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Special Issue Article

Resting state coupling between the amygdala and ventromedial prefrontal cortex is related to household income in childhood and indexes future psychological vulnerability to stress

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Abstract

While child poverty is a significant risk factor for poor mental health, the developmental pathways involved with these associations are poorly understood. To advance knowledge about these important linkages, the present study examined the developmental sequelae of childhood exposure to poverty in a multiyear longitudinal study. Here, we focused on exposure to poverty, neurobiological circuitry connected to emotion dysregulation, later exposure to stressful life events, and symptoms of psychopathology. We grounded our work in a biopsychosocial perspective, with a specific interest in "stress sensitization" and emotion dysregulation. Motivated by past work, we first tested whether exposure to poverty was related to changes in the resting-state coupling between two brain structures centrally involved with emotion processing and regulation (the amygdala and the ventromedial prefrontal cortex; vmPFC). As predicted, we found lower household income at age 10 was related to lower resting-state coupling between these areas at age 15. We then tested if variations in amygdala–vmPFC connectivity interacted with more contemporaneous stressors to predict challenges with mental health at age 16. In line with past reports showing risk for poor mental health is greatest in those exposed to early and then later, more contemporaneous stress, we predicted and found that lower vmPFC–amygdala coupling in the context of greater contemporaneous stress was related to higher levels of internalizing and externalizing symptoms. We believe these important interactions between neurobiology and life history are an additional vantage point for understanding risk and resiliency, and suggest avenues for prediction of psychopathology related to early life challenge.

Keywords: amygdala, brain, poverty, psychopathology, stress

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Child poverty is common and a serious threat to development, as lower socioeconomic status (SES) is associated with an increased risk for a host of mental health difficulties (Cohen, Janicki-Deverts, Chen, & Matthews, 2010; Reiss, 2013). This is true for both internalizing and externalizing psychopathology, as poverty is associated with greater aggression, oppositional behavior, depression, and anxiety (Letourneau, Duffett-Leger, Levac, Watson, & Young-Morris, 2013; McLaughlin et al., 2011; Piotrowska, Stride, Croft, & Rowe, 2015). Although these linkages have been well studied across different disciplines, the developmental pathways of this poverty-related risk are poorly understood.

Surveying a growing body of multidisciplinary research conducted from a biopsychosocial perspective, stress exposure is a strong predictor of psychopathology (e.g., (Ozer, Best, Lipsey, & Weiss, 2003) and may be important to consider when conceptualizing associations between poverty and psychopathology. Throughout development, higher stress is common in low-SES contexts. For example, studies of large adult cohorts (of several thousand) have noted strong links between socioeconomic position and negative life events (Lantz, House, Mero, & Williams, 2005; Orpana & Lemyre, 2004). Looking at children and families, the frequencies of negative life events are also greater in low-income contexts (Dubow, Tisak, Causey, Hryshko, & Reid, 1991; Guerra, Huesmann, Tolan, Van Acker, & Eron, 1995). By one estimate, low-income families experience 35% more negative life events in 1 year than their middle-income counterparts (Attar, Guerra, & Tolan, 1994).

Thinking about stress exposure and psychopathology, experiences of early adversity (e.g., the multiple stressors associated with poverty and maltreatment) also may affect how individuals respond to subsequent life stressors and heighten risk for poor mental health (Hammen, 2018; Monroe & Harkness, 2005). For example, women with exposure to one or more childhood adversities (e.g., family violence, parental psychopathology) were more likely to become depressed following less total stress than women without such adversity (Hammen, Henry, & Daley, 2000). Additional evidence has accumulated validating these "stress sensitization models," with many groups finding interactions

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between childhood adversity and contemporaneous stress predicting significant increases in psychopathology (Dougherty, Klein, & Davila, 2004; Espejo et al., 2007; Harkness, Bruce, & Lumley, 2006; Kendler, Kuhn, & Prescott, 2004; Rudolph & Flynn, 2007). While these associations were initially seen for depression, stress sensitization effects are also present for posttraumatic stress disorder (PTSD) and other anxiety disorders ($N = 34,653$; McLaughlin, Conron, Koenen, & Gilman, 2010), as well as externalizing symptomatology ($N = 18,713$; Meyers et al., 2015).

Although we know that stress exposure is linked to psychopathology, and poverty is related to higher stress, more work is needed to identify how child poverty and stress exposure contribute to increased rates of psychopathology in lower SES contexts. In considering past research, there are three notable gaps in previous studies. First, the core psychosocial processes underlying stress sensitization are unclear. Emotion dysregulation may be one important pathway linking developmental variations in stress exposure to later psychopathology. The ability to regulate emotions is critical to successful development, with poor emotion regulation creating transdiagnostic risk for different forms of psychopathology (for conceptual discussion and review, see Beauchaine & Zisner, 2017). Second, these models have been most commonly talked about in samples exposed to maltreatment, but stress sensitization could similarly be taking place in the context of child poverty. For example, research teams (see McCrory & Viding, 2015) focused on maltreatment have argued that this type of adversity may create a "latent vulnerability" for poor mental health. Third, few studies focused on stress sensitization have centered on neurobiological processes. Initial research found that early adversity (specifically child maltreatment) increases reactivity to acute stress through physiological pathways, such as alterations in blood pressure (Gooding, Milliren, Austin, Sheridan, & McLaughlin, 2015; Leitzke, Hilt, & Pollak, 2015), cardiac output (McLaughlin, Sheridan, Alves, & Mendes, 2014), and cortisol release (Heim, Newport, Mletzko, Miller, & Nemeroff, 2008; Tarullo & Gunnar, 2006). However, few studies have specifically examined how early adversity may relate to changes in the brain, and then how these changes in the brain, in the context of more contemporaneous stress, increase the incidence of psychopathology. Such gaps are important to fill as the brain is positioned to mediate the effects of external stressors, especially those psychosocial in nature, on such physiological pathways. To these ends, by considering neurobiological and environmental risk factors as interactive diatheses that cut across multiple developmental spheres to influence outcomes (e.g., Starr, Hammen, Conway, Raposa, & Brennan, 2014), important progress might be made in conceptualizing, treating, and, ultimately, preventing the mental health challenges commonly seen in those exposed to poverty and related stressors.

Emotion Dysregulation: Core Processes and Connections to Psychopathology

In thinking about stress sensitization effects, emotion regulation likely plays a critical role in our responses to stress, by helping individuals motivate and organize behavior (Cicchetti, Ackerman, & Izard, 1995). Regardless of one's current stress load or developmental history of exposure to stress, an individual must regulate his or her emotional responses (volitionally or unconsciously), all in the service of adaptive behavior (Eisenberg & Spinrad, 2004; Thompson, 1994). This response is multifaceted in nature, involving monitoring, evaluating, and modifying our emotional states and expressions to appropriately fit different contexts and situations (Gross, 1998; Ochsner & Gross, 2005). Emotion dysregulation is a pattern of emotional experience and/or expression that interferes with accomplishing a goal (Beauchaine, 2015). This may be due to challenges in cognitive, social, or behavioral processes, including shifting attention from an overarousing situation, failing to use memories and associations when needing to change emotions, struggling to reappraise emotionally negative situations, or failing to take instrumental action to relieve frustration (Cole, Hall, & Hajal, 2013).

