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Regular Article

Dimensions of adversity in association with adolescents' depression symptoms: Distinct moderating roles of cognitive and autonomic function

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Abstract

Exposure to adverse events is prevalent among youths and robustly associated with risk for depression, particularly during adolescence. The Dimensional Model of Adversity and Psychopathology (DMAP) distinguishes between adverse events that expose youths to deprivation versus threat, positing unique mechanisms of risk (cognitive functioning deficits for deprivation, and altered fear and emotion learning for threat) that may require different approaches to intervention. We examined whether deprivation and threat were distinctly associated with behavioral measures of cognitive processes and autonomic nervous system function in relation to depression symptom severity in a community sample of early adolescents (n = 117; mean age 12.73 years; 54.7% male). Consistent with DMAP, associations between threat and depression symptoms, and between economic deprivation and depression symptoms, were distinctly moderated by physiological and cognitive functions, respectively, at baseline but not follow-up. Under conditions of greater cognitive inhibition, less exposure to deprivation was associated with lower symptom severity. Under conditions of blunted resting-state autonomic response (electrodermal activity and respiratory sinus arrhythmia), greater exposure to threat was associated with higher symptom severity. Our findings support the view that understanding risk for youth depression requires parsing adversity: examining distinct roles played by deprivation and threat, and the associated cognitive and biological processes.

Keywords: childhood adversity, depression, threat, deprivation, psychophysiology

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Exposure to adversity is prevalent among children and adolescents (herein, youths) in the United States, and it is a well-documented risk factor for depression (Thapar, Collishaw, Pine, & Thapar, 2012). It is estimated that more than 50% of youths will experience an adverse event by the age of 18, and adversity-exposure is associated with earlier onset of youth depression, more persistent course, and lack of response to psychotherapy and pharmacological treatments (McLaughlin et al., 2012; Nanni, Uher, & Danese, 2012; Teicher & Samson, 2013). These risks may be especially consequential in adolescence when the prevalence of depression increases, as adolescent depression strongly predicts recurrence during adulthood (Mash & Wolfe, 2016; Merikangas et al., 2010). As depression remains one the world's most burdensome psychiatric disorders, it is critical to better understand how well-documented risk factors influence depressive symptoms and pathology (World Health Organization, 2018).

A prominent model of the association between adversity and psychopathology is cumulative risk (CR; see Evans, Li, & Whipple, 2013 for an extensive review), which holds that accumulated or prolonged exposures to adversity magnify risk for psychopathology. In support of this model, numerous studies have linked cumulative exposures to poor psychological outcomes, including prospective predictions of persistent psychopathology from childhood through adulthood (Clark, Caldwell, Power, & Stansfeld, 2010), and first-onset adolescent psychiatric disorders in a national sample (McLaughlin et al., 2012). Cumulative adverse events are also associated with use of less adaptive coping skills (Vaughn-Coaxum et al., 2018). Cumulative risk theory is grounded in a stress-response model of risk mechanisms, proposing that accumulated exposures facilitate long-term wear and tear on physiological regulatory systems.

A more recent model proposes that stress-response systems alone are not entirely sufficient in accounting for the effects of adversity on social, cognitive, and biological outcomes. This model extends beyond the accumulation of adverse events to propose a distinction between types of adversity that differ in their underlying mechanisms of risk. The Dimensional Model of Adversity and Psychopathology (DMAP; McLaughlin, Sheridan & Lambert, 2014; Sheridan & McLaughlin, 2014) organizes adverse events along two discrete axes: deprivation (the absence of expected cognitive stimulation or input from the environment) and threat (the experience or risk of harm). According to DMAP, experiences along these two dimensions co-occur at high rates and both heighten risk for psychopathology, but through distinct

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neurobiological domains (see McLaughlin & Sheridan, 2016, for a review of CR compared with DMAP). Deprivation is hypothesized to increase risk for psychopathology via the cognitive adaptations individuals make to less stimulating or complex environments, which affects the working memory, language development, and executive functioning skills that are needed for cognitively complex tasks. Threat is hypothesized to increase risk by disrupting fear and emotion-learning processes, undermining the emotion management skills that are needed to regulate emotional reactivity.

A growing body of evidence supports the distinction between threat and deprivation drawn by DMAP. Studies have shown that exposure to deprivation (e.g., lower family income, lower parental education, food scarcity,) is associated with poorer language development, working memory, and executive functioning (Miller et al., 2018; Sheridan, Peverill, Finn, & McLaughlin, 2017; Wade et al., 2017) after controlling for threat. Even earlier research has shown that the effects of low socioeconomic status on poor working memory are accounted for by one of the primary hypothesized DMAP constructs, lower cognitive stimulation (Noble, McCandliss, & Farah, 2007). Deprivation-related impairments, such as cognitive inhibition (Lambert, King, Monahan, & McLaughlin, 2017), may increase risk for depression via reduced capacity to resist interference from competing demands (Tavitian et al., 2014).

Alternatively, exposure to threat (e.g., interpersonal and community violence, sexual and physical abuse) is associated with blunted sympathetic nervous system activity and poor automatic emotion regulation (Heleniak, King, Monahan, & McLaughlin, 2018; Lambert et al., 2017). Threat-related impairments, for example in autonomic nervous system (ANS) function (Busso, McLaughlin, & Sheridan, 2017), may interfere with emotion-regulation efforts when activation of the body's stress-response systems is incongruent (either blunted or overactive) with the demands of the environment. Resting state parasympathetic function, indexed by respiratory sinus arrythmia (RSA; a sensitive index of heart rate variability accounting for respiration), has been consistently associated with emotion regulation capability across a large number of studies and in a variety of populations (e.g., Appelhans & Luecken, 2006; Beauchaine, 2015; Vasilev, Crowell, Beauchaine, Mead, & Gatzke-Kopp, 2009; Williams et al., 2015). In addition, RSA is linked with ventral medial prefrontal cortex (vmPFC) and amygdala function (Thayer et al., 2012), neural regions that play a role in emotion regulation and in fear learning and extinction (Silvers, Buhle, & Ochsner, 2014). Sympathetic activity, indexed by electrodermal activity (EDA, skin conductance), is less robustly linked at rest with emotional response. However, sympathetic activity is associated with neural regions involved in threat-processing and fear learning, also including the amygdala and vmPFC, and EDA reactivity has been shown to mediate links between threat and fear learning (McLaughlin et al., 2016).

While there is growing support for DMAP, few studies have examined both deprivation-specific and threat-specific mechanisms simultaneously (for exception, see Machlin, Miller, Snyder, McLaughlin, & Sheridan, 2019; Lambert et al., 2017) and there is a need for such studies examining these processes in risk for depression symptoms, in particular. Studies have generally focused on a single process (e.g., cognitive deficits) and tested whether deprivation or threat is uniquely associated with dysfunction in that domain—and in some cases, whether the effects of adversity on that domain predict internalizing or externalizing problems. Thus, one question that has not been fully answered is how deprivation- and threat-specific dysfunction, assessed concurrently, relate to risk for depression symptoms. Mediational pathways from early life threat and deprivation to both internalizing and externalizing symptoms have been supported through distinct emotion-regulatory and cognitive processes (McLaughlin et al., 2016; Miller et al., 2018; Sheridan et al., 2018). However, emotion regulation indices (RSA and EDA) have also been shown to moderate associations between recent or ongoing threat exposures and concurrent internalizing symptoms (Erath, Su, & Tu, 2018; McLaughlin et al., 2015). Thus, a related question investigated here is whether there may be a similar moderating relationship for deprivation-specific processes with depression symptoms. As deprivation and threat often co-occur, it is important to examine DMAP fully with threat and deprivation-specific developmental processes in association with depression symptoms. The present study addresses this need.

