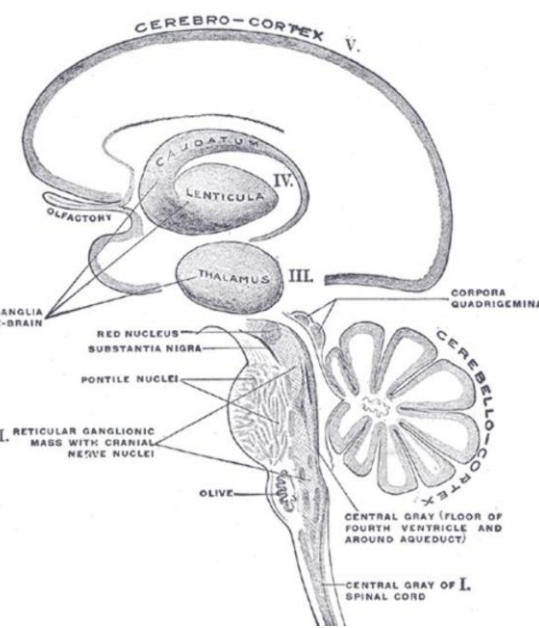


WHAT ARE WE LEARNING?

Specific experiences show distinct brain signatures

UK Biobank | N = 6,751³

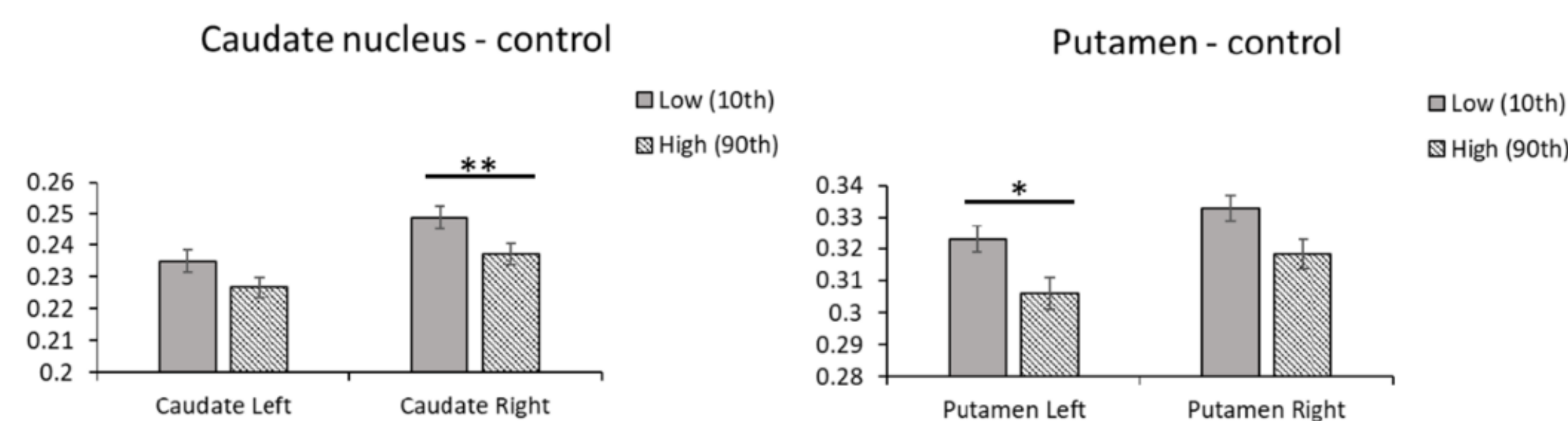
Reported childhood **emotional abuse** was associated with **smaller cerebellar and ventral-striatal volumes** decades later. No corresponding associations were found for physical abuse or emotional neglect.



Effects were small but indicated detectable, adversity-specific differences beyond traditionally studied brain regions, suggesting that cumulative scores may obscure important variation.

NSHD Insight 46 | N = 452⁴

Extreme perceived **maternal control** in childhood was associated with smaller **right caudate** and **left putamen** volumes, and with lower well-being at age 70. No corresponding brain-volume differences were found for maternal care or autonomy, highlighting the specificity of early caregiving dimensions.



Together, these findings suggest that specific early experiences may be associated with **distinct, long-lasting brain-structure differences**.

Different adversities relate to distinct later-life pathways

UK Biobank | N = 502,360 · ELSA | N = 12,653⁵

Across two cohorts, early adversity was most consistently associated with **depressive symptoms**, while associations with **physical health, lifestyle and cognition varied by adversity type and cohort**.

Education accounted for many associations, highlighting a potentially modifiable life-course pathway.

UK Biobank | N = 472,450⁶

Early adversity was associated with greater **mental health** challenges and physical neglect was uniquely associated with **poorer cognition**.

Coping-related behaviours and neuroticism accounted for several indirect associations between early adversity and adult mental health, cognition, and cerebrospinal fluid volume.

Together, these studies suggest that multiple adversity-specific psychosocial pathways, rather than a single mechanism, may contribute to different later-life outcomes.