The consequences of emotion dysregulation may be quite profound, as this may cause emotions to interfere with adaptive behavior, that are contextually inappropriate, and/or that change too abruptly or too slowly (Cole et al., 2013). Emotion dysregulation could then contribute to maladaptive decision making and interpersonal behaviors, as well as cause significant distress for individuals (Leshin & Lindquist, 2019). Given these connections, it is perhaps not surprising that emotion dysregulation plays an etiological role for many different mental health issues (Aldao, Gee, De Los Reyes, & Seager, 2016; Hostinar & Cicchetti, 2019). This is true for disorders on both the internalizing and externalizing spectrum (including major depressive disorder, generalized anxiety disorder, alcohol-related disorders, and substance-related disorders; see Aldao, Nolen-Hoeksema, & Schweizer, 2010, for strong meta-analytic work on the topic).

Poverty and Emotion Dysregulation: Direct and Indirect Pathways

Considering the developmental pathways leading to emotion dysregulation, environmental experiences may impact critical processes and systems relevant for emotion processing and regulation. As a starting point, early parenting practices, caregivers' reactions to children's emotions, and the overall quality of a parent–child relationship may influence emotion regulation (Morris, Silk, Steinberg, Myers, & Robinson, 2007; Thompson & Meyer, 2007). Children may learn maladaptive patterns and be set on a trajectory toward emotion dysregulation through observing and modeling parents and peers (Calkins, & Hill, 2007; Crick & Dodge, 1994; Dodge, 2014). Regarding lower SES and poverty, this adversity has been found to increase emotion dysregulation by impacting youths' developing sense of self, coping skills, and interpersonal competencies (as reviewed in Wadsworth, Evans, Grant, Carter, & Duffy, 2016). Moreover, children from low-SES families tend to carefully monitor their environment for danger and maintain a low threshold for judging situations as threatening (Chen et al., 2006; Chen & Matthews, 2003). When confronted with ambiguous stimuli, whose threat value is uncertain, low-SES youth often exhibit larger cardiovascular responses than higher SES youth (Chen, Langer, Raphaelson, & Matthews, 2004).

In addition, there are a host of risk factors correlated with poverty, including harsher family climates, neighborhood violence, child maltreatment, and so on (Conger & Donnellan, 2007; Owens & Shaw, 2003). These experiences could all contribute to differences in attentional and arousal processes, as well as coping and deployment of emotion regulation strategies (Cicchetti, 2013; McLaughlin & Lambert, 2017; Messman-Moore & Bhuptani, 2017). Such patterns have been confirmed in investigations working at multiple levels of analysis (including psychophysiology and neurocognitive assessments; for review, see Pollak, 2015), as well as in longitudinal investigations (e.g., Kim & Cicchetti, 2010; Kim-Spoon, Cicchetti, & Rogosch, 2013). Collectively, whether

it be through parents and peers, higher levels of cumulative stress, or potential exposure to harsh family climates, poverty and lower SES could increase emotion dysregulation and then contribute to risk for psychopathology. These changes could then leave impoverished youth vulnerable in the face of future stress.

Emotion Neurobiology: Basic Processes and Relations With Early Adversity

In thinking about neurobiological processes related to emotion dysregulation and stress sensitization, there is strong evidence underscoring the importance of two critical nodes in the corticolimbic brain circuit in multiple emotion-related processes: the amygdala and the ventromedial prefrontal cortex (vmPFC). Situated in the anterior portion of the temporal lobes, the amygdala is an information-processing hub, involved in both physiological and behavioral responses to environmental and social challenges (Hariri, 2009; Ledoux, 2000). This includes, but is not limited to, detecting potential environmental danger and adjusting levels of vigilance. Meta-analyses of functional neuroimaging studies in humans find the amygdala is activated by a number of negative emotions, including the processing of anger and fear (Lindquist, Wager, Kober, Bliss-Moreau, & Barrett, 2012). Furthermore, individuals with various mood and anxiety disorders (e.g., major depressive disorder, generalized anxiety disorder, and PTSD) have shown greater amygdala responses to facial displays of fear and anger than individuals without psychiatric disorders (Etkin & Wager, 2007; Hamilton et al., 2012; Karl et al., 2006; Woon & Hedges, 2009). Relevant for models of stress sensitization, a longitudinal study in young adults found amygdala activity interacted with stress exposure to predict internalizing symptomatology 1 to 4 years later (Swartz, Knodt, Radtke, & Hariri, 2015). Individuals with higher amygdala activity and higher exposure to stress had the greatest increase in symptoms of depression and anxiety over this time period.

Exerting a top-down, inhibitory influence on the amygdala, the vmPFC is a portion of the prefrontal cortex (PFC) that aids in emotion regulation (Davidson, Putnam, & Larson, 2000; Ghashghaei & Barbas, 2002; Milad & Quirk, 2012). Work in nonhuman animals suggests damage to the vmPFC impairs the ability to decrease (and eventually extinguish) behavioral responses to cued fear conditioning (for review, see Milad & Quirk, 2012; Sotres-Bayon, Cain, & LeDoux, 2006). Supporting these results, neuroimaging work in humans has found increases in activity in the vmPFC occurring during the extinction of cued fear responses (Kalisch et al., 2006; Phelps, Delgado, Nearing, & Ledoux, 2004) and the reappraisal of negative emotions (Johnstone, Van Reekum, Urry, Kalin, & Davidson, 2007; Urry et al., 2006). Moreover, the structure and function of the vmPFC are related to different mental health issues, including forms of addiction, depression, and PTSD (for review, see Hiser & Koenigs, 2018).

Turning to potential neurobiological alterations related to lower SES contexts and the stressors associated with poverty, a number of investigations have focused on the brain circuitry involved with emotion processing and regulation, such as the amygdala and the vmPFC. For example, there have been multiple reports of smaller volumes in the amygdala and the vmPFC in relation to childhood exposure to poverty (Edmiston et al., 2011; Hanson, Nacewicz, et al., 2015; Holz et al., 2015; Luby et al., 2013). Relatedly, Dufford and Kim (2017) found reduced fractional anisotropy, a measure of structural integrity, in white matter tracts connecting the vmPFC and the amygdala after exposure to poverty in middle childhood. In terms of amygdala function, there is evidence showing an association between child poverty and increased adult amygdala activation to emotional stimuli (Gianaros et al., 2008; Javanbakht et al., 2015). Similarly, SES-related measures have also been linked to differences in vmPFC activation during functional magnetic resonance imaging (fMRI) tasks. In a sample of adults aged 31–54, lower parental education was related to reduced dorsal anterior cingulate–vmPFC and dorsolateral PFC–vmPFC connectivity during positive feedback in a reward task (Gianaros et al., 2011).