This study examined cognitive and autonomic self-regulatory processes associated with exposures to deprivation and threat in relation to depression symptoms in a community sample of adolescents. Working memory capacity and cognitive inhibition are not only associated with deprivation, but meta-analytic research indicates these domains are impaired in youth depression (Wagner et al. 2015), suggesting these are good candidate risk domains for associations between deprivation and youths' symptoms. Blunted ANS function is not only associated with exposure to threat, but lower RSA and EDA have also been observed in depressed individuals (Bylsma, Morris, & Rottenberg, 2008; Hamilton & Alloy, 2016). Findings support ANS function as a good candidate risk domain for associations between threat and symptoms. Given evidence of associations between threat and both resting-ANS and ANS reactivity, it is also pertinent to explore whether resting RSA and EDA moderate associations between threat and depression symptoms, while reactivity might function as a mediator, based on the results from prior studies.

The primary purpose of the present study was to investigate the contributions of adversity, in the form of deprivation and threat, and regulatory function to adolescents' depression symptoms. We sought to answer this question: *How do cognitive and physiological self-regulatory functioning relate to the association between depression symptoms and two dimensions of adversity?* Our first hypothesis was that cognitive function will moderate the association between depression symptoms and deprivation, but not threat. A related hypothesis derived from DMAP is that cognitive function *mediates* associations between deprivation and depression symptoms. Our study design was appropriate for standard mediation tests, permitting us to examine this alternate hypothesis linking deprivation with prospective symptoms. Our second hypothesis was that autonomic function will moderate associations between depression symptoms and threat, but not deprivation. Given robust evidence linking RSA with emotion regulation, we predicted that resting-state RSA will be selectively associated with exposure to threat. Electrodermal activity was also examined for potential moderating effects across both ANS branches, which work in a balance, as less is known about the association between resting EDA and threat. A related hypothesis from DMAP is that autonomic function *mediates* the association between depression symptoms and threat. Therefore, we also tested this alternate hypothesis with RSA and EDA reactivity as potential mediators. Finally, we investigated whether the hypothesized associations were unique to threat and deprivation by examining associations among self-regulatory processes and CR to test specificity and determine whether CR similarly interacts with cognitive and autonomic function.

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To address the primary research question, we sought to account for additional factors that might influence or confound the association between adverse experiences, cognitive and autonomic function, and depression symptom severity. Pubertal development was assessed due to the age range of participants. Research shows that exposure to adversity accelerates pubertal onset (Sumner et al., 2019), and earlier onset is associated with greater risk for psychopathology (Hamlat, Snyder, Young, & Hankin, 2019). We also assessed general cognitive abilities given evidence that the effects of adversity on cognitive function are attenuated by baseline IQ (Danese et al., 2017). Further, physical activity and sleep were accounted for due to associations with autonomic activity and psychological wellbeing (Brand & Kirov, 2011; Nabkasorn et al., 2006).

Adolescent participants and their parents each reported on exposures to adversity (threat, deprivation, cumulative negative life events), and youths completed behavioral tasks assessing working memory capacity and cognitive inhibition. Psychophysiological measurement of RSA and EDA activity were collected at rest and during a distressing task. Depression symptoms were examined continuously (consistent with the National Institute of Mental Health approach to dimensional measurement, 2015) at two time-points. The middle-school age range was selected to target the period when prevalence rates for depression symptoms increase and to reduce variability in developmental stage.

Method

Participants

Early adolescents (6th to 8th grade) along with a parent were recruited for the present study from community, school, and healthcare settings (e.g., community centers, primary care clinics, parent-teacher organizations). Youths were ineligible to participate if their parent reported any of the following during a phone-screen: suicidality in the last 12 months requiring hospitalization; history of psychosis; diagnosis of Autism Spectrum Disorder, Pervasive Developmental Disorder or Intellectual Disability, lack of English language fluency. All youths not meeting exclusion criteria, and within the study grade-level range, were included with parent consent and youth assent. The final sample included 117 youth–parent dyads. Youths were predominantly male (54.7%), aged 10–14 ($M = 12.74$, $SD = 3.64$), with 53% identifying as ethnic-minority or multiracial (Asian or Asian American = 11.1%, Black or African American = 19.7%, Hispanic or Latino(a) = 6%, Multiracial = 16.2%) and 47% as White or Caucasian. Of participating parents, 93.7% were mothers, 67.0% were married, and education level was varied, with 43.6% holding graduate/professional degrees, 30.8% holding college degrees, and 25.6% completing no more than one year of college. Household family income was below the federal poverty line for 15.4% of participants, and 32.5% were in the low-income range (income to needs ratio < 2.5) for the metropolitan area where participants lived (see Table 1).

Measures

Exposure to threat

Community violence exposure. The Screen for Adolescent Violence Exposure (SAVE; Hastings & Kelley, 1997) is a 32-item measure used to assess adolescents' exposure to violence in their communities. Frequency of indirect (e.g., "I have heard about someone getting shot") and direct (e.g., "I have had shots fired at me,"and "Someone my age has threatened to beat me up") violence and victimization were reported on a 5-point Likert-type scale from 1 = *Never* to 5 = *Always*. Subscales have been shown to predict internalizing and externalizing problems in youths and correspond with neighborhood and school crime data in communities where the measure was validated (Hastings & Kelley, 1997). The internal consistency alpha was .96 in the present sample.

Peer victimization. The Reduced Aggression and Victimization Scale (RAVS; Orpinas & Frankowski, 2001) is a 12-item scale designed to assess peer victimization and aggression toward peers. Victimization items were used in the present study, and items asked how many times youths were victimized (e.g., another kid "made up something about you to make other kids not like you anymore?" or "said they were going to hit you?") in the past week. In middle school students, reliability has ranged from $\alpha = 0.84–0.89$, and Orpinas and Frankowski (2001) demonstrated that RAVS scores predict school and community delinquency/discipline, drug use, and lower academic performance. The internal consistency alpha in the current sample was .87.

Economic deprivation

Family Information Form. The Family Information Form was a comprehensive parent-reported demographic and background questionnaire. Parents reported on youths' demographics (e.g., race, age, grade in school, type of school attended, household members) and medical and mental health treatment history, including medications youths were currently prescribed and if they had ever received treatment for emotional or behavioral problems. Parents reported their individual and household demographics, including household income, highest level of education, and marital status. Income to needs ratio was calculated based on federal Census cut-offs and combined with parental education to index economic deprivation.

Cumulative exposures

Adverse life events. A modified version of The Life Event Checklist (LEC; Johnson & McCutcheon, 1980) was used to assess parent-report of negative life events. Life events were rated dichotomously as having *happened* (1) or *not happened* (0) in the last 12 months and summed for a total event score. A subset of items additionally asked whether the event happened in the child's lifetime (e.g., parental incarceration, parental divorce, death of parent, homelessness, removal from the home, child pregnancy). The LEC was modified to remove outdated items and 14 items were added from the Child Life Events Checklist from the Longitudinal Study of Abuse and Neglect (LONGSCAN; Runyan et al., 1998). These items were added to capture domains not assessed originally (e.g., parental incarceration, homelessness, etc.). Tests of validity indicate that negative life change scores are correlated with anxiety, depression, maladjustment, and low levels of locus of control (Brand & Johnson, 1982; Johnson & McCutcheon, 1980).

