Cumulative risk exposure moderates the association between parasympathetic reactivity and inhibitory control in preschool-age children

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Abstract
A child’s cumulative risk for early exposure to stress has been linked to alterations of self-regulation outcomes, including neurobiological correlates of inhibitory control (IC). We examined whether children’s ability to engage the parasympathetic nervous system impacts how risk affects IC. Children ages 3–5 years completed two laboratory measures of IC while respiratory sinus arrhythmia (RSA) was measured, indexing parasympathetic activity. Children with greater risk demonstrated lower IC; risk also moderated associations between RSA reactivity and IC. For children with less risk, greater RSA withdrawal during IC tasks was associated with better IC. In contrast, greater risk was associated with poor IC, regardless of RSA withdrawal. Effects of risk were more pronounced for cumulative than individual measures. Results suggest that cumulative risk exposure disrupts connectivity between physiological and behavioral components of self-regulation in early childhood. Parasympathetic withdrawal to cognitive tasks may be less relevant for performance in developmental samples experiencing greater life stress.

Keywords
cumulative risk, early adversity, individual differences, inhibitory control, respiratory sinus arrhythmia

1 INTRODUCTION
Children exposed to a range of early life stressors, such as poverty, maltreatment, and chaotic home environments have been documented to exhibit deficits in inhibitory control (IC), the ability to control impulsive or prepotent responses (Hostinar, Stellern, Schaefer, Carlson, & Gunnar, 2012; McDermott, Westerlund, Zeanah, Nelson, & Fox, 2012; Mueller et al., 2012; Pears, Fisher, Bruce, Kim, & Yoerger, 2010; Skowron, Cipriano-Essel, Gatzke-Kopp, Teti, & Ammerman, 2014). Thus, it is perhaps unsurprising that children with a history of early life stress who show high IC performance are often resilient from other negative outcomes. For example, IC has been found to fully mediate links between maltreatment status and socio-emotional competence (Pears, Fisher, Bruce, Kim & Yoerger, 2010). Within samples of economically-disadvantaged children, IC has been found to predict higher social skills and lower internalizing problem behaviors (Rhoades, Greenberg, & Domitrovich, 2009) in addition to mediating longitudinal links between family adversity (income, instability, chaos) and poor school readiness (Brown, Ackerman, & Moore, 2013).

Characterizing biological processes underlying effective IC is particularly important in at-risk children exposed to multiple poverty-related stressors and in those who disproportionately experience negative health and well-being outcomes linked to IC deficits, such as academic failure, psychopathology, and risk-taking behavior (Evans & Kim, 2012). Such poverty-related stressors include known socio-demographic, environmental, and psychosocial risk factors that
aggregate to form an index of cumulative risk exposure and place children at risk for deficits in self-regulation (Evans & Kim, 2013). Measures of the autonomic nervous system may be critical for addressing this issue, given that early exposure to stressful experiences has profound effects on autonomic function, beginning as early as the prenatal environment (Propper & Holochwost, 2013). More specifically, activity of the parasympathetic branch of the autonomic nervous system has gained attention as a mechanism by which children flexibly engage and regulate their physiological state in the service of behavioral regulation (Graziano & Derefinko, 2013).

A growing body of research describes how regulation of peripheral physiology occurs via top-down influences on the parasympathetic nervous system (PNS) as a fast-acting mechanism to control heart rate (Berntson, Cacioppo, & Quigley, 1993). Flexible engagement and withdrawal of the PNS is theorized to help individuals rapidly alternate between states of engagement and disengagement in the context of environmental demands (Porges, 2001). When the PNS withdraws, heart rate and cardiac output increase, and when the PNS is activated (the vagal brake is applied), heart rate and cardiac output are reduced. Respiratory sinus arrhythmia (RSA) reflects the measurement of parasympathetic influence on heart rate and serves as an index of physiological regulation (Berntson et al., 1993).