Illustrative synthesis across studies



Emotional adversity may be linked to frontoparietal–default mode signatures

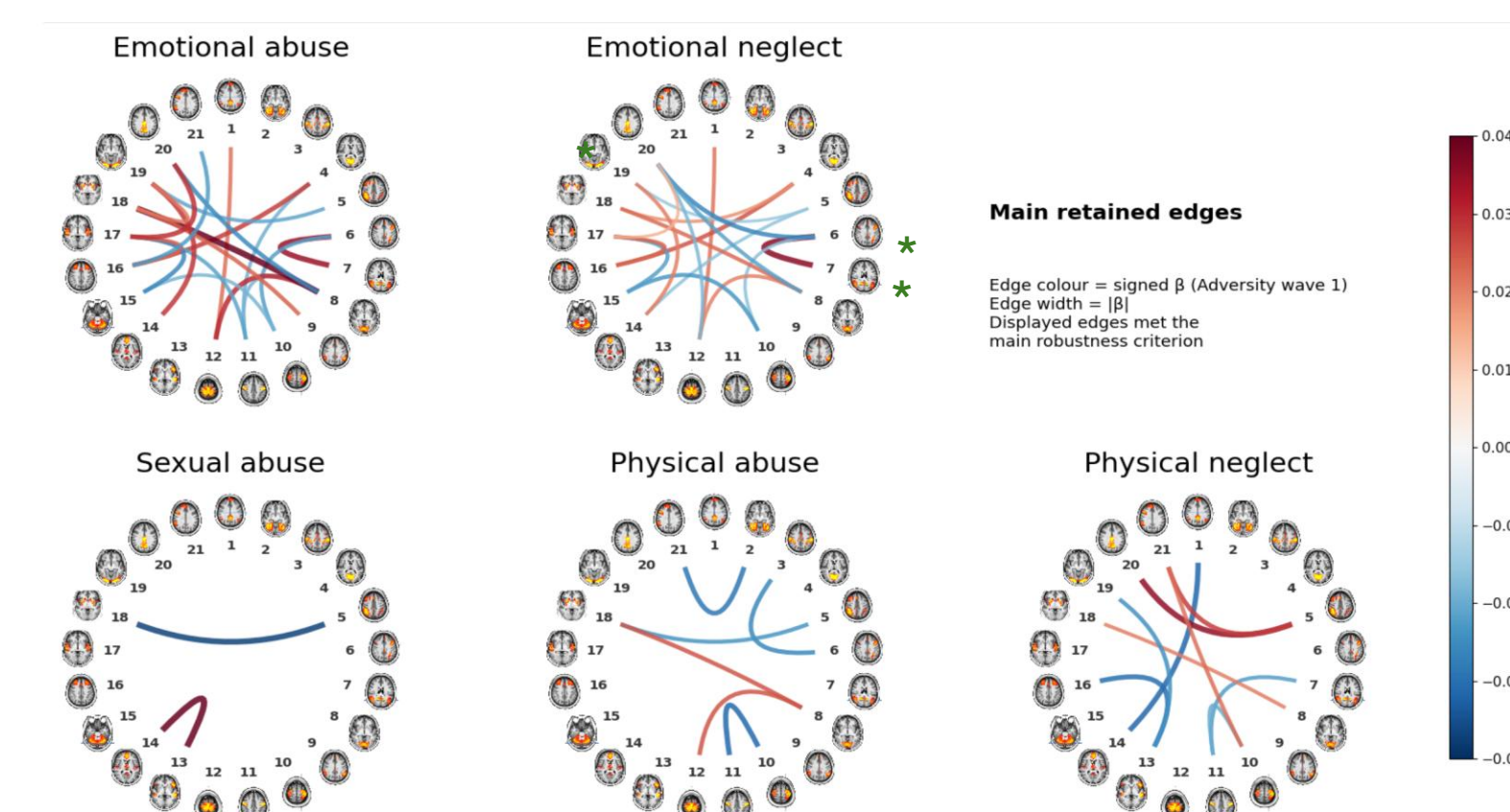
UK Biobank | N = 62,246

The robustness-first design used independent discovery (N = 58,021) and replication (N = 4,225) imaging samples.

Across five adversity domains and 210 resting-state connections, 53 associations were consistent across two adversity assessments conducted seven years apart; **68% involved emotional abuse or emotional neglect**. Effects were small and distributed across multiple large-scale brain networks.

After adjustment for current depression and anxiety, 17 associations remained, with **emotional neglect–left frontoparietal–default mode connectivity** showing the most consistent replicated association.

A validated **neuroticism polygenic risk score** is being used to test whether genetic susceptibility to stressful environments contributes to individual differences in these associations.



Sensitivity-retained adversity–connectivity associations



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Funding: DAG is supported through HORIZON-WIDERA-2022-TALENTS-02-01, project number 101090316; MK is funded through COMMUTE; Dementias Platform UK (DPUK) is funded by the Medical Research Council MR/T0333771

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