Commentary

Biological Psychiatry

Beyond the Absence of Distress: Episodic Memory Development and the Missing Half of Child Mental Health

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Middle childhood is a period of remarkable growth in episodic memory, the capacity to bind the what, where, and when of experience into retrievable events. It is also the period when internalizing symptoms first emerge for many children. Because the two share neural substrates, most prominently hippocampal-prefrontal circuitry, it has long been assumed that they are intertwined. However, which comes first has remained unclear. Do disruptions in memory systems confer risk for stress-related symptoms, or do symptoms constrain the development of memory? In the current issue of *Biological Psychiatry*, Foster *et al.* (1) take up this question in 3466 children from the Adolescent Brain Cognitive Development (ABCD) Study, followed from ages 9 to 10 years through 11 to 12 years.

Their results are clear in direction, if modest in size. In a bivariate latent change score model, higher baseline parent-reported stress-related symptoms predicted less improvement in a multi-indicator episodic memory factor over 2 years, whereas baseline memory did not reliably predict changes in symptoms. Using connectome-based predictive modeling, baseline resting-state connectivity predicted memory change in cross-validation. Connections weighted toward the cerebellum and subcortex, including dorsolateral prefrontal–hippocampal edges, predicted greater improvement, whereas connections weighted toward visual networks predicted less. Finally, early-life stress was related to memory change not directly but indirectly, through stress-related symptoms and, separately, through connectivity in these networks.

The study has considerable strengths. It is longitudinal in a field dominated by cross-sectional designs, it models change at the construct level rather than relying on a single task, it tests robustness across estimation methods, and it uses cross-validated prediction rather than in-sample association. The authors are also admirably candid about the small effect sizes and about the limits of reverse inference from resting-state data. I want to use this commentary to consider what the findings imply and what they leave out from the perspective of a science of human flourishing.

Symptoms First: A Hopeful Reading

The central claim that stress-related symptoms precede and predict slower memory development is usually framed as a warning. It can equally be read as a source of hope. If a child's affective state shapes the trajectory of a core cognitive capacity during middle childhood, then that trajectory is not fixed by early adversity or baseline brain organization alone. It is responsive to something that can change. Notably, early-life stress had no direct effect on memory change once symptoms and connectivity were modeled, suggesting that the consequences of adversity for memory are carried by processes that are themselves potentially modifiable. Decades of work on stress and neuroplasticity show that the same hippocampal and prefrontal systems that are vulnerable to chronic stress remain plastic and can be shaped by social context and deliberate intervention (2). The developing memory system described by Foster *et al.* (1) appears to be one such system.

Some caution about direction is warranted. With 2 waves of data, a latent change model can establish temporal precedence but not causation. The reverse path, from memory to change in symptoms, was near the threshold of significance, so the asymmetry is modest. Early-life stress, symptoms, and connectivity were all measured at baseline, making the mediation models effectively cross-sectional. Unmeasured factors such as sleep, which is critical for both memory consolidation and emotional regulation, could drive both trajectories. The claim in the title that psychopathology shapes memory development is best treated as a strong hypothesis that now calls for experimental tests.

The Missing Half: Measuring Flourishing, Not Only Distress

The more fundamental issue is what was not measured. The Child Behavior Checklist (CBCL) Stress Problems scale is strongly zero-inflated in this community sample. Most children score at or near the floor, and mean scores barely changed over 2 years. In practice, the study characterizes variation at the symptomatic end of the distribution and is silent about variation among the majority of children who have few or no symptoms. Symptoms were also reported by parents rather than by children, and parents are generally better informants about observable behavior than about their children's inner experiences. This limitation matters even more for well-being, which is best assessed from the child's own perspective.

However, the absence of distress is not the presence of well-being. The dual-continua model of mental health (3) holds that languishing and flourishing vary substantially among people who are free of psychiatric symptoms and that positive functioning predicts outcomes independently of symptoms. Children at the floor of the CBCL are not a homogeneous group. Some are curious, engaged, and deeply connected to others, and some are not. An obvious question follows from the present data: Do markers of positive functioning, such as

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positive affect, sense of connection, engagement, and purpose, predict better-than-expected memory development, and do they do so over and above the absence of symptoms? Large cohorts like ABCD are ideal places to ask this question, and the answer would tell us whether the relationship between mental health and cognitive development runs along one continuum or two.

The same asymmetry applies to the environment. Early-life stress was indexed as a count of potentially traumatic events, with a mean of fewer than one event per child. Positive childhood experiences, such as feeling able to talk with family, feeling supported by friends, and feeling a sense of belonging, predict adult mental and relational health in a dose-dependent way that is independent of adversity (4). The senior author's own work has shown that caregivers can buffer the developing amygdala-prefrontal circuitry in children (5). Testing whether such protective factors moderate the pathway from adversity to symptoms to memory would move the field from cataloging risk to identifying the conditions that allow children to thrive despite it.