Measures of RSA taken in “resting” conditions, often while individuals sit passively and watch an affectively neutral film clip, have been proposed to reflect trait-like qualities of self-regulation (Beauchaine & Thayer, 2015). Higher resting RSA is commonly associated with better affective and behavioral regulation (e.g., Calkins, Graziano, & Keane, 2007; Gorka et al., 2013; Graziano & Derefinko, 2013; Hastings et al., 2008); however, recent meta-analyses suggest this is a small effect with a high degree of heterogeneity and thus should be interpreted with caution (Holzman & Bridgett, 2017; Zahn et al., 2016). Notably, adults who are more resilient to acute stressors show higher resting RSA (Lü, Wang, & You, 2016), raising the possibility that high resting RSA may be protective for individuals exposed to a greater number of stressful experiences earlier in life. In other words, reports of inconsistent associations between resting RSA and cognitive function may be due to this relationship being moderated by differences in individuals’ life experiences.

RSA reactivity reflects the extent to which individuals engage or disengage PNS activity relative to resting levels to meet the demands of a given context. Research that has examined how children’s RSA reactivity to cognitively challenging tasks supports behavioral regulation has typically found that RSA withdrawal supports higher cognitive function in children (for a review, see Graziano & Derefinko, 2013). In their meta-analysis, Graziano and Derefinko (2013) found that higher levels of RSA withdrawal were associated with better cognitive and academic performance. These results suggest that, on average, it is helpful for children to withdraw vagal control of heart rate and increase physiological arousal in order to facilitate attentional control and successfully engage in assessments of cognitive function. A recent meta-analysis found that RSA reactivity and resting RSA have similar effect sizes with regard to their association with cognitive function but with far fewer studies in the literature examining RSA reactivity during cognitive function (Holzman & Bridgett, 2017); therefore, a key aim of the present study was to characterize child cognitive function as a function of both resting RSA and RSA reactivity.

In contrast to the group average effects reported in the meta-analytic study, a growing number of findings suggests that the observed pattern of RSA withdrawal during cognitive challenge may not facilitate performance for all children. For example, Conradt et al. (2016) found that greater RSA withdrawal at age 1 month predicted greater behavioral dysregulation at age 3 years in children exposed to higher degrees of caregiver stress; however, a significant relationship between RSA reactivity and later behavioral dysregulation was not observed in children of caregivers reporting less stress related to caregiving. Amongst the same sample, Conradt et al. (2014) reported a similar pattern of increasing RSA withdrawal, assessed in yearly longitudinal measurements at ages 3–6 years, associated with more disinhibited behavior problems later in life (ages 8–14) for children exposed to a greater number of cumulative life stressors. Conversely, for children with less exposure to early risk factors, increases in RSA withdrawal from ages 3 to 6 were associated with fewer disinhibited behavior problems later in life (Conradt et al., 2014). These findings may explain results from a separate study of maltreatment-exposed children which did not examine RSA reactivity but found that lower levels of RSA during challenging parent–child dyadic interaction tasks were associated with worse performance on two separate measures of IC (Skowron et al., 2014). The reverse pattern was observed in children not exposed to child maltreatment: Higher RSA levels during challenging tasks with a parent were associated with better IC.

One theoretical explanation for these findings is that children who are more physiologically reactive may be more sensitive to environmental effects, such that greater parasympathetic reactivity may exacerbate the impact of exposure to a high degree of daily stressors on self-regulation abilities (Conradt et al., 2016). An alternative, and perhaps complementary, explanation is that children with higher cumulative risk exposure have less-coordinated activity amongst biological systems underlying self-regulation (El-Sheikh et al., 2009). Given that control of parasympathetic function is largely related to neural networks involving prefrontal cortical activity (Thayer, Åhs, Fredrikson, Sollers, & Wager, 2012), which is strongly implicated in behavioral control, it may be that cumulative stress exposure disrupts the ability to effectively leverage parasympathetic activity for concurrent cognitive demands. For example, children with relatively lower cumulative risk exposure may have fewer “bottom-up” impulsive arousal demands to control; thus, releasing vagal control over heart rate may facilitate speeded responding without increased distraction from other bottom-up impulses linked to chronically heightened arousal.

We suggest that it is critical to establish the extent to which PNS function, characterized by RSA withdrawal during cognitively challenging tasks, may or may not reflect resiliency for all children (Obradović, 2012). Research in this area is particularly important because interventions are increasingly looking to biological markers as indices of intervention efficacy, and increased RSA withdrawal may not reflect an adaptive profile for children with the highest levels of risk exposure. In particular, it is unknown if cumulative risk moderates links between RSA reactivity to cognitive challenge and regulated behaviors.
such as IC. What evidence does exist suggests that studies of samples characterized by sociodemographic risk report smaller effect sizes between RSA and cognitive function than studies not reporting sociodemographic risk (Holzman & Bridgett, 2017). Similarly, we have previously demonstrated that RSA reactivity shows stronger associations with cognitive function in adults reporting lower levels of cumulative life stress and whom have lower resting sympathetic nervous system activity, relative to adults reporting higher life stress with higher sympathetic activity at rest (Giuliano, Gatzke-Kopp, Roos, & Skowron, 2017). Therefore, we predict that relationships between RSA and IC observed here will be less pronounced in children with higher cumulative risk exposure.