It is important to note that while many studies found that childhood poverty is associated with alterations in the structure and function of the amygdala and the vmPFC, fewer investigations have focused on the functional connectivity between these two brain regions. This is notable because disrupted amygdala–prefrontal functional connectivity has been reported in multiple forms of psychopathology (Gold et al., 2016; Stevens et al., 2013). Furthermore, many studies focused on other forms of adversity (e.g., maltreatment) have noted differences in the resting-state coupling between the amygdala and the vmPFC. For example, Herringa et al. (2013) found that maltreatment was related to lower amygdala–vmPFC connectivity in females at 18 years of age, and this altered connectivity mediated the development of internalizing symptoms. Earlier in development, exposure to more stressful life events was related to decreased functional coupling between the amygdala and the vmPFC in a sample of children between 4 and 8 years of age (Park et al., 2018). Analogous patterns have also been noted in urban youth, age 9–15, exposed to trauma (Thomason et al., 2015).

Integrating across these studies, alterations in vmPFC–amygdala resting connectivity may index diminished capacity to downregulate maladaptive emotional responses to environmental challenges. There has, however, been limited work focused on impoverished samples during childhood and adolescence. Studies focused on poverty and using resting-state connectivity could fill in these important gaps and provide new insights about the impact of experience on brain organization. Spontaneous brain activity (assessed at rest) is highly correlated between multiple brain regions, predicts task-response properties of neural circuits, and can identify subjects' aptitude for different cognitive tasks (Fox & Greicius, 2010). Thinking about stress exposure and sensitization, different forms of psychopathology often are being preceded by life events that possess a high degree of threat and unpleasantness (Hammen, 2005; Kessler, 1997; Monroe & Reid, 2008; Paykel, 2003); thus, effective regulation of negative emotion, potentially through vmPFC–amygdala pathways, is critical to preventing increases in symptoms of various mental health issues.

The Current Study

Motivated by these different bodies of research, we turn to an ethnically and socioeconomically diverse sample of youth currently enrolled in a longitudinal study of family processes and child development (the Parenting Across Cultures study). First, to investigate links between poverty and neurobiology, we examined the correlation between variations in SES measured in middle childhood and resting-state fMRI measured in adolescence. We specifically focused on amygdala–vmPFC connectivity as this functional coupling has been related to different forms of emotion dysregulation. On the basis of this past research, we hypothesized

that lower SES would be related to lower coupling between these brain regions. Second, we tested the hypothesis that exposure to more contemporaneous stress, at the time of the neuroimaging session, would moderate the association between vmPFC–amygdala connectivity and psychopathology. We also investigated the competing hypothesis that vmPFC–amygdala coupling would be related to different symptom-level indicators of psychopathology (without moderation by recent stress exposure).

Method

Participants

Participants for this neuroimaging project were recruited from a larger, prospective longitudinal study of parenting and child adjustment (the Parenting Across Cultures [PAC], study; see http://parentingacrosscultures.org for an overview). Specifically, participants were recruited from Durham, North Carolina, formerly a manufacturing hub in the tobacco industry and still, largely, a working-class city. Durham's population is 250,000, located in a larger metropolitan area of North Carolina with a population of 1.2 million. For the full PAC study, university institutional review board approval and approval from the appropriate elementary school authorities was obtained and then families were recruited from 15 socioeconomically diverse public and 2 private elementary schools using recruitment letters written in both English and Spanish. Participating families were African American, European American, and Hispanic. The full Durham PAC cohort was 311 families: 109 European American, 103 African American, and 99 Hispanic. Following this initial assessment, children and parents were interviewed annually; as of this writing, 8 waves of interviews have been completed. For this neuroimaging project, all participants at the Durham site were offered the opportunity to participate in this supplemental study. Ninety-two families chose to participate in this neuroimaging subproject. Compared to the full PAC sample, youth in this subproject were more likely to be European American ($\chi^2 = 9.1$, $p = .02$) and from more affluent households ($t = 2.2$, $p = .02$), but did not differ on other variables of interest (all other ps > .07). Of note, early family income was missing from 5 participants; therefore, our sample size with usable data was $n = 87$. Table 1 presents the demographic characteristics of these families at the initial assessment. Table 2 shows the interrelations between our variables of interest.

Table 1. Descriptive statistics for demographic variables

Variables	Statistic
Sex	
Male	56.5% (*N* = 52)
Female	43.5% (*N* = 40)
Ethnicity	
European American	47% (*N* = 43)
African American	29% (*N* = 27)
Hispanic	24% (*N* = 22)
Participant age (start of the study)	
Mean (*SD*), years	9.02 (0.531)
Range	7.83–10.1
Participant age (at scanning session)	
Mean (*SD*), years	15.2 (0.671)
Range	13.6–16.9
Participant age (at follow-up)	
Mean (*SD*), years	16.4 (0.615)
Range	15.06–17.63
Household income (mean, *SD*)	6.66 (2.89)
Youth internalizing (mean *T*-score, *SD*)	
Males (near scanning session)	49.04 (11.73)
Males (at follow-up)	51.38 (12.38)
Females (near scanning session)	54.08 (13.94)
Females (at follow-up)	61.25 (15.50)
Youth externalizing	
Males (near scanning session)	46.81 (9.67)
Males (at follow-up)	49.52 (10.09)
Females (near scanning session)	47.92 (10.75)
Females (at follow-up)	50.25 (11.86)

Note: Household income measured on 1–10 scale (1 = up to $5,000; 2 = between $5,000 and $10,000; 3 = between $11,000 and $15,000; 4 = between $16,000 and $29,000; 5 = between $30,000 and $40,000; 6 = between $41,000 and $50,000; 7 = between $51,000 and $60,000; 8 = between $61,000 and $70,000; 9 = between $71,000 and $80,000; and 10 = beyond $81,000).

Procedure

Study design and data overview

The current study was a supplemental project that recruited PAC participants 6 years after the start of that project. Here, we connected neuroimaging to longitudinal data collected at four PAC study time points: when participants were approximately age 10, 13, 15, and 16 years of age (Study Waves 2, 5, 6, and 7). The Wave 2 and 6 time points leveraged parental self-reports (participant age: 10 and 15 years of age). The Wave 5 and 7 time points focused on child self-report data (participant age: 13 and 16 years of age).

Self-report measures

Each time point focused on self-report questionnaires to assess experiences and behavior, including the Family Information Form (specifically information about yearly household income; Wave 2); the Life Events Scale (Dodge, Pettit, & Bates, 1994; Wave 6); and the Youth Self-Report (Achenbach & Rescorla, 2001; Waves 5 and 7). Of note, we used parental reports (only) from the Family Information Form and the Life Events Scale. These different measures were correlated and connected to neuroimaging data collected near Wave 6 of the project (participants were between 13 and 16 years of age; additional information in Table 1).

SES. SES was measured via the Family Information Form. For this measure, parents selected an answer to the statement "Indicate the gross annual income of your family" on a 10-point scale with options ranging from *up to $5,000* to *beyond $81,000*. We specifically focused on yearly household income at PAC Wave 2 (mean participant age = 10.04 +/−0.54 years; range = 8.78–10.9), as this was the earliest study time point when this information was collected.

