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Research report

Behavioral activation sensitivity and default mode network-subgenual cingulate cortex connectivity in youth

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ABSTRACT

Increased resting-state functional connectivity (rsFC) between the default mode network (DMN) and subgenual anterior cingulate cortex (sgACC) is consistently reported in adults and youth with psychopathologies related to affect dysregulation (e.g. depression, posttraumatic stress disorder). This pattern of increased rsFC is thought to underlie ruminative thought patterns through integration of negative affect (via sgACC) into self-referential operations supported by the DMN. Neurobiological studies in adults show that behavioral activation system (BAS) sensitivity is a potential protective factor against the development of psychopathology, particularly in the context of stress and trauma exposure. However, whether BAS sensitivity is associated with variation in DMN-sgACC stress-vulnerability circuitry in youth, particularly those at risk for affect dysregulation, has not yet been studied. This association was tested in a sample of ninety-eight children and adolescents (ages 6–17) at high sociodemographic risk for psychopathology (i.e., urban, lower income, high frequency of violence and abuse exposure). Participants underwent a six-minute resting-state functional magnetic resonance imaging scan. Using a targeted, small-volume corrected approach, we found that youth with higher BAS sensitivity demonstrated lower DMN-sgACC rsFC, suggesting a potential link between the purported protective effects of BAS sensitivity and stress-vulnerability circuitry. This work suggests that interventions that augment BAS sensitivity, such as behavioral activation therapy, may protect against the development of stress-related psychopathology by modifying a critical rumination circuitry in the brain. Such interventions may be especially important for bolstering resiliency in at-risk urban youth, who are disproportionately burdened by early stress and associated psychopathology.

1. Introduction

Roughly one out of five children and adolescents [youth] in the U.S. are living below the poverty line [1] and over half of all youth report being exposed to one or more types of violence or abuse [2]. These negative early experiences constitute a significant risk factor for the development of psychopathologies related to affect dysregulation [e.g., mood disorders; [3,4]]. Fortunately, not every youth who experiences early stressful life events will go on to develop psychopathology [5]. In fact, only as many as 44% and 32% with a history of early stress exposure have been reported to develop childhood-onset and later-onset psychopathology, respectively [6–8]. Why is it, then, that some youth develop psychopathology while others maintain psychological well-being into adulthood, even when both have similar exposure histories? This particular question has motivated the fields of developmental neuroscience and biological psychiatry to identify potential factors that may increase a child's risk for, as well as protect against, the development of psychopathology following exposure to early stress [9–11].

Rumination is a well-established risk factor for emotional psychopathology in adults and in youth [12–14]. Rumination is the tendency to repetitively think about the causes and consequences of one's distressing symptoms, as opposed to its solutions [15] and is a symptom

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frequently observed in individuals suffering from depressive disorders [16,17]. Exposure to stressful life events during development has been associated with higher levels of rumination and was recently reported to mediate the longitudinal association between early stress exposure and increases in depressive and anxious symptomology among adolescents and adults [18]. In line with these findings, neuroimaging studies have identified functional alterations in the neural circuitry underlying rumination in adults and youth with depressive disorders [19–21]. In particular, patients with depressive disorders have increased resting-state functional connectivity (rsFC) between the default mode network (DMN) and subgenual anterior cingulate cortex [sgACC; [22,23]]. This pattern of rsFC may reflect ruminative thought patterns through integration of negative affect (via sgACC) into self-referential operations supported by the DMN [22]. Prior research suggests that sgACC-DMN rsFC increases from childhood to young adulthood [24,25] and heightened DMN-sgACC rsFC is associated with depressive disorder severity as well increased levels of rumination from adolescence to adulthood [26,27]. Further, heightened DMN-sgACC rsFC is predictive of treatment response [28], highlighting the relevance of this system for clinical and preventive interventions.

Like rumination, sensitivity of the behavioral activation system (BAS), which regulates motivated and approach behavior, reward-seeking, and positive affect [29], has also been associated with depressive disorders, but, unlike rumination, may be protective against psychopathology [30]. For instance, onset and severity of depressive symptoms following early stress are often predicted by low BAS sensitivity [31], whereas higher BAS sensitivity in youth has been associated with decreased risk for psychopathology following stressful life events [32–36]. Youth with increased BAS sensitivity report less negative affect and depressive symptoms [37], which may reflect a better ability to disengage from negative mood states and ruminative thought [18,38].