1.1 Present study

The first aim of the present study was to examine links between parasympathetic function and IC performance in a large sample of preschool-age children from a wide range of socio-economic backgrounds. We were particularly interested in how RSA reactivity during cognitively challenging IC tasks supports effective IC performance in those same tasks. This is a notable design strength compared with those of previous studies that have measured RSA and cognitive function at separate time points, given that our design allowed for assessment of how physiological regulation supports concurrent behavioral regulation. In characterizing the link between parasympathetic function and IC behavior, we were interested in both resting RSA (indexing baseline function) and RSA reactivity (indexing regulatory flexibility). Consistent with previous research, we hypothesized that higher baseline RSA and greater RSA withdrawal would both predict better IC.

The second study aim was to examine the role of adversity experienced early in life in understanding the RSA and IC relationship. We examined early adversity as a comprehensive measure of cumulative risk exposure. Building on previous research (Atkinson et al., 2015; Evans & Kim, 2007; Evans, Li, & Whipple, 2013), we conceptualized cumulative risk as an umbrella term for several known risk factors implicated in early adversity, including sociodemographic influences (i.e., single-parent status, household income, mother’s educational attainment), psychosocial influences (i.e., family turmoil, child separation from family, exposure to violence), and physical environmental influences (i.e., residential crowding, noise, housing problems). The cumulative risk framework was chosen to quantify early adversity, given that exposure to multiple risk factors are associated with worse child outcomes than exposure to single risks (Atkinson et al., 2015; Evans & Kim, 2010; Sameroff, Seifer, & McDonough, 2004). Such unweighted, additive scores of cumulative risk have been argued to better capture the nonlinear nature of the variety of stressors associated with early childhood adversity (Atkinson et al., 2015). Greater cumulative risk was hypothesized to predict lower IC performance, as has been shown by previous research (Evans & Kim, 2013; Lengua et al., 2014).

Moderation analyses were specified to examine the interrelationship between RSA reactivity and IC performance as a function of cumulative risk. Based on previous findings (Conradt et al., 2014, 2016), we hypothesized that cumulative risk would moderate the relationship between RSA reactivity and IC, such that patterns of RSA reactivity would be differentially predictive of regulated behavior on the basis of degree of cumulative risk exposure. Specifically, we were interested in whether patterns of RSA withdrawal are similarly facilitative of behavioral regulation (indexed by IC) for children who have experienced lower and higher levels of cumulative risk.

2 METHODS

2.1 Participants

Mother–child dyads were recruited for participation as part of a larger study of parenting and family processes (for more details, see Skowron, Cipriano-Essel, Benjamin, Pincus, & Van Ryzin, 2013). Dyads qualified for participation if mothers were age 18 years or older, spoke fluent English, and were living with their preschool-age children. Participants were 184 preschool children ranging in age from 3 to 5 years (M = 3.81, SD = 0.76). The majority of participants were female (54.5%), White (80.1%), lived in two-parent households (59.2%), and had mothers who had obtained at least a high school diploma (89.0%). A majority of families reported annual household incomes at or below $30,000 (70.6%; range <$10,000–$50,000+). Of the sample, 44.0% had no documented child maltreatment present, and the remaining had experienced physical abuse or neglect to varying degrees of severity, based on documentation from Child Protective Services.