Table 2. Correlation matrix of variables

	Sex	Household income (age 10)	Internalizing (age 15)	Externalizing (age 15)	Life stress (age 15)	Internalizing (age 16)	Externalizing (age 16)
Sex	—	—	—	—	—	—	—
Household income (age 10)	β = 0.017	—	—	—	—	—	—
Internalizing (age 15)	β = 0.205	*r* = .185	—	—	—	—	—
Externalizing (age 15)	β = 0.069	*r* = .098	*r* = .606***	—	—	—	—
Life stress (age 15)	β = 0.108	*r* = −.184	*r* = .023	*r* = .006	—	—	—
Internalizing (age 16)	β = 0.339**	*r* =.197	*r* = .638***	*r* = .347*	*r* = .157	—	—
Externalizing (age 16)	β = 0.051	*r* =.020	*r* = .484***	*r* = .565***	*r* = .048	*r* = .595	—

Note: Associations with child sex were determined by constructing regression models where sex (coded as a binary factor, with 0 = *male*, 1 = *female*) was entered as an independent variable (and the other variable of interest entered as the dependent variable). Statistically significant correlations are noted with the following conventions: *$p \leq .05$; **$p \leq .01$; *** $p \leq .001$. Of note, the association between sex and internalizing symptoms at age 15 was $p = .06$.

Stressful life events. To identify stressful events that families had encountered in the past year, parents completed the Life Events Scale. Questions followed a dichotomous yes/no format for parents to indicate the occurrence of 19 difficult events. This list of life events included the following: moving; major repairs/remodeling to home; severe and/or frequent illness for any child in the home; accidents and/or injuries for any child in the home; other medical problems for any child in the home; medical problems for close family members; death of close family member (or other important person); divorce and/or separation for the child's parent and her/his husband/wife; parent and child were separated (due to illness, divorce, work, etc.); money problems that made it hard to pay for basic living expenses; legal problems; problems and conflicts with relatives; birth of a baby; problems at school for child; problems at work for parents; loss of a job; remarriage or marital reconciliation; and participation in any parenting programs. The total number of "yes" answers were summed, with higher scores indicating greater numbers of experienced stressful life events. Given our interest in "stress sensitization," we examined this variable at PAC Wave 6 (mean participant age = 15.4 +/−0.63 years; range = 13.82–16.44), which was near or after the collection of the neuroimaging data. Reliability for this measure was α = 0.43.

Child behavior. To measure problem behaviors, child participants completed the widely used Youth Self-Report. For this measure, adolescents completed the 29-item internalizing subscale and the 30-item externalizing subscale (Achenbach, Dumenci, & Rescorla, 2003). Informants rated to what extent each item is applicable (0 = *not true*, 1 = *somewhat true*, and 2 = *very true*). We examined this approximately 1 year after the neuroimaging session (mean participant age = 16.38+/−0.61 years; range = 15.07–17.62). We also used this measure before the neuroimaging session (mean participant age = 13.8+/−0.571 years; range = 12.42–14.83) to control for preexisting levels of problem behaviors. Reliability for this measure was high before and after the neuroimaging session (1 year after the neuroimaging session α = 0.95; before the neuroimaging session α = 0.94).

MRI data

Structural and functional MR images were acquired at the Duke-UNC Brain Imaging and Analysis Center on a 3.0 Tesla General Electric scanner (Signa EXCITE, GE Healthcare; Waukesha, WI, USA). In regard to brain anatomy, high-resolution T1- weighted anatomical images were acquired with 162 axial slices using a fast-spoiled gradient echo pulse sequence (repetition time = 7.584 ms; echo time = 2.936 ms; field of view = 256 mm; image matrix = 256 × 256; voxel size = 1 × 1 × 1 mm; flip angle = 121 degrees) and were used for normalization and co-registration with the functional data. During acquisition, this image was aligned in a near axial plane defined by the anterior and posterior commissures. Whole-brain functional images were then acquired using a SENSE inverse-spiral sequence (repetition time = 2000 ms; echo time = 32 ms; field of view = 256 mm; image matrix, 64 × 64; flip angle = 77 degrees; voxel size, 4.0 × 4.0 × 4.0 mm; 34 axial slices). The resting-state functional scan was 364 seconds long, and participants were instructed to rest comfortably with their eyes open while viewing a gray fixation cross. A semiautomated high-order shimming program ensured global field homogeneity.

Neuroimaging preprocessing and analysis. The acquired fMRI data were then processed by using tools in the FMRIB Software Library (FSL; http://www.fmrib.ox.ac.uk/fsl; Jenkinson et al., 2012; Smith et al., 2004) and were customized using Matlab code developed in-house (MathWorks, Natick, MA). This processing scheme was developed in service of long-term reproducibility of resting-state connectivity (see Chou, Panych, Dickey, Petrella, & Chen, 2012; information and scripts available at https://wiki.biac.duke.edu/biac:analysis:resting_pipeline). In brief, the first four volumes of each functional imaging data set were discarded to allow for magnetic field stabilization. Slice-timing correction, motion correction, intensity normalization, brain extraction, and smoothing (6-mm Gaussian blur) were then performed using FSL (version 5.0.1; specifically slicetimer, mcflirt, fslmaths, and bet). Of note, after motion correction, the six motion parameters were then regressed out of each individual voxel using linear regression. Participants' anatomical scans were then nonlinearly registered using FNIRT to the MNI-152 template (2 mm). Resulting warping parameters were then applied to the functional volumes. Next, the signal from white matter and cerebrospinal fluid masks (generated from FMRIB's Automated Segmentation Tool; FAST) were regressed out of the resting-state volumes, using the same method as the motion parameters. Finally, data were bandpass filtered between 0.008 and 0.1 Hz using custom python scripts. Motion was censored at 0.2 mm between frames, using the derivative and Euclidean

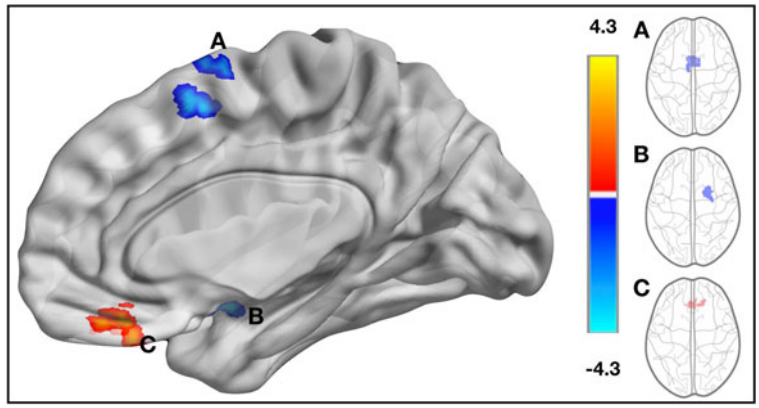

Figure 1. Early household income was related to significant differences in amygdala resting-state coupling. Lower income was associated with greater coupling between the amygdala and (a) the paracingulate gyrus and (b) the putamen. Lower income was correlated with lower coupling between the amygdala and (c) the ventromedial prefrontal cortex. A surface-based rendering is shown on the left side of the figure, while the right side of the figure uses "glass brains" to depict the extent of these associations. These areas were significant at $p = .05$, after correcting for multiple comparisons within a whole-brain gray matter mask (thresholded at 50%, initial uncorrected threshold $p = .01$).

norm. Participants were excluded if >20% of frames were censored ($n = 1$), yielding at least 5 min of resting-state data. Of further note, given recent controversies in the field, we chose not to regress global signal intensity (Saad et al., 2012).