Developmental and physical functioning

Pubertal development. The Pubertal Development Scale (PDS; Petersen, Crockett, Richardson, & Boxer, 1988) is a five-item scale assessing physical, developmental change (i.e., growth of body hair, height, and changes in skin). Female participants were additionally asked about the development of breasts and menstruation; male participants were asked about deepening of

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Table 1. Sample descriptives

	Total Sample	Sub-group comparisons		
	N (%) / *M*(*SD*)	*N* (%) / *M*(*SD*)	*F* value	*p* value
Income to needs ratio*	4.09 (2.41)			
< 2.5 (low)	38 (32.5%)			
2.5–4.0 (low–middle)	13 (11.1%)			
4.1–5.9 (middle–high)	35 (29.9%)			
≥ 6.0 (high)	31 (26.5%)			
Age	12.74 (3.65)			
Sex				
Male	64 (54.7%)			
Female	53 (45.3%)			
Race				
White	55 (47%)			
Black	23 (19.7%)			
Asian	13 (11.1%)			
Latino	7 (6%)			
Multiethnic	19 (16.2%)			
Parental Education				
Less than college degree	30 (25.6%)			
College degree	36 (30.8%)			
Graduate/professional degree	51 (43.6%)			
CDI-2 Total Depression				
Youth report (T-score) **T1/T2**	49.55 (9.08)/49.35 (9.10)		1.35/1.92	.258/.116
White		48.10 (7.39)/ 46.83 (7.63)		
Black		52.61(12.81)/ 53.20 (9.86)		
Asian		49.00 (9.99)/ 52.33 (8.44)		
Latino		47.00 (6.83)/ 47.50 (10.61)		
Multiethnic		51.32 (7.78)/ 51.08 (11.43)		
Parent report (T-score) **T1/T2**	50.74 (9.40)/51.51 (7.31)		3.91/1.18	.005/.324
White		48.95 (7.44) [a] / 50.96 (6.50)		
Black		56.78 (11.37) [a, b] / 54.29 (8.23)		
Asian		46.54 (6.68) [b] / 48.40 (7.55)		
Latino		50.71 (10.37)/ 51.25 (12.12		
Multiethnic		51.53 (10.51)/ 52.19 (7.07)		
Threat Composite	0.00 (0.88)		6.63	<.001
White		−0.24 (0.46) [a]		
Black		0.76 (1.47) [a, b, c]		
Asian		−0.25 (0.41) [b]		
Latino		−0.10 (0.48)		
Multiethnic		−0.02 (0.74) [c]		
Deprivation Composite	0.00 (0.88)		25.84	<.001
White		0.52 (0.56) [a, b, c]		
Black		−1.014 (0.54) [a, d]		

(*Continued*)

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Table 1. (*Continued.*)

	Total Sample	Sub-group comparisons		
	N (%) / *M*(*SD*)	*N* (%) / *M*(*SD*)	*F* value	*p* value
Asian		0.22 (0.73) [d]		
Latino		−0.72 (0.76) [b]		
Multiethnic		−0.16 (0.87) [c, d]		

Note: *Classification of "low," "low-middle," "middle-high," and "high" are based on income to needs data for the metropolitan area from which the sample was drawn (City of Boston, 2017). Raw income to needs ratios were derived from U.S. Census cut-offs. T1 = Time 1 (baseline assessment). T2 = Time 2 (three-month follow-up assessment). Superscripts represent groups that differ significantly from one another at the $p < .05$ level after Tukey HSD correction for multiple comparisons.

their voice and growth of facial hair. All items were rated dichotomously (1 = *No*, 2 = *Yes*) or on a 4-point scale from 1 = *No*, 2 = *Yes* (*Barely*), 3 = *Yes* (*Definitely*), and 4 = *Development completed*. Internal consistency has been shown to be acceptable (mean $\alpha = .77$ across grades 6 to 8; Petersen et al., 1988) and PDS scores correlate significantly with physician ratings ($r = .61–.67$; Brooks-Gunn et al., 1987). Internal consistency was low in the present sample ($\alpha = .50$ for girls, $\alpha = .70$ for boys), but perhaps this was because of actual variability in the timing of different components of pubertal development. For example, only 11% of boys reported that physical and body hair growth had *not* yet begun, yet 35–40% reported that changes in their voice and facial hair growth had *not* yet started. Similar patterns were reported for girls' development.

Lifestyle: Sleep and physical activity. Youths' sleep and physical activity level were assessed using modified versions of the questionnaires in the National Longitudinal Study of Adolescent Health (Add Health; Harris, King, & Gordon-Larsen, 2005). Questionnaire items asked youths to report on typical sleep/wake times on each day, and a weighted average of hours of sleep per night was computed from weekday and weekend reports. Inactivity levels were assessed via the number of hours per week spent watching TV, playing video games, or watching videos for non-school related purposes.

Depression symptoms

The Children's Depression Inventory-2 (CDI-2; Kovacs, 2011) is a 28-item self-report and 17-item parent-report measure of depression symptoms. Parents endorsed the frequency of their child's experience of each symptom over the past two weeks on a 4-point Likert-type scale from "*Not at all*" to "*Much or most of the time*." Example items include "cries or looks tearful," and "is cranky or irritable." Youths read three statements for each item (e.g., "I feel like crying every day," "I feel like crying many days," and "I feel like crying once in a while") and endorsed the one that best described them over the last two weeks. Subscales include Emotional Problems (e.g., sadness, irritability, self-blame, sleep issues) and Functional Problems (e.g., loneliness, self-criticism, concentration and memory difficulties). Age- and sex-normed T-scores were generated for both reporters, and internal consistency alpha for both reporters was 0.90. Prior discriminant validity evidence indicates that the CDI-2 differentiates between youths with Major Depressive Disorder and matched controls (Kovacs, 2011).

Cognitive functioning

General cognitive ability. The Wechsler Abbreviated Scale for Intelligence-II (WASI-II; Wechsler, 2011) was used to estimate youths' general cognitive ability. The brief WASI-II includes two tasks, vocabulary skills and matrix reasoning, providing a reliable estimate of cognitive ability in individuals over six years of age. Standardized scores are computed in relation to population norms. The Full-Scale IQ score (FSIQ) computed from both subtests demonstrates strong, positive correlations with FSIQ scores on the more comprehensive, longer, Wechsler Intelligence Scale for Children (McCrimmon & Smith, 2013).

Working memory and inhibition. A visual-spatial working memory task (adapted from Peverill, McLaughlin, Finn, & Sheridan et al., 2016) assessed working memory capacity (encoding at high vs. low load), and inhibition (efficiency at filtering out task-irrelevant information during encoding). Participants viewed a fixation cross in the center of the screen (2000 ms) followed by an array of 16 circles (1000 ms) with combinations of red (target) and yellow (distractor) stars inside four different circles. Participants were instructed to remember the location of the red target stars and ignore yellow distractors. In the inhibition trials, cognitive load was static; participants needed to encode the location of two red target stars and ignore the location of two yellow distractor stars. In the capacity trials, cognitive load varied such that participants needed to encode either two (control condition) or four (load manipulation) red stars (Figure 1); on capacity trials, no yellow stars were present. Following encoding, another fixation cross appeared (delay period; 2000 ms) followed by an empty array with a single "?" in one circle (probe; 2000 ms). Participants indicated via keyboard button-press whether a red star target was in the position with the "?". Inhibition was defined as accuracy on trials with both red targets and yellow distractors (low load, distractors present) controlling for accuracy on trials with only two targets. Working memory capacity was defined as accuracy on trials with four red targets (high load, no distractors) controlling for accuracy on trials with only two targets.