Memory as a Resource for Resilience

Episodic memory is treated here as an outcome that stress impairs. It is also a resource that supports well-being. The ability to recall specific positive experiences underlies savoring, gratitude, and the construction of a coherent and hopeful self-narrative; it draws on the same hippocampal-prefrontal systems highlighted in this study. In adolescents at elevated risk for depression, greater specificity of positive memories was associated with lower cortisol and fewer negative self-cognitions in response to later stress (6). The emotional recognition task used by Foster *et al.* (1) includes happy, fearful, and neutral faces, but valence was collapsed into a single factor. Examining whether stress-related symptoms selectively affect the development of memory for positive material and whether preserved positive memory protects children exposed to adversity would be a natural and illuminating next step.

The network findings, although necessarily speculative, point in a compatible direction. Dorsolateral prefrontal coupling with the hippocampus in the network predicting greater improvement, the predictive value of default mode connectivity in the sensitivity analyses, and the apparent disadvantage of visually weighted connectivity together suggest a developmental shift from perception-bound encoding toward more internally organized, self-referential memory. These are the same large-scale systems that support autobiographical thought and the capacity to imagine the future, both of which are central to meaning and purpose.

From Prediction to Cultivation

The connectivity effects are small (cross-validated $r \approx 0.07–0.08$), explaining $<1\%$ of the variance in memory change. This is typical of brainwide association studies, which require thousands of participants to detect reproducible effects (7). Such findings offer insight into mechanisms at the population level, but they do not provide biomarkers that can be used for individual children, and they should not be presented as such.

The more actionable lever in these data is psychological. If stress-related symptoms constrain memory development, then interventions that reduce distress and cultivate positive capacities may protect it. There is growing evidence that the core components of well-being, including awareness, connection, insight, and purpose, can be understood as skills that are trainable and supported by neural plasticity (8). In preschool children, a brief mindfulness-based kindness curriculum improved social competence and self-regulation (9). The field has also learned that such programs are not universally effective. A large trial of school-based mindfulness training in early adolescence found no advantage over the usual socioemotional teaching (10). This suggests that timing, delivery, and fit to developmental stage are critical. The window studied by Foster *et al.* (1)—middle childhood, before internalizing symptoms consolidate—may be an especially promising target.

Two concrete recommendations follow. First, longitudinal cohorts and trials should measure positive functioning alongside symptoms, so that the full range of child mental health, and not only its deficit end, can be linked to brain and cognitive development. Second, trials of interventions that reduce stress symptoms or cultivate well-being in children should include episodic memory and network-level connectivity as outcomes. Such trials provide the experimental test of direction that observational cohorts cannot.

Foster *et al.* (1) have shown that a child's emotional life is entwined with the development of one of the mind's most fundamental capacities. A developmental system this sensitive to distress is likely to be just as responsive to cultivated well-being. The task ahead is to measure both.

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References

1. Foster JC, Hardi FA, Keding TJ, Sisk LM, Mishra S, Goldfarb EV, Gee DG (2026): Stress-related psychopathology shapes episodic memory development in youth: Network-level brain predictors and relations with early-life stress. Biol Psychiatry 100:1190–1198.
2. Davidson RJ, McEwen BS (2012): Social influences on neuroplasticity: Stress and interventions to promote well-being. Nat Neurosci 15:689–695.
3. Keyes CLM (2002): The mental health continuum: From languishing to flourishing in life. J Health Soc Behav 43:207–222.
4. Bethell C, Jones J, Gombojav N, Linkenbach J, Sege R (2019): Positive childhood experiences and adult mental and relational health in a statewide sample: Associations across adverse childhood experiences levels. JAMA Pediatr 173:e193007.
5. Gee DG, Gabard-Durnam L, Telzer EH, Humphreys KL, Goff B, Shapiro M, *et al.* (2014): Maternal buffering of human amygdala-prefrontal circuitry during childhood but not during adolescence. Psychol Sci 25:2067–2078.

6. Askelund AD, Schweizer S, Goodyer IM, van Harmelen A-L (2019): Positive memory specificity is associated with reduced vulnerability to depression. Nat Hum Behav 3:265–273.
7. Marek S, Tervo-Clemmens B, Calabro FJ, Montez DF, Kay BP, Hatoum AS, *et al.* (2022): Reproducible brain-wide association studies require thousands of individuals. Nature 603:654–660.
8. Dahl CJ, Wilson-Mendenhall CD, Davidson RJ (2020): The plasticity of well-being: A training-based framework for the cultivation of human flourishing. Proc Natl Acad Sci U S A 117:32197–32206.
9. Flook L, Goldberg SB, Pinger L, Davidson RJ (2015): Promoting prosocial behavior and self-regulatory skills in preschool children through a mindfulness-based kindness curriculum. Dev Psychol 51:44–51.
10. Kuyken W, Ball S, Crane C, Ganguli P, Jones B, Montero-Marin J, *et al.* (2022): Effectiveness and cost-effectiveness of universal school-based mindfulness training compared with normal school provision in reducing risk of mental health problems and promoting well-being in adolescence: The MYRIAD cluster randomised controlled trial. Evid Based Ment Health 25:99–109.