We next extracted the time course of the amygdala at rest, using a bilateral probabilistic region of interest from the Harvard–Oxford subcortical atlas (distributed with FSL; thresholded at 50%). For each participant, a regression was performed including this bilateral amygdala time course in Analysis of Functional NeuroImages (AFNI; Cox, 1996), generating subject-level maps of the correlations between this region's time course and every other voxel's time course. Next, these subject-level maps were Fisher z-transformed and entered into a whole-brain linear regression as the dependent variable; household income when participants were approximately 10 years of age was entered as the independent variable. This whole-brain regression yielded t statistics at each voxel noting associations between resting-state coupling (for a bilateral amygdala seed) and household income.

For these analyses, we limited our search space using a whole-brain gray matter mask (thresholded at 50%). To correct for multiple comparisons, we deployed AFNI's 3dClustSim using cluster-size thresholding based on Monte Carlo simulation and new, mixed-model (non-Gaussian) auto-correlation functions and using an initial, uncorrected statistical threshold of $p < .01$ (Cox, Chen, Glen, Reynolds, & Taylor, 2017). Based on this threshold, the number of comparisons in our mask, and the smoothness of our imaging data, a minimum cluster size of 160 voxels was required to have a corrected $p \leq .05$. For any regions above this threshold, mean functional connectivity estimates were then extracted by averaging across every voxel in each regional cluster. These procedures are in keeping with other past reports from our group (e.g., Hanson et al., 2019).

Statistical analysis

Linear regression models were next constructed (outside of FSL and AFNI) using the R statistical package (http://cran.r-project.org). These models examined associations between household income, amygdala connectivity, child behavioral problems, and contemporaneous stress exposure. We examined associations between household income when participants were approximately 10 years of age (entered as an independent variable) and resting-state coupling for any brain regions above our multiple comparisons threshold (mean coupling for each regional cluster, entered as the dependent variable). While primarily confirmatory in nature, these regressions allowed us to visually check and test for potential outliers, and to see if the inclusion of child sex or ethnicity reduced any associations (both entered as independent variables). Next, based on our interest in stress sensitization, we tested if contemporaneous stress exposure moderated associations between child behavioral problems and amygdala connectivity (for any brain regions above our multiple comparisons threshold). To be concrete, in two separate regression models, child behavioral problems (internalizing or externalizing total scores, when participants were approximately 16 years of age) were entered as the dependent variable, while child sex and ethnicity, amygdala connectivity, contemporaneous stress exposure (when participants were approximately 15 years of age), and the interaction between amygdala connectivity and contemporaneous stress exposure were entered as independent variables. Finally, we examined associations between child behavioral problems when participants were approximately 16 years of age (internalizing and externalizing total scores, entered in separate models, as independent variables) and any brain regions above our multiple comparisons threshold (again, entered as the dependent variable). In full transparency, we did not predict that there would be simple (main effect) associations between psychopathology and resting-state variables; however, we wanted to complete these analyses as a form of "competing hypothesis" (in contrast to our predictions for stress sensitization).

Results

Whole-brain connectivity results

Voxelwise analyses of resting-state fMRI data revealed that the functional coupling for the bilateral amygdala was related to early household family income. Specifically, and as predicted, lower income at age 10 was related to lower connectivity between the bilateral amygdala and the vmPFC (peak at $x = -4$, $y = +36$, $z = -16$, cluster size = 187, max voxel $t = 4.153$) at age 15. Of note, this relation remained significant after controlling for race/ethnicity and sex ($t = 3.54$, $p < .005$). In addition to the lower coupling for vmPFC, early household family income was related to greater connectivity between the bilateral amygdala and two other brain regions: (a) the paracingulate gyrus (peak at $x = +10$, $y = +10$, $z = +50$, cluster size = 434, max voxel $t = 4.136$); and (b) the putamen (peak at $x = +22$, $y = +4$, $z = -8$, cluster size = 284, max voxel $t = 4.315$). Effects were, again, similar when controlling for race/ethnicity and sex (all ps $< .005$). These associations are depicted in Figure 1 (surface-based

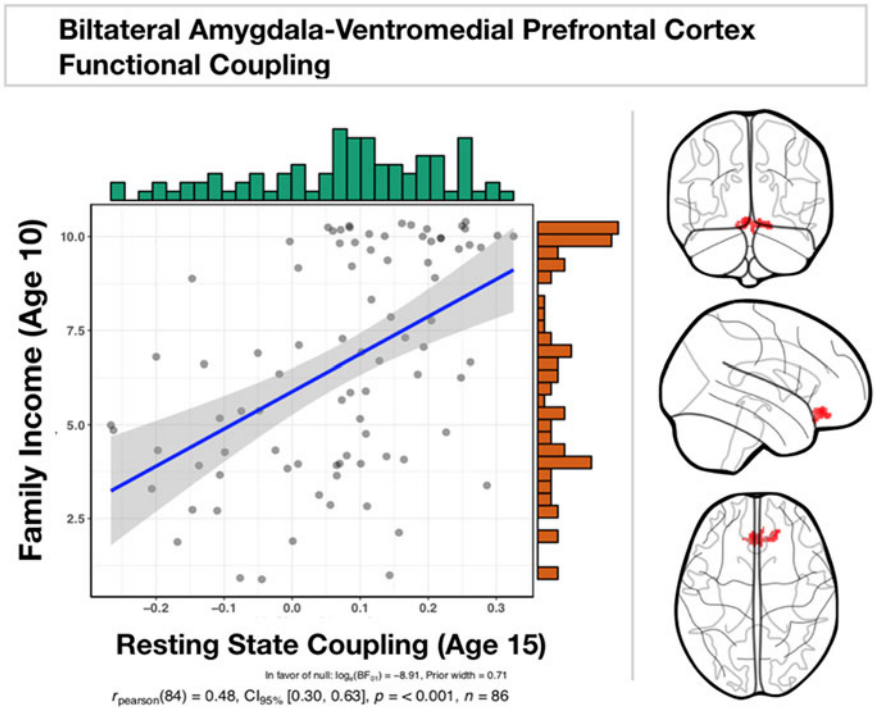

Figure 2. Scatterplots show relations between early household income and extracted resting-state coupling estimates for the bilateral amygdala and the ventromedial prefrontal cortex. This association was positive in nature, with lower income relating to lower coupling between these areas. The horizontal axis depicts resting-state coupling between regions (Fisher's *Z*-transformed correlation coefficients, with higher values indicating greater coherence between brain regions), while the vertical axis shows family income (on a 1–10 scale, with lower numbers indicating lower income). This scatterplot is shown on the left side of the figure, while the right side of the figure uses "glass brains" to depict the extent of these associations.

renderings). Figure 2 depicts a scatterplot for our a priori focus, amygdala–vmPFC resting-state coupling.