Distress tolerance

The computerized Mirror Tracing Task is a widely used frustration/distress tolerance task (Daughters et al., 2005) that was used to elicit psychophysiological reactivity. Participants used a computer mouse to move a small probe along the perimeter of a star-shape on the monitor. However, the mouse was programmed to move in the opposite (mirrored) direction of motorization and when participants made an error (tracing outside the lines) or stalled for more than two seconds, a buzzer sounded, and the task automatically restarted. Participants completed three trials of increasing difficulty, titrated to their skill level, and during a final trial they were instructed to persist as long as possible, but could quit at any time with a keyboard press. To increase social evaluation, participants

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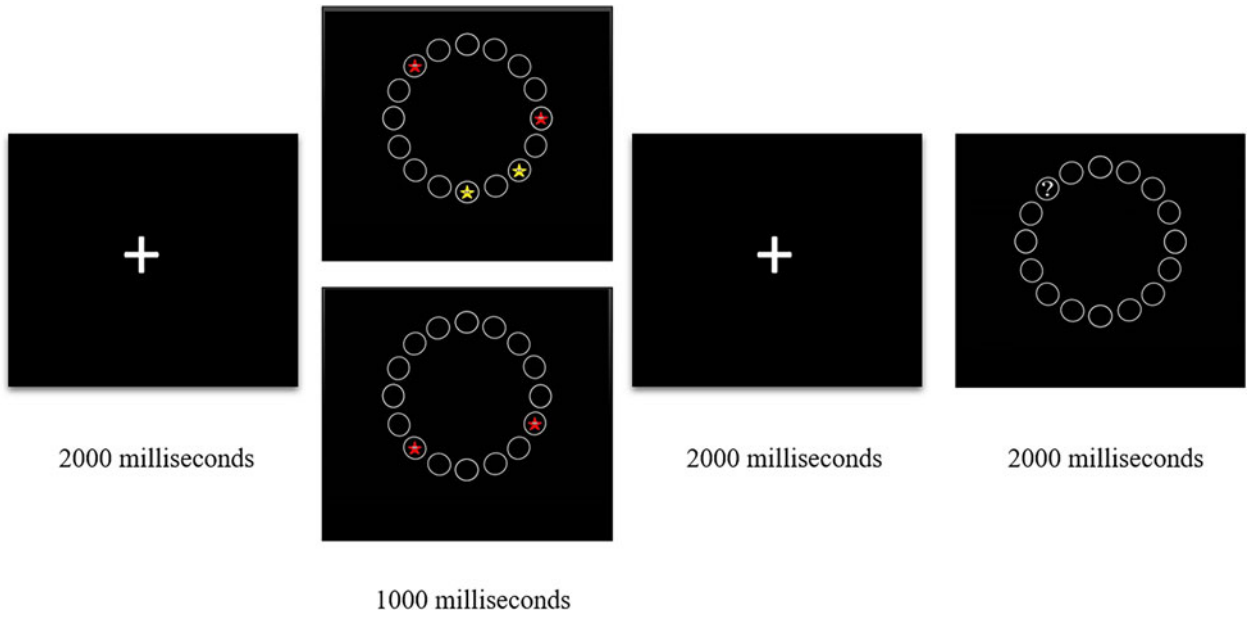

Figure 1. Illustration of the working memory task with a cognitive inhibition trial (top) and a control condition trial (bottom). Participants viewed a fixation cross (first panel) before each trial followed by either an array that includes targets (red stars) to be encoded and distractors (yellow stars) to be inhibited from encoding (top trial), or a trial with only targets of varying load size (2–4) to be encoded (bottom trial). A second fixation cross cued an empty array (last panel) with a probe in one of the circles ("?"). Participants indicated via button-press (yes/no) whether a target star had occupied the location of the probe.

were told that their performance was being recorded and posted to a study website for all participants to see videos of how well others did. No recording occurred, and youths and parents were debriefed about the deception at the end of the study.

Psychophysiological functioning

Electrodermal activity (EDA) and electrocardiogram (ECG) data were recorded continuously using a Biopac MP150 system at a sampling rate of 1000 Hz per second and a 0.5–1 Hz bandpass filter. EDA recordings were obtained using a Bionomadix BN-PPGED amplifier and PPGED-T wireless transducer (Biopac Inc.), connected to two disposable isotonic gel electrodes (Biopac Inc. EL507 disposable EDA electrodes) by a BN-EDA-LEAD2 set. The two electrodes were attached to the thenar and hypothenar eminences of the youth's nondominant hand. ECG and respiration (RSP) recordings were obtained with a Bionomadix BN-RSPEC amplifier and RSPEC-T wireless transducer that was connected to a BN-RESP-XDCR respiration transducer. The wireless respiration transducer was attached by placing a band around the participant's torso at the location of maximum respiratory expansion. Leads were attached to disposable isotonic gel electrodes (Biopac Inc. EL501 ECG electrodes) on the inner left and right wrists, with the ground electrode placed above the outer left ankle. Participants were instructed to minimize physical movement as much as possible during baseline and task periods to avoid movement artifacts or metabolic effects in the EDA and ECG signals. Electrodermal activity and ECG recordings were acquired and processed in AcqKnowledge 4.2.0 (Biopac Inc.). Electrodermal activity and ECG signals were analyzed from a three-minute baseline period and a five-minute stressor task period.

Electrodermal activity recordings were visually inspected by trained research team members, and all signals were subjected to median smoothing and a low-pass filter at 1 Hz. Waveforms were down-sampled to approximately one sample per second prior to analyses. Waveforms were manually inspected for artifacts, which were removed. Any 30-second epoch with more than 10% of unusable data due to signal noise or artifacts was excluded (4.7% of baseline epochs; 3.7% of task epochs). The amplitude and size of skin conductance responses (SCRs) in the baseline period and task period were derived from the waveforms. Electrodermal activity response was operationalized from recommendations by the Society for Psychophysiological Research Ad Hoc Committee on Electrodermal Measures (Boucsein et al., 2012). Resting EDA and reactivity were both defined as the percent change in average amplitude of SCRs in the first compared with the last epoch of that measurement period.

Electrocardiogram and RSP data were processed in the Heart Rate Variability (HRV) module of MindWare 3.0.25 (MindWare Tech Inc.) Respiratory sinus arrythmia (RSA) was calculated from the interbeat interval time series by using the high frequency band (HF-HRV) in 60-second epochs. Data were visually inspected for irregularities and movement artifacts. Epochs with greater than 10% of data that required editing were excluded from analyses (5% of baseline epochs; 5.2% of task epochs), and HF-HRV was calculated by means of spectral analysis and the Fast Fourier Transformation Technique. Baseline and task RSA were calculated as average RSA value across epochs. Respiratory sinus arrythmia reactivity was calculated as the percent change in mean RSA from the baseline to the task period. Participants with at least two-thirds of usable epochs were included in analysis. The number of excluded participants due to too few usable epochs was low (baseline = 5%; task period = 3.4%).

Procedures

All study procedures were approved by the institutional review board. Interested caregivers completed a phone screen, and families that met eligibility criteria were invited to a single laboratory visit lasting approximately two hours. Upon arrival, youths and parents provided written, informed assent and consent

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(respectively). Parents then completed a battery of questionnaires. Youths were administered the WASI-II, a battery of self-report measures, and the computerized behavioral task assessing working memory capacity and inhibition. Next, youths and parents were oriented to the Biopac equipment, and youths completed the three-minute baseline period with EDA and ECG data collected continuously while the participants sat at rest. Following the baseline period, youths completed the computerized, distress-tolerance Mirror Tracing Task. ANS response was measured continuously throughout the task. After all study tasks were completed, participants were debriefed about the social evaluative deception during the task. An online assessment was sent to families three months following the laboratory assessment, with youths and parents asked to complete depression symptom ratings. Up to two reminder emails and two phone calls were made to each family in the event of nonresponse. Youths were compensated $30 USD and parents were compensated $50 USD for participation.

