EDITORIAL

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# Editorial: The Preschool Emotional Brain

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There is broad consensus that children's ability to regulate emotion, particularly negative affect, can have enormous implications for the cascading processes underlying social and emotional development. With the burgeoning autonomy of toddlerhood comes a rudimentary understanding of the varieties of emotional experience, and initial awareness that a child's actions can augment or attenuate the intensity of those experiences. Successful forays into emotion regulation are crucial for healthy psychological development, allowing children to accommodate life's difficulties by purposefully altering their emotional state (ie, coping) when necessary. By contrast, persistent negative affect in childhood is known to increase the risk for depression by late adolescence.[1] Neuroimaging studies in youth and adults have implicated a key circuit in the generation and regulation of negative affect including the amygdala, a subcortical structure that detects emotionally salient information, and the medial prefrontal cortex (mPFC), a cortical region known to exert regulatory influence on the amygdala. Synchronous activation of these regions, reflecting functional transmission of information between them, is conceptually and empirically linked to individual differences in the intensity and purposeful modulation of emotion.[2] Furthermore, amygdala reactivity is associated with negative affect in preschoolers,[3] whereas emotion-related amygdala−mPFC connectivity may shape the subsequent development of resting (intrinsic) amygdala−mPFC connectivity, particularly in childhood.[4]

Yet, despite this circuit's critical relevance for socioemotional development, little remains known about how functioning in this circuit relates to childhood emotion regulation ability. In an important contribution to the field, Gaffrey *et al.* investigate the interrelationships of amygdala reactivity, amygdala−mPFC functional connectivity, and emotion regulation in a community sample of preschoolers.[5] Understanding the functioning of this circuit in early childhood has the potential to inform both our biological understanding of how internalizing disorders develop in childhood and our ability to better predict future risk of internalizing disorders by using objective biological measures. To that end, the authors recruited a cross-sectional sample of 132 preschool children (aged 4−6 years) from the community to participate in clinical assessments and neuroimaging. The sample was enriched for variance in caregiver-reported child depressive symptoms, although children were otherwise typically developing. Caregivers further reported on their child's ability to regulate emotional experiences, frequency of emotion dysregulation (negativity), and their own (maternal) depressive symptoms. All children underwent resting state functional magnetic resonance imaging (fMRI), whereas a subset completed an emotional face viewing task during fMRI. After stringent quality control checks, 66 children had usable resting state fMRI data, of whom a subset (n = 24) further completed the emotional face task. Of the 66 children, approximately one-third had a diagnosis of an internalizing and/or externalizing disorder based on assessment.

The authors next computed temporal correlations to identify brain regions with synchronous activation to the left and right amygdala. In resting-state data, connectivity of the right, but not the left, amygdala with the mPFC was linked to higher scores in caregiver-reported emotion regulation. Moreover, the strength of this connection was inversely correlated with caregiver reports of their child's negativity. Among the subset of children completing the emotional face task, those with depressive symptoms tended to exhibit greater right amygdala reactivity to emotion faces than those without. Similarly, amygdala reactivity to faces was positively correlated with overall negativity, as well as reduced resting state connectivity between right amygdala and mPFC. Notably, these findings remained when adjusting for factors including age, sex, and maternal depression. Finally, the authors conducted statistical mediation analyses to explore whether resting amygdala−mPFC connectivity mediated the relationship between amygdala reactivity and negative affect. Indeed, resting state amygdala−mPFC did mediate the relationship between amygdala reactivity and negative affect, although the reverse model did not hold significance. As above, this statistical mediation remained significant when adjusting for child age

and sex and maternal depression, suggesting that results were not simply due to bias in caregiver reporting of child symptoms.

As with any study, there are limitations that should be noted. As the authors point out, this is a modest sample size, particularly when examining the subset of children completing all tasks. Future studies with expanded samples of children will be needed to replicate and to extend these findings. Second, this study is cross-sectional and thus cannot provide information on neurodevelopmental trajectories underlying emotion regulation in early childhood.

Altogether, these findings add to a growing literature that is unveiling key neural substrates underlying emotional experience in early development. There are several key findings in this study of which readers should be aware. First, this study is one of the few implicating the amygdala−mPFC circuit in emotional reactivity and regulation in preschool children, a population that is understandably difficult to successfully recruit and to scan. Second, this study suggests that individual differences in the neural circuitry underlying emotion regulation are detectable at an early age. This is particularly exciting for its potential to serve as an additional marker that could help guide clinicians and policymakers in identifying at-risk children for early and timely intervention. Third, using statistical techniques, these findings suggest that amygdala−mPFC connectivity mediates the relationship between amygdala reactivity and persistent negative affect. This gives cause for optimism—your preschooler is not doomed simply because he or she has a hyperactive amygdala: rather, the prefrontal cortex is already capable, at this early age, of modulating the amygdala and achieving successful emotion regulation. Notably, the authors caution us to temper our optimism given the small sample size, particularly in the task-related (emotional faces) results. However, their ability to detect these effects given such limited power is encouraging for future research replicating these results in larger contexts.

So where do we go from here? Certainly, as stated, it will be important to expand this work to larger samples of children assessed longitudinally, ideally beginning in the prenatal period. Such studies, although challenging, will be crucial for identifying early life brain trajectories indicative of child mental health, akin to establishing a growth curve for the brain and psychological well-being in children and adolescents. We direct interested readers to the Healthy Brain and Child Development (HBCD) Study,[6] which will provide a unique opportunity to examine these very questions. Second, the dynamics of this circuit's operation, its integration in broader brain function, and its activity in different contexts remain unclear. The role of these structures in different brain networks and environmental operating contexts remains unclear. For example, what is the temporally causal flow of information in this and emotion regulatory circuitry? How does amygdala−mPFC connectivity interplay with the greater organizing networks of the brain (eg, default mode)? Under what contexts is this circuit recruited, and, just as important, when is it not? Third, how do caregivers influence the functioning and development of neural circuits underlying emotion regulation? Caregivers not only represent a crucial emotional buffer for children but can also sculpt a child's emotional development through their own modeling and guidance, a critical avenue for parenting interventions.[7] Clearly, it will be important for future studies of child development to capture as much information as possible on caregivers. This should include caregivers' own mental health history, emotion regulation abilities, and ideally even dyadic observational and biological measures such as neural synchrony.[8] Such studies can help us to better understand the most relevant building blocks in our children's emotional brains and to strengthen the connections, both inside and outside of the brain, that matter most.

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