Moderation analyses (tests of stress sensitization)

Given that lower household income was related to differences in emotion regulatory neurobiology, we next sought to test the potential behavioral consequences of this brain difference. We investigated if brain differences interacted with later stress exposure to predict symptoms of internalizing psychopathology. For these analyses, we constructed linear regression models examining the main effect of amygdala–vmPFC resting-state coupling, stress exposure at or after the time of the neuroimaging session (parental report), and the interactions of these two factors. We predicted that higher rates of later psychopathology would be seen only when a participant had faced higher, recent stress and showed lower amygdala–vmPFC resting coupling. In line with our hypotheses, there was a significant interaction of amygdala–vmPFC resting-state coupling and stress exposure in predicting youths' later self-report of internalizing symptoms ($\beta = -0.274$, $t = 3.257$, $p = .0017$). There was no simple (main effect) association between internalizing and amygdala–vmPFC resting-state coupling ($p = .382$) or stress-exposure ($p = .518$). This interaction is shown in Figure 3. Simple slope analyses revealed that stress exposure had a significant and positive correlation with youth reports of internalizing when amygdala–vmPFC resting-state coupling was at below-average levels (at −1 *SD* below the mean of amygdala–vmPFC resting-state coupling, the simple slope = 3.241, $SE = 1.124$, $t = 2.88$, $p = .005$); however, at average or above average (+1 *SD*) levels of amygdala–vmPFC resting-state coupling, this relation was not statistically significant (at +1 *SD* above the mean of amygdala-vmPFC resting state coupling, the simple slope = −2.15, $SE = 1.43$, $p = .13$, and at the mean, the simple slope = 0.541, $SE = 0.98$, $p = .58$).

In addition to youth reports of internalizing, we also examined if brain differences interacted with later stress exposure (again, as measured by parental report) to predict symptoms of externalizing psychopathology. Similar to the results for internalizing symptomatology, there was a significant interaction of amygdala–vmPFC resting-state coupling and stress exposure in predicting youths' self-reported symptoms ($\beta = -0.203$, $t = 2.007$, $p = .048$). Again, there was no simple (main effect) association between externalizing and amygdala–vmPFC resting-state coupling ($p = .96$) or stress exposure ($p = .84$). However, in contrast to the simple slope analyses with internalizing symptoms, no simple slopes from the interaction groups were significant (−1 *SD* amygdala–vmPFC resting-state coupling, $p = .2$; mean amygdala–vmPFC resting-state coupling, $p = .79$; +1 *SD* amygdala–vmPFC resting-state coupling, $p = .18$). These effects were, however, in a similar direction, as stress exposure had a positive correlation with youth reports of externalizing when amygdala–vmPFC resting-state coupling was at below-average levels (at −1 *SD* below the mean of amygdala–vmPFC resting-state coupling, the simple slope = 1.07, $SE = 0.846$, $t = 1.27$).

Speaking to the specificity of these associations, interactions between stress exposure and resting-state coupling between the amygdala and other brain regions (paracingulate and putamen) were not significant in predicting internalizing (interactions of all ps < .48) and externalizing (interactions of all ps < .25) symptoms.

Competing analyses (nonmoderated models)

In a series of competing hypotheses, we also tested the associations between resting-state functional coupling and symptoms of psychopathology in regression models that did not include interaction terms. Related to our primary brain variable of interest, amygdala–vmPFC functional coupling was not significantly associated with internalizing ($p = .54$) or externalizing ($p = .84$)

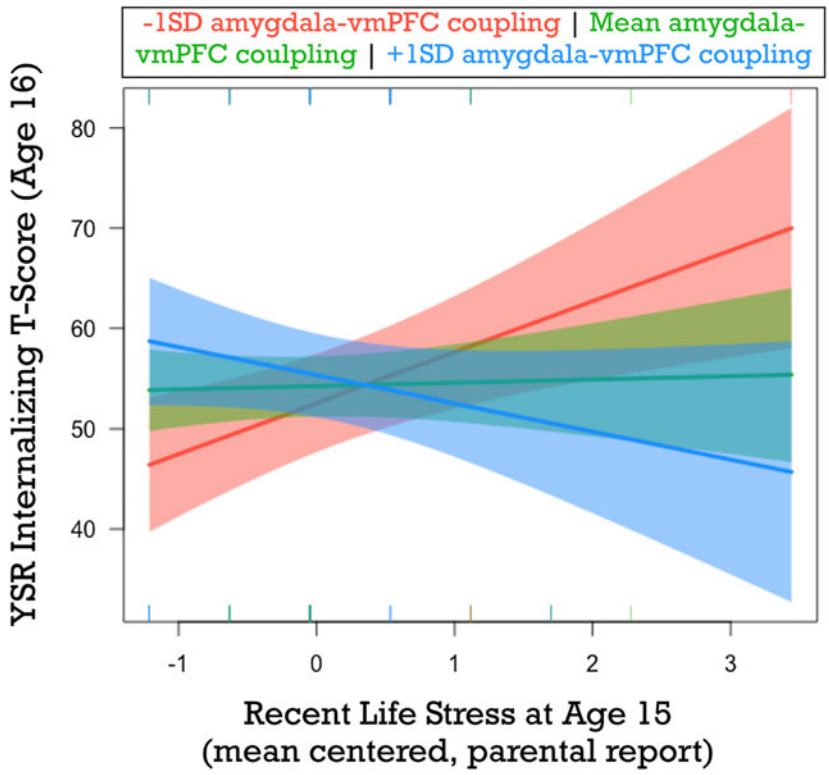

Figure 3. This graphic shows the interaction between amygdala–ventromedial prefrontal cortex (vmPFC) resting coupling and recent life stress, in predicting internalizing symptomatology. Recent life stress is graphed on the horizontal axis and internalizing symptomatology on the vertical axis. Levels of amygdala–vmPFC resting coupling are also shown, with lower (red), mean (green), and higher (blue) levels depicted in the figure. The interaction of amygdala–vmPFC resting coupling and recent life stress was related to greater internalizing problems ($\beta = -0.274$, $p < .005$).

symptoms. We also explored associations with psychopathology for amygdala–paracingulate coupling and amygdala–putamen coupling. There were no associations between these brain areas and internalizing symptoms (all *p*s < .49). Similarly, there were no significant relations between these regions and externalizing symptoms (all *p*s < .82).

Discussion

Grounding our work in stress sensitization and emotion dysregulation perspectives, we provide novel evidence regarding the developmental pathways through which lower SES and exposure to child poverty play a precipitating role in the development of poor mental health. We focused on poverty-related alterations in functional coupling between neurobiological nodes involved with emotion reactivity and regulation, and the degree to which these alterations sensitize individuals to the deleterious impacts of stressful life experiences. In regard to neurobiology, we found that lower household income in middle childhood (age 10) was related to lower coupling between the amygdala and the vmPFC in adolescence (age 15). Such a finding is particularly important given that past research has noted variations in this functional coupling are often related to different forms of emotion dysregulation. We also demonstrated that lower coupling between the amygdala and the vmPFC interacted with more recent exposure to stress, to predict increases in internalizing problems (age 16). There was also evidence that a similar interaction existed in relation to externalizing problems. Collectively, these results provide powerful evidence of the transactions between neurobiological, environmental, and psychosocial processes and how such interactions may convey risk for psychopathology.