Data analytic plan

All data were examined and tested for normality, missingness, and out of range values. Correlations among all study variables were examined and demographic characteristics that were significantly associated with CDI-2 scores and predictors were covaried (see results). Composites were created by averaging standardized total scores across the set of measures that assessed economic deprivation ($\alpha = 0.72$, inter-item $r = .56$) and threat ($\alpha = 0.70$, inter-item $r = .54$). Life event checklist sum scores were preserved as cumulative total risk. The primary objective was to determine *how cognitive and physiological self-regulatory functioning relate to the association between depressive symptoms and the two different forms of adversity*. To address this objective, linear regression models were constructed with youth- and parent-report total depression symptom severity scores examined as separate dependent variables. Adversity composites were entered simultaneously in the same step of the regression to estimate associations between each composite and CDI-2 scores, controlling for the effect of the other domain. Regulatory function was entered in the last step of each model (working memory capacity, inhibition, RSA, EDA) to estimate additional variance accounted for by self-regulatory processes, above and beyond adversity. To test our hypotheses that cognitive and autonomic function would uniquely moderate associations between deprivation and threat, respectively, with depression symptoms, interaction terms were computed between each regulatory process and each adversity composite. The PROCESS macro (Hayes, 2017) was used in SPSS 24 to examine interactions among regulatory processes and adversity associated with symptom severity. Follow-up analyses were based on prior results from DMAP studies that identify cognitive function as a mediator of the association between adversity and psychological symptoms, as opposed to our hypothesized moderating role. Thus, we examined alternate hypotheses of the association between adversity and symptom severity. The PROCESS macro was used to test indirect associations between each form of adversity and prospective symptom severity at follow-up, through the pathway of self-regulatory functions. Finally, to test specificity of effects to DMAP, primary analyses were replicated with cumulative adversity.

Results

Missingness was less than 5% across all self-report and behavioral variables of interest. Results of Little's test for the randomness of missing data supported the assumption that data were missing completely at random, Little's MCAR Test: χ^2 (363) = 281.22, $p = .999$. An expectation-maximum likelihood (EM) method of imputation was used to estimate missing values given the small percentage of missingness. Violations of distribution normality varied across questionnaire and behavioral variables. Given evidence that transformations (e.g., loglinear) may mask effects (Feng et al., 2014; Fields, 2013), bootstrapping was used to account for violations of assumptions for parametric tests and to control for any dependence across primary statistical tests (Westfall & Young, 1993) with 1000 bootstrap re-samples for each model. Bootstrap bias-corrected *p*-values and confidence intervals are reported.

Descriptives and covariates

The sample reflected the population-expected distribution of depression symptoms (Table 1) on the CDI-2, with 15.4% of youth-reports and 17.1% of parent-reports falling above the 85th percentile cut-off for clinically elevated scores (raw total ≥ 13 for youth-report; ≥ 17 for parent-report). Response rates for complete, three-month follow-up surveys were 68% for youths (with six additional incomplete responses) and 82% for parents. Analyses were performed first with complete cases and then with multiple imputation using the Monte Carlo Markov Chain method. No differences were found, and thus complete-case analyses were retained to avoid increased standard errors from the imputed estimates and to allow for bootstrap corrections.

Significant racial and ethnic differences were identified for both adversity composites, and for parent-reported depression symptom levels (Table 1). In both cases ethnic minority youths' exposure to adversity and symptom levels were generally higher. Gender differences were identified in youth-reported total depression symptom levels, but not parent-reported symptoms, with higher scores for girls than boys, t (115) = 2.15, $p = .034$. Based on correlations among primary variables (Table 2) youth race/ethnicity, gender, and full-scale IQ scores were covaried in all analyses. Youths' physical inactivity and average sleep duration were covaried in all analyses of RSA and EDA response. Consistent with the narrow developmental period selected for the sample, pubertal development was not significantly associated with primary variables and was not included in further analyses. One-way ANOVAs were used to examine whether RSA and EDA responses differed by youth medication use prior to psychophysiological analyses. There were no significant differences (all *p*s > .05) on RSA or EDA levels based on medication status and all usable psychophysiological data were analyzed.

Performance on the working memory task showed expected variation in accuracy across conditions. Accuracy was highest in the control (Mean % of correct responses = .762, $SE = .01$) and inhibition (Mean % of correct responses = .756, $SE = .01$) conditions, and it was significantly poorer in the high load condition (Mean % of correct responses = .698, $SE = .01$). Six participants were excluded from analyses for either scoring lower than 50% accuracy on the control condition (suggesting inability to complete the task), consecutive nonresponses on trials that indicate significant distraction, or responding with the same button press for every trial.

Primary outcomes

The broad question addressed in our study was how cognitive and physiological self-regulatory functioning relate to the association

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Table 2. Pearson correlations among primary independent variables, dependent variables, and covariates

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
1. Threat composite	—	−.410***	−.057	−.093	−.125	.451***	.443***	.079	.071	.105	.059	.072	.100	−.324***	.522***	.073
2. Deprivation composite		—	.204*	.139*	.110	−.237**	−.381***	−.297**	−.229*	−.001	.012	−.052	−.173	.302**	−.324***	−.318**
3. Working memory control			—	.683***	.638***	.028	−.088	−.073	−.131	.097	.104	−.047	−.089	.020	−.113	−.030
4. Cognitive inhibition				—	.554***	−.098	−.256**	−.151	−.168	.007	−.063	−.033	−.072	.110	−.099	−.052
5. Working memory load					—	−.084	−.217*	−.147	−.313**	.093	.046	.045	−.179	−.018	−.131	−.068
6. CDI youth total T-score						—	.470***	.559***	.377***	.083	−.157	.006	.071	−.233*	.385***	.139
7. CDI parent total T-score							—	.172	.605***	.049	−.134	−.009	.224*	−.284**	.177	.093
8. T2 CDI youth total T-score								—	.434***	−.087	−.035	−.133	.126	−.262*	−.044	.302**
9. T2 CDI parent total T-score									—	.052	−.082	−.166	.305**	−.191	−.118	.122
10. Resting EDA amplitude										—	.064	−.103	.101	.037	.064	.074
11. EDA reactivity											—	−.046	.011	−.043	.040	−.020
12. Resting RSA												—	−.508***	−.124	.109	−.118
13. RSA reactivity													—	−.195*	.004	.199*
14. Average sleep														—	−.281**	−.260**
15. Hours/week spent Watching videos															—	.166
16. Pubertal development																—

Note: Threat composite = higher scores indicate greater exposure to community violence and peer victimization; Deprivation composite = higher scores indicate greater family resources (less deprivation) in parental education and income to needs ratio; Working Memory Control = task condition with low load and no distractors for encoding, Cognitive Inhibition = task condition with targets and distractors that must be filtered out before encoding; Working Memory Load = task condition with highest load of targets for encoding; T2 (Time 2) CDI = total T-scores at three-month follow-up; Resting EDA amplitude = amplitude of skin conductance responses during baseline period; EDA reactivity = percent change in average skin conductance response amplitude between baseline and stressor task periods; Resting RSA = average RSA value during baseline period; RSA reactivity = percent change in average RSA between baseline period and stressor task periods; Average Sleep = seven day average of hours of sleep per night, weighted for weekdays and weekends; Pubertal development = higher scores indicate farther progress in pubertal development, across gender neutral and gender specific developmental domains. * $p < .05$ ** $p < .01$ *** $p < .001$.