2.2 Measures

2.2.1 Cumulative risk

Early exposure to life stressors was quantified by a composite measure of cumulative risk exposure. Cumulative risk was calculated as an unweighted additive scale comprising: (a) sociodemographic; (b) psychosocial; and (c) child maltreatment risk factors, with eight potential risk factors included in this study. Each risk factor was coded dichotomously as 1 for present or 0 for not present, with the exception of child maltreatment, which was coded based on severity of maltreatment. Three measures of sociodemographic risk included low income (<$30,000), single-parent status, and maternal high school dropout status. In addition, there were three measures of psychosocial risk: family turmoil, child separation from home, and exposure to community violence. Family turmoil was assessed using the Life Experiences Survey (LES; Sarason, Johnson, & Siegel, 1978), which is a 60-item questionnaire that aims to assess mothers’ perceptions of positive and negative life events experienced during the previous year. Scores ranged from 0 to 17, and respondents in the upper quartile (at or above a score of 4) were coded as 1 (risk present). Child separation from home was assessed according to whether or not the child had been placed in foster care (1 = yes, 0 = no). Exposure to violence was assessed via a parent-reported Children’s Exposure to Community Violence (CECV) survey (Rich特斯 & Saltzman, 1990), a 54-item scale that is used to assess the frequency at which a child has been victimized.
by, witnessed, or heard about 20 forms of violence and violence-related activities in their community. As with family turmoil, respondents in the upper quartile (at or above a score of 8) were coded as 1 (risk present). Last, child maltreatment (CM) subtype and severity was coded from child welfare case records using the Maltreatment Classification System (MCS; Barnett, Manly, & Cicchetti, 1993). Risk in the form of CM exposure was calculated on a three-point scale (0–2): 0 signified no CM exposure, 1 point was given for the presence of CM, and 2 points given for a MCS severity score of 3 or above (severity ranged on a 5-point scale from 1 to 5, with 1 being least severe). Composite scores of cumulative risk (with a total of eight possible risk factors) were available for 184 participants ($M = 2.21$, $SD = 1.54$; range, 1–6). Full descriptive statistics for cumulative risk responses are shown in Table 1.

### Inhibitory control

IC was measured as a composite performance score from two separate Stroop-like tasks administered by a researcher. First, children performed the Shapes Stroop task (Kochanska, Murray, & Coy, 1997), followed by the more difficult Day/Night Stroop task (Gerstadt, Hong, & Diamond, 1994). During the Shapes task, children were shown pictures of three large fruits and three small fruits, then shown three pictures of a small fruit embedded within a different large fruit (e.g., a small orange embedded in a large banana). Children were asked to point to each of the small fruits embedded within larger fruits (e.g., “show me the little banana”). For each trial, scores ranged from 0 (incorrect), to 1 (incorrect, followed by spontaneous self-correction), and 2 (correct on first response). Final scores ranged from 0 to 6 ($M = 4.80$, $SD = 1.72$). During the Day/Night task, children completed 16 trials during which they were shown a series of cards with pictures of a sun or moon and instructed to say “day” whenever they saw a card with a sun on it and to say “night” when they saw a card with a moon on it. For each trial, scores ranged from 0 (incorrect), to 1 (incorrect followed by spontaneous self-correction), and 2 (correct on first response). Final scores ranged from 0 to 32 ($M = 2.21$, $SD = 1.54$; range, 1–6). Full descriptive statistics for cumulative risk responses are shown in Table 1.

### Respiratory sinus arrhythmia (RSA)

Electrocardiogram (ECG) data were acquired through the use of Ag/AgCl electrodes placed on each participant’s chest in a modified Lead II placement, on the distal end of the clavicle, lower left rib cage, and lower abdomen. The ECG signal was recorded with Mindware Technologies ambulatory electrocardiograph (MW1000A; Gahanna, OH) at a sampling rate of 500 Hz and transmitted via a wireless signal to a computer monitored by a research assistant. The ECG signal was synchronized at acquisition with video recordings of behavior to enable identification of task onset and offset. ECG data were processed offline using Mindware HRV software. Trained research assistants visually inspected 30-s epochs for erroneously identified heartbeats. The resulting interbeat interval time series was then subjected to a fast-Fourier transformation, and power in the respiratory frequency band common to children in this age group (0.24–1.04 Hz) was derived from the spectral density function to estimate RSA values. Child RSA values were averaged across 30-s epochs to create a single score for the baseline period (RSA$_{\text{base}}$) and one for each of the IC tasks (RSA$_{\text{day-night}}$, RSA$_{\text{Shapes}}$). RSA$_{\text{base}}$ data were available for 184 participants ($M = 6.09$, $SD = 1.39$; range, 1.61–9.88). RSA$_{\text{day-night}}$ and RSA$_{\text{Shapes}}$ were averaged together to create a single measure of RSA during IC tasks (RSA$_{\text{task}}$) and were available for 151 participants ($M = 6.03$, $SD = 1.31$; range, 2.40–9.65). RSA reactivity was then calculated for each participant by subtracting RSA$_{\text{base}}$ from RSA$_{\text{task}}$ values. Using this subtraction method, negative RSA reactivity values indicate that RSA levels decreased from baseline to task (i.e., RSA withdrawal), and positive values reflect RSA increases from baseline to task. Outlier values with Z-scores exceeding ±2.73 were winsorized while preserving rank-order.