Given that the neural circuitry underlying rumination (i.e., DMN-sgACC) is altered in individuals with depressive disorders and higher BAS sensitivity is associated with lower rumination [39], a key unanswered question is whether higher BAS sensitivity is associated with lower (i.e., more normalized) DMN-sgACC rsFC. The purported protective effects of BAS sensitivity against the development of mood disorders may be associated with alterations in this circuitry. A possible link between DMN-sgACC circuitry and BAS sensitivity in youth, in particular, is critical given that affective disorders frequently begin during childhood and adolescence [40]. Identification of early potential neurobiological protective factors may provide novel mechanistic targets for interventions capable of circumventing the onset of psychopathology in at-risk youth.

Here, we tested whether BAS sensitivity affects DMN-sgACC rsFC. Importantly, we tested this prediction in a sample of youth at high risk (i.e., lower income, high incidence of violence and abuse exposure) for the development of stress-related affective disorders [3,4]. Based on prior studies showing heightened DMN-sgACC rsFC [22,23] and reduced BAS sensitivity [31,41] in individuals with depressive/affective disorders, we predicted that higher BAS sensitivity would correspond with *lower* DMN-sgACC rsFC. We tested for specificity of effects to BAS sensitivity by also evaluating effects of anxiety and depressive symptomology, and environmental risk factors (i.e., violence/abuse exposure, community distress, and household income) on DMN-sgACC circuitry.

2. Materials and methods

2.1. Participants

This study reports on ninety-eight youth (aged 6–17 years, 90% right-handed via parent report) recruited from Detroit, Michigan and the surrounding metro area. Participant demographics and characteristics are provided in Table 1. Parent-child dyads were recruited via community advertisements and local mental health clinics. Families were compensated for their time. All procedures were approved by the Wayne State University Institutional Review Board. All parent and youth participants completed informed consent and/or assent, respectively.

Table 1
Participant demographics and characteristics.

	(*N* = 98)	
	M	*SD*
Age (years)	11.27	2.73
Tanner Stage	2.83	1.36
KBIT-2 Score (IQ)	101.51	16.45
Movement during scan		
– Maximum FD	4.47	6.46
– Mean FD	0.48	0.66
BAS Sensitivity (sum score)	40.22	5.13
SCR total score	21.91	16.07
CDI-S score	2.62	3.1
	N	%
Gender (female)	61	62.2
Ethnicity (Hispanic/Latino)	2	2.0
Race		
– African American	31	31.6
– Caucasian	23	23.5
– More than one race	6	6.1
– Not reported	36	36.7
Household annual income		
– $40,000 or less	49	50.0
– Above $40,000	43	43.9
– Not reported		
Trauma type endorsed		
– Exposure to domestic violence	35	81.4
– Exposure to other violence	15	34.9
– Neglect	11	25.6
– Sexual abuse	10	23.3
– Physical abuse	4	9.3
– Emotional abuse	1	2.3
– More than one type	24	55.8
Community distress score		
– 0–20	26	26.5
– 20–40	8	8.2
– 40–60	16	16.3
– 60–80	8	8.2
– 80–100	40	40.8
SCR total score above threshold for detecting pathological anxiety	36	36.7
CDI-S score above threshold for detecting pathological depression	39	39.8

KBIT-2, Kaufman Brief Intelligence Test; IQ, Intelligence Quotient; FD, framewise displacement; BAS, Behavioral Activation System; SCR, Screen for Child Anxiety Related Emotional Disorders; CDI-S, Children's Depression Inventory Short Form; Tanner scale measures self-reported pubertal maturation [42]. The mean score (2.83) corresponds to the 'early-mid' pubertal stage. Community distress scores were derived from the Distressed Communities Index (http://eig.org/dci). Ranging from 0 to 100, high scores represent high economic distress.

A relatively wide age range was included to examine neural correlates of BAS sensitivity prior to and during adolescence, a critical developmental period that is considered to be high risk for the development of depressive symptomology [40]. Thus, we additionally tested for effects of age on neural correlates of BAS sensitivity, which may shed new light on developmental changes in protective factors and/or periods of sensitivity for intervention.

Given inherent differences in comprehension ability across the wide age range, participants were assisted by research staff in completing all self-report measures. Specifically, research staff read questions aloud to the participants, provided explanation and clarification when necessary, and made sure younger children understood the questions.