Tracing the impacts of early life adversity across the lifecourse: Brain, behaviour, and beyond

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Dimensions of Childhood Adversity and Midlife Heart Rate Variability in the 1946 British Birth Cohort
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Childhood adversity (CA) is linked to altered physiological and psychological stress systems, including subtle changes in vagal function and increased worry and rumination. Research focus has shifted from cumulative risk scores to dimensional models (i.e., deprivation, threat, and unpredictability) to better understand CA's impact on brain development and later psychopathology risk. Few studies have examined how dimensions of adversity affect autonomic nervous system (ANS) regulation. While findings on the link between CA and resting-state vagal function and reactivity remain mixed, apparent associations emerge when accounting for the modifying effect of age and current mental and physical health. This relationship remains largely untested in UK population survey data.

Heart rate variability (HRV), reflecting overall autonomic balance and adaptability to environmental changes, serves as a potential index for cardiac vagal tone, self-regulation, and unconscious stress.

Using data from the 1946 UK National Study of Health and Development birth cohort (n=5,362), this study will examine how CA dimensions (material and cognitive/social deprivation, threat, and unpredictability) relate to midlife HRV. Multiple imputation by chained equations will be used to impute missing data. Linear regression will be used to model HRV at age 64, accounting for confounders.

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Longitudinal associations between adversity, resilience and reward processing development in early adolescence – implications for anhedonia
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Background: Reward processing is an aspect of neurocognitive development which holds relevance for a variety of psychiatric disorders, particularly in the context of anhedonia – a transdiagnostic component of mood disorders and schizophrenia. The mechanistic relationship between early adversity, resilience and reward processing development is not well understood.

Method: Using 3 timepoints from the Adolescent Brain and Cognitive Development study (n = 11868), we constructed longitudinal mixed models to examine associations between previously derived adversity factors, potential markers of resilience, and reward responsiveness across ages 9-14. We fit similar models of striatal activation during phases of an fMRI monetary incentive delay task. General estimated equation models were used to identify adversity correlates of emergent anhedonia and low mood.

Results: Behavioural reward responsiveness was negatively associated with the "lack of caregiver support" factor ($p<0.001$) and "lack of supervision" ($p<0.001$); and positively associated with "youth report of family conflict" ($p<0.001$), prosocial behaviour ($p<0.001$) and positive school environment ($p<0.001$). Of the behavioural correlates "lack of supervision" was also negatively associated with striatal activation during reward anticipation ($p<0.001$). Behavioural reward responsiveness significantly decreased with age ($p<0.001$), as did both anticipatory ($p<0.001$) and consummatory ($p<0.05$) reward response in the nucleus accumbens. Correlates of emergent anhedonia were similar to that of emergent low mood – featuring both threat and deprivation related adversities.

Conclusion: We observed a typical trajectory of neural and behavioural blunting of reward systems with age during early adolescence in a large, diverse sample. Our findings also support previous work suggesting deprivation is associated with exacerbated reward blunting, whereas threat seems to be associated with increased reward sensitivity.

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Amygdala Burnout: Altered Trajectory of Amygdala Volume Associated with Early Life Stress

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Introduction:

Early life stress (ELS) plays a critical role in shaping brain development, especially affecting the amygdala—a key region in emotion and stress regulation. Research has linked ELS to both increased and decreased amygdala volume, but results vary due to differences in developmental timing. A lifespan model may help clarify these inconsistencies by tracking how amygdala volume evolves after ELS exposure.

Methods:

Human data from the UK Biobank (n=25,413; ages 40–69), excluding those with neurological illness, were analyzed using regression models to assess global amygdala volume changes associated with ELS. A focused subgroup (n=715) compared individuals with no ELS to those with extreme ELS exposure. Additionally, a voxel-based morphometry (VBM) region-of-interest analysis was conducted to detect voxel-wise volume differences in the amygdala. Both analyses controlled for age and sex.

In parallel, an experimental rat model (n=34; 17 stressed) exposed animals to restraint stress in adolescence (postnatal day 39), followed by MRI assessment. Deformation-based morphometry was used to measure volume changes in the central and basolateral amygdala nuclei.

Results/Conclusion:

Regression models revealed significant global reductions in amygdala volume in older adults with ELS (R^2 = 19.81%, p = .001). The VBM identified localized reductions in specific amygdala regions. Conversely, stressed rats showed short-term volume increases in the central amygdala. Together, these findings suggest a biphasic trajectory—early hypertrophy followed by later-life atrophy—supporting the hypothesis that prolonged ELS exposure may lead to amygdala "burnout" over time.

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