### Procedures

All procedures used in this study were approved and monitored by the Office for Research Protections. Mother–child dyads completed a 2.5-hr laboratory visit to assess physiological function and parent–child interaction, conducted by a team of two trained experimenters. Upon arrival to the laboratory, participants first gave informed consent, then electrodes for ECG monitoring were applied to mother and child (only child data are reported here). Then, a baseline physiological recording was taken during a 5-min resting period while mother and child were seated together on a couch in a room with dimmed lights, viewing a relaxing video depicting slow-moving animals with soothing music. Following the baseline data collection, children, accompanied by their mothers, participated in four tasks lasting approximately 40 min (not reported here) and a short snack break, then completed the Shapes and Day/Night tasks while physiology was monitored. Families were paid $150 to complete the protocol and were provided transportation, snacks, and small toys for the children.

### Table 1

<table>
<thead>
<tr>
<th>Component</th>
<th>M (SD)</th>
<th>N (%)</th>
</tr>
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<tbody>
<tr>
<td>Sociodemographic risk</td>
<td>0.96 (0.93)</td>
<td></td>
</tr>
<tr>
<td>Low income (≤$30,000)</td>
<td>117 (70.9)</td>
<td></td>
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<tr>
<td>Maternal education less than high school diploma</td>
<td>18 (11.0)</td>
<td></td>
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<tr>
<td>Single-mother household</td>
<td>25 (15.2)</td>
<td></td>
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<tr>
<td>Psychosocial risk</td>
<td>0.62 (0.80)</td>
<td></td>
</tr>
<tr>
<td>Family turmoil (upper quartile)</td>
<td>63 (34.8)</td>
<td></td>
</tr>
<tr>
<td>Child placed in foster care</td>
<td>12 (6.6)</td>
<td></td>
</tr>
<tr>
<td>Exposure to violence (upper quartile)</td>
<td>37 (20.3)</td>
<td></td>
</tr>
<tr>
<td>Child maltreatment risk</td>
<td>0.63 (0.61)</td>
<td></td>
</tr>
<tr>
<td>Positive history, low to moderate severity</td>
<td>90 (48.9)</td>
<td></td>
</tr>
<tr>
<td>Positive history, high severity</td>
<td>13 (7.1)</td>
<td></td>
</tr>
</tbody>
</table>
2.4 Analysis plan

Zero-order correlations were calculated between inhibitory control performance, demographic measures, RSA, and cumulative risk to characterize these associations and identify demographics to be included as covariate predictors of inhibitory control in the multiple regression analyses. Next, we centered RSA and cumulative risk variables for a series of multiple regression analyses in Mplus. First, we examined a basic model predicting inhibitory control from relevant covariates, baseline RSA, RSA reactivity, and cumulative risk. We then examined separate models in which interactions between baseline RSA, cumulative risk, and RSA reactivity were included individually, to identify significant interactions. Finally, post-hoc analyses characterizing the subscales of cumulative risk were conducted to determine the degree to which the results were attributable to maltreatment (0 = no maltreatment, 1 = maltreatment history, 2 = severe maltreatment history), socio-economic (low income, single-parent status, parent high-school dropout), and psychosocial (family turmoil, child separation, exposure to violence) risk factors.

Amongst demographic, baseline RSA, and inhibitory control variables, there was minimal missing data (range 0.0–3.8%). Due to insufficient artifact free data, RSA reactivity was missing for 17.9% of participants. Due to refusal to answer cumulative risk questions, cumulative risk data was also missing for 14.1% of participants. To characterize the nature of participants with missing RSA reactivity and/or cumulative risk data we examined the extent to which missing data was associated with all other variables of interest in a series of independent sample t-tests (for continuous variables) and χ² analyses (for categorical). Neither RSA reactivity missingness nor cumulative risk missingness were associated with any study variable (all ps > 0.10). It was possible to include all participants in the multiple regression analyses using the built-in full information maximum likelihood function in Mplus. We also conducted analyses exclusive to the subsample of participants with full data on all analysis variables in order to replicate results in this subsample.