Table 3. Associations among exposure to adversity and cognitive inhibition with depression symptom severity

	Parent report total symptom severity					Youth self-report total symptom severity				
	Beta	*SE*	*p*-value	LL	UL	Beta	*SE*	*p*-value	LL	UL
Step 1	R^2 = .15*					R^2 = .07				
Full-scale IQ score	−.053	.066	.597	−.156	.078	.042	.069	.704	−.095	.139
Black	.038	3.232	.781	−5.597	6.902	−.039	2.998	.776	−6.747	5.257
Asian	−.061	2.583	.503	−6.499	3.688	.032	2.561	.728	−3.763	6.549
Latino	−.084	4.723	.492	−12.186	7.130	−.070	3.579	.430	−8.204	5.002
Multiracial	−.023	2.529	.815	−4.821	4.888	.034	2.079	.687	−3.266	5.186
Male gender	−.111	1.682	.245	−5.417	1.397	−.194	1.599	.042	−6.953	−.131
Step 2	ΔR^2 = .17*					ΔR^2 = .21*				
WM control	.292	10.526	.045	2.064	40.636	.270	8.605	.031	3.049	34.824
Threat	.307	1.673	.054	−.626	5.523	.403	1.523	.003	1.160	6.577
Deprivation	−.245	1.571	.090	−5.462	.638	−.078	1.246	.534	−3.227	1.911
Cognitive inhibition	−.241	10.235	.090	−41.580	4.632	−.116	9.799	.397	−28.079	12.714
WM capacity	−.103	6.595	.309	−18.531	3.261	−.090	7.621	.467	−21.703	8.283
Step 3	ΔR^2 = .00					ΔR^2 = .06*				
Cognitive Inhibition X Deprivation	.044	9.188	.682	−12.625	23.433	−.248	9.312	.021	−40.222	−.558

Note: Threat = community violence and peer victimization. Deprivation = Income to needs ratio and parental education. WM Control = % correct on low load trials with no distractors in working memory task. Cognitive inhibition = % correct on distractor trials in task. WM Capacity = % correct on high load trials with no distractors in task. Bootstrap corrected standard errors, *p*-values, and confidence intervals are presented. *$p < .05$.

between depressive symptoms and the two different forms of adversity. To address that question, we tested four hypotheses, two that we proposed based on the DMAP framework that were related to moderation effects and two alternate hypotheses derived from DMAP that were related to mediation.

Associations with cognitive self-regulation

The primary hypothesis about cognitive processes was that cognitive function would moderate the association between depression symptoms and deprivation, but not threat. To test this hypothesis, linear regression models were used to examine main effects and interactions between adverse events and cognitive function in association with depression symptoms. Separate models were run with parent- and youth-reported total symptom severity as outcome variables. Table 3 provides main effects results for parent- and youth-reported depression symptoms. Each model examined concurrent contributions of threat, economic deprivation, and cognitive function to CDI-2 total depression T-scores. For parent-reported symptoms, no associations between adversity (threat or economic deprivation) and symptom severity survived bootstrap correction. An initial main effect of lower cognitive inhibition associated with higher symptom severity also did not survive bootstrap correction. In contrast, for youth-reported symptoms, threat was positively associated with higher symptom severity ($\beta = 4.15$, $p = .003$), but not economic deprivation ($\beta = -.82$, $p = .534$), and again there was no main effect of inhibition.

Consistent with the hypothesis, there was a significant interaction between economic deprivation and inhibition in association with youth-reported depression symptoms, $\beta = 21.15$, $p = .021$ ($\Delta R^2 = .06$). Lower economic deprivation (or greater economic resources) was associated with lower symptom severity, but only in the context of higher inhibition scores ($t = -2.53$, $p = .013$). When inhibition was poorer, there was no negative association between family resources and symptom severity ($t = 0.89$, $p = .375$). In other words, the apparent buffering effect of economic resources against higher depression symptoms emerged only when youths had better inhibitory capability (see Figure 2). This moderating effect was specific to economic deprivation, and not threat ($p > .05$), consistent with DMAP theory. Working memory capacity was not significantly associated with parent- or youth-reported symptoms and did not interact with either form of exposure to adversity (all *p*s > .05).

Associations with physiological self-regulation

The primary hypothesis about physiological processes was that autonomic function (resting-state RSA, with resting EDA examined exploratorily) would moderate the association between depression symptoms and threat, but not deprivation. To test this hypothesis, linear regression models were used to examine main effects and interactions between adverse events and autonomic response. Results revealed that resting-state RSA was not directly associated with baseline symptom levels as reported by parents ($\beta = .10$, $p = .921$) or youths ($\beta = -.01$, $p = .590$) when threat and economic deprivation were accounted for. Baseline EDA was also unassociated with parent-reported symptom levels ($\beta = .14$, $p = .178$) and marginally associated with youth-reported symptom levels ($\beta = .19$, $p = .051$). However, consistent with the hypothesis, both resting RSA and EDA moderated associations between threat exposure and symptom severity. Higher threat exposure was associated with higher total symptom severity, but only in the context of lower resting RSA. There was no association between threat and symptom severity at higher levels of resting RSA. The moderating effect was significant for parent-reported

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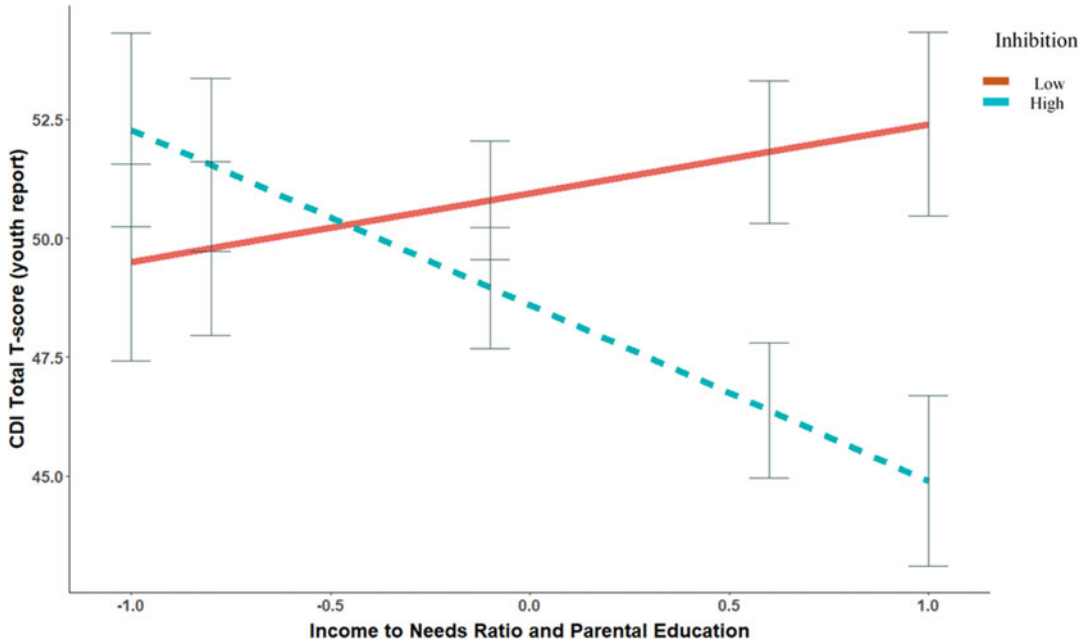

Figure 2. Higher scores on the *x*-axis (to the right) indicate higher economic resources (lower deprivation). The slope for associations between symptom levels (*y*-axis) and deprivation (*x*-axis) is illustrated at +/– 1 *SD* from the mean inhibition score (% of correct responses on trials in the condition with distractor stimuli) F (1, 99) = 6.01, p = .016. At higher levels of inhibition, lower deprivation is associated with lower youth-report depression symptom levels. The slope for low inhibition (1 *SD* below the mean) illustrates a nonsignificant association between deprivation and symptom severity; greater economic resources do not have a protective effect on symptom levels when inhibition is poorer.

symptom severity, $\beta = -1.70$, $p = .003$ ($\Delta R^2 = .073$), as seen in Figure 3 (high RSA: $t = 0.68$, $p = .501$; low RSA: $t = 5.15$, $p < .001$). In other words, greater threat exposure was associated with higher symptom severity, but only when youths' baseline cardiac flexibility was blunted. The overall interaction effect by youth report was initially marginal, and it was nonsignificant after bootstrap correction, $\beta = -0.16$, $p = .115$, but the pattern was the same (high RSA: $t = 1.15$, $p = .252$; low RSA: $t = 3.28$, $p = .001$).