Thinking first about the neurobiological correlates of household income that we noted, structural and functional alterations in the amygdala and the vmPFC may have significant behavioral consequences across development. Research in rodents indicates that portions of the vmPFC inhibit amygdala-based responses (Milad & Quirk, 2012). These patterns connect to a number of neuroimaging studies that suggest activity in the vmPFC and amygdala are inversely related during the extinction of conditioned fear (e.g., Kalisch et al., 2006; Phelps et al., 2004). Furthermore, research in healthy adolescents and adults has found greater vmPFC activity, in combination with reduced amygdala activity, to be associated with reduced negative affect and the intentional suppression of negative emotions (Johnstone et al., 2007; Pitskel, Bolling, Kaiser, Crowley, & Pelphrey, 2011; Urry et al., 2006; also as reviewed in Hiser & Koenigs, 2018). Altered functional connectivity between these regions may represent a lessened ability for "top-down" emotion regulatory structures, such as the vmPFC, to exert an influence on "bottom-up" emotion reactivity regions such as the amygdala. This lower influence may mean greater neural (and behavioral) responses to environmental threats and other negatively valanced stimuli.

Related to past research on early adversity, our reported results connect to and expand on both behavioral and neurobiological findings. In regard to the amygdala and the vmPFC, structural alterations have also been commonly reported in these regions for individuals exposed to different forms of early adversity, including child poverty (Hanson et al., 2010; Hanson, Nacewicz, et al., 2015; Holz et al., 2015; for review, also see Palacios-Barrios & Hanson, 2019). In terms of brain function, increased amygdala activation has been reported after exposure to child poverty (Kim et al., 2013), as well as child maltreatment (Dannlowski et al., 2012; McCrory et al., 2011). For functional connectivity, Herringa et al. (2013) found resting-state coupling between these regions was lower for adult female participants reporting experiences of maltreatment during childhood. Burghy et al. (2012) similarly reported that greater childhood

adversity predicted increased childhood cortisol levels, which then predicted lower amygdala and vmPFC coupling 14 years later. More recently, Park et al. (2018) found more adverse experiences, measured cross-sectionally, were related to lower amygdala and vmPFC coupling in middle childhood. These patterns fit with structural connectivity work by Dufford and Kim (2017) that found exposure to poverty was associated with reduced integrity in the white matter tracts connecting the vmPFC and amygdala in middle childhood.

Behaviorally, our results connect to past work on emotion dysregulation in impoverished samples. Exposure to poverty is connected to lessened emotion regulation and coping skills, differences in threat sensitivity, and altered cardiovascular responses to threat. Changes in the amygdala and vmPFC coupling may be neural markers of these behavioral processes and/or explain the development of negative behaviors over the course of development. More broadly, our findings suggest a neurobiological mechanism that may connect experiences of child poverty to later mental health issues. Altered functional connectivity within the brain's threat-detection and regulation circuitry may, over time, give rise to difficulties in the processing and regulation of emotion, causing poverty-exposed individuals to experience greater negative mood and affect especially after exposure to more contemporaneous stress.

This study also connects to past work on stress sensitization. Accumulating evidence has found contemporaneous life stress may interact with early adversities and then cause subsequent increases in symptoms and formal diagnoses of different mental health issues (e.g., internalizing and externalizing; Grasso, Ford, & Briggs-Gowan, 2012; McLaughlin et al., 2010). The strongest evidence of stress sensitization comes from different studies examining stress exposure in relation to self-report and clinical ratings of psychopathology. Additional work has also found changes in blood pressure, cardiac functioning, and the functioning of the hypothalamic–pituitary–adrenal axis in connection to multiple exposures to stress. While important, many of the past results can be seen as the output of stress-response systems. Fewer reports have provided neurobiological evidence for the idea of stress sensitization after child adversity. For example, Hanson, Knodt, Brigidi, and Hariri (2015) found that the structural connectivity between the amygdala and the vmPFC (as indexed by fractional anisotropy) was lower in young adults who retrospectively reported experiences of child maltreatment and child trauma. In that work, these investigators then demonstrated that individuals with lower structural connectivity who subsequently experience stressful life events reported higher levels of internalizing symptomatology at follow-up. Situating our findings in the multiple levels of analyses central to a developmental psychopathology perspective, our results provide further neurobiological evidence related to this theory.

Connected to stress sensitization, neurobiology, and psychopathology, and in contrast to past reports (Hanson, Knodt, et al., 2015, 2018; Ho et al., 2017), we found significant interactions between neurobiology and stress exposure for both internalizing and externalizing symptoms. It is unclear if these previous reports have robustly interrogated both forms of "broad band" psychopathology, or if the associations were only present for internalizing symptoms and diagnoses. Collectively, the reported results underscore that neurobiological changes may be taking place in relation to stress sensitization, and this may explain the increase in symptoms of psychopathology commonly reported in samples exposed to early, and then more recent, life stress.

Relevant for psychopathology, the findings presented here underscore the importance of corticolimbic connectivity in relation to different types of affective and behavioral dysregulation. For internalizing psychopathology, a number of past reports have found lower structural and functional connectivity between the amygdala and the vmPFC in those presenting with major depressive, generalized anxiety, or social anxiety disorders (Etkin & Schatzberg, 2011; Tromp et al., 2012). These patterns fit nicely with a recent report by Connolly et al. (2017) that found depressed adolescents showed reduced amygdala–vmPFC connectivity, compared to healthy comparison subjects. Similarly, depressed adolescents displayed reduced vmPFC functional connectivity during emotional reappraisal of negative images (Perlman et al., 2012). Similar patterns are often noted in externalizing psychopathology. For example, adults with psychopathy show lower functional connectivity between the amygdala and the vmPFC at rest (Motzkin, Newman, Kiehl, & Koenigs, 2011), as well as during socioemotional reactivity tasks (Kiehl et al., 2001; Yoder, Harenski, Kiehl, & Decety, 2015). In interesting recent work by Waller et al. (2018), lower functional connectivity between the amygdala and the vmPFC during the processing of different emotional facial expressions was prospectively related to psychopathic traits in a racially diverse, low-income male sample. In toto, strong evidence suggests neurobiological signatures of effective emotion regulation, with alterations in the amygdala and vmPFC connectivity associating with both internalizing and externalizing forms of psychopathology. Lower functional coupling between these areas may connect to increases in negative affect, and this may eventually give way to symptoms of depression and anxiety, as well as aggression and disruptive behavioral outbursts.

Thinking about future research, we believe continued work in line with and building on the current study could advance knowledge of basic scientific issues, as well as applied questions related to atypical development and different forms of psychopathology. This additional information will likely come through research focused on neurobiology, emotion dysregulation, and related constructs, as well as research that integrates insights from multiple levels of analysis.