A similar pattern was found for sympathetic functioning; baseline EDA moderated the association between threat and depression symptoms. Greater threat exposure was significantly associated with higher symptom levels, but only under conditions of lower sympathetic sensitivity (decreasing SCR amplitude over time). There was no significant association between threat exposure and symptom severity under conditions of greater sympathetic flexibility (increasing SCR amplitude) at rest. Results were significant and in the same direction for both parent-report symptoms, $\beta = .29$, $p = .007$; $\Delta R^2 = .06$ (high EDA: $t = -.07$, $p = .946$; low EDA: $t = 4.74$, $p < .001$), and youth-report, $\beta = .34$, $p = .002$; $\Delta R^2 = .07$ (high EDA: $t = -.71$, $p = .480$; low EDA: $t = 4.65$, $p < .001$), as seen in Figure 3. Importantly, all moderating effects were specific to threat and not deprivation, consistent with DMAP theory and our hypothesis.

Secondary outcomes based on follow-up assessments

Prospective symptom levels and indirect effects

An alternate, DMAP consistent hypothesis about cognitive processes was that cognitive function would *mediate* the association between depression symptoms and deprivation, but not threat. To test this alternate hypothesis, we regressed symptom severity at three-month follow-up onto inhibition (controlling for baseline symptom severity) and regressed inhibition onto each form of adversity at baseline. Neither domain of adversity was significantly associated with inhibition (threat: $\beta = -.01$, $p = .932$; economic deprivation: $\beta = -.02$, $p = .879$), and inhibition was not significantly associated with symptom severity at the three-month follow-up ($\beta = -.02$, $p = .900$). Based on these results, further tests of indirect effects were not supported.

The alternate hypothesis about physiological processes was that autonomic function would *mediate* the association between depression symptoms and threat, but not deprivation. To test this alternate hypothesis, we regressed symptom severity at three-month follow-up onto each form of physiological reactivity, controlling for baseline depression symptom severity and resting-state EDA and RSA response. Then, we regressed each form of physiological reactivity onto each form of adversity at baseline. Results demonstrated that adversity was not statistically predictive of EDA reactivity (threat: $\beta = -.04$, $p = .733$; deprivation: $\beta = .04$, $p = .864$) or RSA reactivity (threat: $\beta = .05$, $p = .658$; deprivation: $\beta = -.12$, $p = .431$). Neither form of reactivity was associated with symptom severity at follow-up (RSA: $\beta = .09$, $p = .369$; EDA: $\beta = .11$, $p = .139$). Based on these results, further tests of indirect effects were not supported.

In addition to testing alternate hypotheses regarding potential mediating effects of cognitive and physiological function, we examined whether the processes tested in our primary analyses predicted symptom severity at follow-up. After accounting for baseline symptoms, there were no main effects of either form of adversity, cognitive function, or resting-state EDA on symptom severity at follow-up (all ps > .05). However, lower resting RSA at baseline predicted higher youth-reported symptom severity three months later ($\beta = -.22$, $p = .022$), with baseline symptoms controlled.

Cumulative risk model vs. DMAP

Finally, primary analyses were repeated with total adverse events (in the last 12 months and lifetime) as a comparison of the DMAP and CR models. Higher cumulative scores were directly

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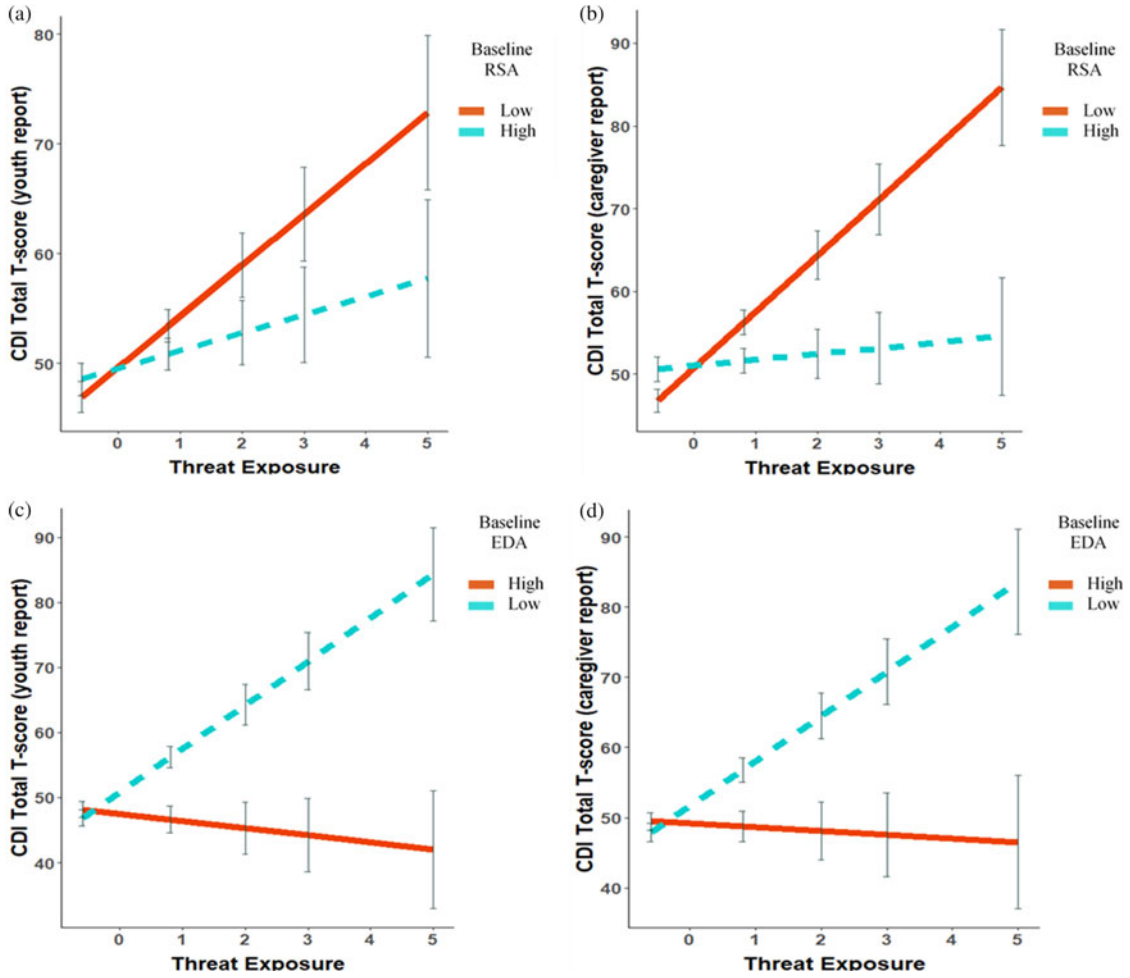

Figure 3. All panels illustrate interaction effects plotted at +/– 1 *SD* from the mean level or RSA or EDA, respectively. Left panels illustrate youth-report total depression symptoms on the *y*-axis, right panels illustrate caregiver-report total depression symptoms on the *y*-axis. Top panels represent resting RSA. When RSA is lower, higher threat exposure is associated with higher depression symptom levels reported by caregivers (p < .001, Panel B) and youths (p =.001, Panel A). The slope for RSA at 1 *SD* above the mean (higher RSA) is nonsignificant for both reporters. *The omnibus interaction is nonsignificant in Panel A, which is displayed for comparison of pattern across reporters. Overall interaction effect is significant (p < .05) for Panels B–D. Bottom panels represent resting EDA (% change in SCR amplitude when subtracting the last epoch from the first). Positive values represent greater amplitude at the start of the rest period compared to the end (habituation). Greater habituation at rest is associated with higher symptom levels as threat exposure increases (youth-report, p < .001, Panel C; caregiver-report, p < .001, Panel D). The slope for EDA at 1 *SD* below the mean is nonsignificant for both reporters.

associated with higher youth-reported symptoms, however, neither cumulative score interacted with regulatory processes (see supplemental Table 1). Moderating effects were specific to dimensional exposures to adversity.