In regard to neurobiology and risk for psychopathology, our neuroimaging data connect to many "neuro-maturational" theories focused on risk for poor mental health across development. These models argue that different developmental trajectories for subcortical versus cortical brain structures (e.g., amygdala vs. PFC) may lead to poor mental health (for thoughtful discussion, see Beauchaine, Constantino, & Hayden, 2018). We speculate that aberrant connectivity between the amygdala and the vmPFC may be due to the stressors associated with poverty impacting the development of the PFC. While we do not have direct evidence related to this idea, the PFC has a particularly protracted postnatal developmental timeline (for review, see Marín, 2016) and has been shown to be impacted by poverty and the stressors associated with poverty (e.g., Hanson et al., 2012, 2013). However, we believe a great deal of additional work is needed related to this conjecture (and associated "neuro-maturational" theories). First, through longitudinal investigations, especially those earlier in development, it may be possible to understand if the stressors associated with poverty are uniquely impacting the PFC, compared to the amygdala and other subcortical regions. Second, those interested in neuro-maturation will need to realize that risk for psychopathology likely emerges from the dynamic interactions of multiple brain regions, and we will need to probe the brain in a fashion

commensurate with these synergies. For example, Silvers et al. (2016) found that differences in one form of emotion regulation (cognitive reappraisal) were related to interactions between the amygdala, the vmPFC, and also the ventrolateral PFC. Similarly, Hare, Camerer, and Rangel (2009) and Hare, Hakimi, and Rangel (2014) have found the dorsolateral PFC moderates the vmPFC during different self-regulation and decision-making tasks. Given these suggestive results, researchers would be well served to deploy advanced analytic approaches drawn from network science, such as graph theory, with resting-state fMRI and diffusion-weighted imaging (for review, see Lydon-Staley & Bassett, 2018). It is possible to investigate multiple brain regions at once, understanding if these are diffusely correlated with other regions or if they are regions so tightly coupled that they form clusters that function in unison (Bullmore & Sporns, 2009).

Relevant for emotion dysregulation, linking clear behavioral metrics of emotion regulation to brain development, stress exposure, and symptoms of psychopathology will be critical in the future. In the current work, we used amygdala–vmPFC resting-state coupling as a neurobiological marker of emotion dysregulation based on past research. However, emotion dysregulation may manifest as many different cognitive, social, or behavioral processes, including an inability to shift attention from an overarousing situation, failing to use memories and associations when needing to change emotions, and so on (see Cole et al., 2013, for thoughtful discussion on this issue). Surveying the affective neuroscience literature, there are powerful paradigms to probe both "explicit" and "implicit" forms of emotion regulation (for review, see Gyurak, Gross, & Etkin, 2011). Direct investigations of attentional, memory, coping, and other relevant emotion-related processes in relation to neurobiology, as well as testing connections between neurobiology and physiological reactions to acute stress (e.g., blood pressure), could be of interest to those studying adversity and psychopathology, as well as researchers in intervention and prevention science. To fully understand the developmental connections among stress exposure, emotion dysregulation, neurobiology, and psychopathology, we will need to leverage these types of approaches, as well as think about the bidirectional influences between these constructs. Our work suggests many open questions of inquiry, including: Do changes in neurobiology give rise to emotion dysregulation, or vice versa? Are there instead oscillating cascades happening where modest levels of emotion dysregulation impact the brain, and then neural changes that give rise to greater manifestations of emotion dysregulation? Answering such questions could greatly advance understanding of not only how the early social environment may shape behavior but also how we might promote resilience in those that have suffered stress.

Several methodological strengths of this study bolster our confidence in the robustness of the central results that we reported. First, we employed a longitudinal research design with multiple informants and measures centered on different levels of analysis. We examined parental self-reports of household income and stress exposure, while employing youth self-reports of psychopathology. Many past reports have leveraged data from the same individuals; these types of designs may suffer from specific sources of bias (e.g., Reid, Kavanagh, & Baldwin, 1987), reducing the reliability of results. Second, our sample includes a great deal of socioeconomic and ethnic diversity, with high numbers of European American, African American, and Latin American families. This would suggest that study findings are likely relevant to a broad range of youth populations and communities. Third, resting-state fMRI may be particularly useful in developing samples because it is not biased by a task, instead reflecting a dynamic measure of the history of coactivation between brain regions (Park et al., 2018). Recent work also suggests resting-state fMRI may have stronger reliability than task-based measures (e.g., Elliott et al., 2019).

Of important note, the connectivity between the amygdala and two other brain regions (the paracingulate gyrus and the putamen) emerged in our analyses. These additional brain regions were not hypothesized and did not relate to measures of psychopathology. Furthermore, these regions did not interact with more contemporaneous stress to predict psychopathology. It is currently unclear what these differences may mean and if/how they contribute to different behavioral outcomes. The paracingulate gyrus has previously been connected to response selection (e.g., Sakai et al., 2000). Heightened connections between this area and the amygdala could mean that there are action selection and motor biases in responses involving emotion for lower SES youth. The putamen has been connected to the processing of rewards, aspects of learning. Acute stress has been found to increase amygdala–striatal connectivity (including the putamen). This hyperconnectivity may shift learning processes away from flexible approaches to more habitual responding (for additional discussion, see Vogel, Fernández, Joëls, & Schwabe, 2016). While speculative, this is an interesting route for future focus, as there are recent reports of issues with reward learning in adversity exposed youth (Hanson et al., 2017; Harms, Shannon Bowen, Hanson, & Pollak, 2018).

Despite these important strengths, our work is not without limitations. First, we investigated the effects of poverty and the interactions between contemporaneous stress exposure and neurobiology in separate models. Ideally, we would have probed a three-way interaction, focused on early household income, contemporaneous stress, and neurobiology. This approach has been employed in other work focused on this topic (Hanson, Knodt, et al., 2015); however, our sample size with usable fMRI data is relatively modest. Assuming similar effects as Hanson, Knodt, et al. (2015), we would be underpowered to detect a significant three-way interaction. Future work should aim to test interactions in larger samples with the appropriate power to detect such effects. Second, only one neuroimaging time point was available for our analyses. With the pathways to either maladaptive or positive adaptive functioning being influenced by a complex matrix of factors (Cicchetti & Tucker, 1994), additional research with longitudinal measures of both brain and behavior are needed. Such work could elucidate how neurobiological, environmental, and psychosocial factors continue to interact and potentially amplify (or diminish) risk over development. Related to this, the age range of our participants (15–17 years of age) is relatively young compared to the average onset of many mental health disorders (Burke, Burke, Regier, & Rae, 1990; Kessler et al., 2005). It is possible that many of these participants will go on to develop psychopathology later in development; we therefore could be underestimating different associations between stress exposure, neurobiology, and mental health. Third, we specifically examined household income as our measure of SES. This measure, however, does not richly assess all elements of poverty likely to influence outcomes, including the chronicity of poverty, parental education, subjective social status, and so on. Information about these parameters could further clarify individual differences in neurobiological and psychological development.

These limitations notwithstanding, our results provide strong empirical evidence that lower household income impacts

important neurobiological circuitry involved with emotion regulation. Variations at this neurobiological level may then convey risk for increases in internalizing and externalizing symptomatology, particularly in the context of more recent exposure to stress. These results have important implications for the development and implementation of novel resilience-promoting interventions in those exposed to child poverty and other early life adversities. Additional research is needed to clarify the complex relations between child adversity and long-term physical and mental difficulties; our data are, however, a needed step in the ability to predict, prevent, and treat stress-related psychopathology.

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