Discussion

Exposure to adversity during development is a potent risk factor for depressive pathology; however, we do not fully understand the interplay between adverse experiences and the cognitive and biological domains that increase this risk. One effort to improve our understanding is the DMAP framework (Sheridan & McLaughlin, 2014). This model holds that disruptions across multiple biological and behavioral self-regulatory processes may be distinctly influenced by different types of adverse events—threat and deprivation—and that examining these unique influences may clarify the connection between risk factors and depression symptoms. Consistent with DMAP, we found that associations between threat and depression symptoms, and between economic deprivation and depression symptoms, were distinctly moderated by physiological and cognitive functions, respectively. We also found that these effects were specific to dimensions of adversity. Cumulative events (the CR model; Evans et al., 2013) did not interact with either cognitive or physiological processes. Our findings support the growing body of literature suggesting that risk for youth problems is influenced by the effect of adversity on multiple, distinct cognitive and biological domains of functioning.

Our research focused on the question of how cognitive and physiological self-regulatory functioning relate to the association between depressive symptoms and two different domains of adversity. Prior studies have not generally examined both deprivation-specific and threat-specific risk processes concurrently within the same sample or had a specific focus on depression symptoms. Our primary hypothesis that cognitive function would moderate the association between economic deprivation and depression symptoms was supported. Higher levels of family resources (lower deprivation) were associated with decreased, concurrent symptom severity, but only under conditions of higher inhibitory capability. When inhibitory performance was poorer, there was no protective effect of greater economic resources.

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This effect was specific to economic deprivation, with no significant interactions between threat and inhibition.

Interestingly, the interaction took a somewhat different form than we expected, since DMAP theory posits that poorer cognitive function augments risk for symptoms. However, the precise form of the buffering effect we detected is consistent with prior research and theory. For example, higher SES has been shown to correlate negatively with depression (Quon & McGrath, 2014) and cognitive function has been theorized as a buffer between SES and poorer emotional and behavioral outcomes (Bradley & Corwyn, 2002; Farah, 2018). Our findings suggest that the protective effect of family resources may be attenuated by self-regulatory capacity. Without effective cognitive self-regulatory propensity, more family resources may not be as advantageous in buffering against depression symptoms.

We also hypothesized that autonomic function would distinctly moderate the association between threat, but not deprivation, and depression symptom severity. Our results supported this hypothesis, with some variation across reporter, and they are consistent with prior evidence that blunted resting-state RSA augments the association between greater exposure to threat and higher concurrent internalizing symptoms (McLaughlin et al., 2015). Interestingly, blunted resting-state EDA similarly moderated the relation between threat and depression symptoms. The consistency of these moderating associations across both youth- and parent-reported depression symptoms suggests a particularly robust effect, especially given the typical lack of agreement between youth- and parent-reported symptoms (De Los Reyes & Kazdin, 2005). This pattern extends prior research findings and indicates that lower resting-state autonomic activity across both ANS branches may amplify the association between threat exposure and symptom severity. We also found that blunted resting-RSA predicted higher levels of depression symptoms at the three-month follow-up, which is consistent with research identifying cardiac vagal tone (RSA) as a marker of risk for depression (Hamilton & Alloy, 2016).

The moderating effects identified for both economic deprivation and threat exposures were specific to concurrent depressive symptoms, but not depression symptom severity at follow-up (controlling for baseline symptoms). Several factors may have contributed to this pattern of results, including the nature of our sample and timing of assessments. We selected a short-term follow-up period, and overall depression symptom scores did not change significantly. After controlling for the contribution of Time 1 severity on Time 2 scores, the interplay between adverse experiences and cognitive and autonomic function may not have accounted for enough additional variance to significantly predict follow-up symptoms given the stability of symptom severity levels over three months in our sample.

Further, we tested two alternate hypotheses derived from DMAP examining cognitive and autonomic responses as mediators between adversity-exposure (economic deprivation and threat, respectively) and prospective depression symptoms at follow-up. We did not find support for mediation. These results were unsurprising, again, given the lack of change in symptom severity at follow-up. Further, based on DMAP theory, mediating effects may account for the influence of early life adversity on alterations in cognitive and autonomic function (McLaughlin et al., 2014; Sheridan & McLaughlin, 2014). This has been observed in both high adversity and community samples (e.g., Miller et al., 2018; Sheridan et al., 2018). However, here we focused on the measurement of threat and economic deprivation primarily in the last year, positioning us to answer questions about the application of DMAP to more recent adverse experiences.

Indeed, similar to prior studies examining threat- or deprivation-specific adverse events in adolescence (Lambert et al., 2017; McLaughlin et al., 2015), our findings suggest that the DMAP distinction between dimensions of adverse experiences is meaningful even for more recent exposures, though patterns of associations between adversity in later development and biological dysfunction may differ from patterns between early adversity and biological dysfunction. In sum, our findings are consistent with the interpretation that risk processes specific to threat and economic deprivation still influence the association between adversity and depression symptoms during adolescence, but may magnify instead of mediating these associations in the context of recent or ongoing adverse experiences. To explore this hypothesis, the distinction between early and later-childhood adversity in relation to DMAP-specific risk pathways warrants further study.

The study has several limitations. First, while a community sample was appropriate for a dimensional assessment of depression symptoms, our sample did not over-represent the extreme end of the distribution of adversity or symptom severity. Though many studies of threat and deprivation use community samples with similar variance in adverse experiences (e.g., Lambert et al., 2017), the nature of associations between adversity and biological functions may differ in relation to symptoms of psychopathology on the more severe end of both adversity exposure and symptom severity. Oversampling for adversity and clinically elevated depression symptoms in future studies would help shed light on the range of adverse experience and symptom severity where disruptions to cognitive and autonomic function have the most robust effects. Second, the mainly cross-sectional nature of the study also limits conclusions regarding temporal associations between adversity, regulatory processes, and depression symptoms; a larger sample and longer follow-up period would have provided a more sensitive examination. Third, the study was not designed to assess lifetime exposure to threat and deprivation. While there is some evidence that recency effects for exposure to adversity are most strongly associated with psychopathology (Dunn et al., 2018), capturing early life deprivation and threat exposures would have allowed for more direct comparisons of the CR and dimensional models. Early life measures would also have allowed for temporal tests of self-regulatory processes as mechanisms of risk, facilitating a more robust test of moderating versus mediating effects on depression symptoms. Further, we assessed CR with reports of adverse life events via a Life Event Checklist designed to ensure exact standardization of questions and wording across participants. Recent evidence from adult samples suggests that structured interviews may generate reports of more life events, and they may also be psychometrically and qualitatively superior (Harkness & Monroe, 2016). Thus, it is possible that our findings regarding life events (e.g., their timing and/or chronicity) would have been different had we used an interview-based measure. This suggests a useful direction for future research. Finally, our measure of deprivation was solely economic. A more comprehensive measure (including cognitive stimulation in the home environment and amount of parental attention; Sheridan & McLaughlin, 2014) could provide a more multifaceted picture of deprivation-related associations with regulatory processes and depression symptoms.

Our findings support the meaningfulness of a dimensional conceptualization of childhood adversity, even for adverse experiences occurring during adolescence, and the potential

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contribution of that perspective to our understanding of risk for depression. McLaughlin and Sheridan (2016) highlight learning mechanisms (e.g., associative learning, fear conditioning, reward learning) as intervenable targets to ameliorate poorer cognitive and affective regulatory functioning. Determining whether modification of these learning mechanisms can attenuate the association between adverse experiences and distinct types of regulatory function (i.e., cognitive and autonomic) is relevant to research efforts focused on identifying mechanisms of risk that could be targeted in personalized interventions. Viewing our findings in the context of the broader DMAP literature underscores the need for future studies to elucidate the different ways exposure to adversity influences depressive pathology, particularly in terms of more recent compared with early adverse experiences. Given the large proportion of youths who experience adverse events and the significant proportion who are at risk for depression during adolescence it is important to characterize the biological and cognitive domains that may distinctly magnify or reduce risk during this vulnerable developmental stage.

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