3 RESULTS

Zero-order correlations between all variables of interest are shown in Table 2. Child age was positively correlated with IC performance, r(177) = 0.43, p < .001; therefore age was used as a predictor for all models and as a covariate with other predictors. Greater IC performance was also associated with lower cumulative risk (r(150) = −0.261, p = .001) and higher baseline RSA, r(175) = 0.162, p = .031. Partial correlations were then examined, controlling for the effects of age, and both associations remained consistent (IC × cumulative risk, r(149) = −0.257, p = .001; IC × baseline RSA, r(174) = 0.181, p = .016).

3.1 Multiple regression analyses

In the baseline model older age was a significant predictor of IC scores (Estimate = 0.424, SE = .061, p < .001) as was cumulative risk (Estimate = −0.228, SE = .068, p = .001) with an estimated 26.1% of variance in IC explained (R-square = 0.261, SE = .058, p < .001). IC scores were not predicted by baseline RSA, or RSA reactivity (all ps > 0.10). Results for the second model, which included an interaction between baseline RSA × cumulative risk were nearly identical with older age (Estimate = 0.505, SE = .08, p < .001) and (Estimate = 0.489, SE = .078, p < .001) and cumulative risk predicting IC (Estimate = −0.130, SE = .038, p = .001). Baseline RSA, RSA reactivity, and baseline RSA × cumulative risk did not predict IC scores (all ps > 0.10; IC R-square = 0.261, SE = .057, p < .001).

The third model included an interaction between RSA reactivity × cumulative risk, indicated that older age (Estimate = 0.508, SE = .076, p < .001), cumulative risk (Estimate = −0.111, SE = .039, p = .005), and the RSA reactivity × cumulative risk interaction (Estimate = 0.107, SE = .042, p = .011) predicted inhibitory control. Higher baseline RSA was also marginally predictive (Estimate = .088, SE = .048, p = .067). An estimated 29.6% of variance in IC was explained (R-square = 0.295, SE = .060, p < .001). We reran this model only including a subsample of participants with full data on all predictors (n = 130) and results were entirely consistent. As shown in Figure 1, greater RSA withdrawal during the task from baseline levels was associated with better IC scores, and this effect decreased as a function of increasing cumulative risk. For children exposed to high cumulative risk, RSA reactivity was not related to IC performance.

Next, post-hoc analyses were conducted exploring the cumulative risk results to investigate whether results were attributable to experiences of maltreatment alone, or related to psychosocial or socio-economic risk. In the model investigating maltreatment risk, older
The goal of this study was to characterize the links between PNS function and IC performance in a sample of preschool-age children with heterogeneous exposure to a range of early life stressors, with particular interest in the extent to which measures of cumulative risk would affect the association between PNS activity and IC performance. Building on previous research, we hypothesized that (a) baseline RSA would be associated with better IC; (b) children who had experienced greater levels of cumulative risk would have lower IC; (c) greater parasympathetic reactivity (i.e., withdrawal) would be associated with better IC; and (d) parasympathetic reactivity (withdrawal) would be more predictive of IC for lower risk children. Results reported here support all four hypotheses and thus provide further evidence that, in young children, the presence of early environmental risk is associated with deficits on cognitive measures of self-regulation (Fisher, Leve, Delker, Roos, & Cooper, 2016) and altered associations between physiological and behavioral indices of regulatory processes (Conradt et al., 2014, 2016; Skowron et al., 2014).

The chaotic nature of early environments characterized by a high number of risk factors may help explain the links between cumulative risk and IC impairment (Evans et al., 2013). Specifically, IC development is believed to be supported by environmental factors, such as responsive caregiving (Kochanska, Murray, & Harlan, 2000), low household chaos (Vernon-Feagans, Willoughby, & Garrett-Peters, 2016), and positive maternal mental health (Crandall, Deater-Deckard, & Riley, 2015). Each of these factors is more likely to be limited in households characterized by high cumulative adversity (Evans et al., 2013). Future research should systematically assess intrinsic and extrinsic factors such as these to identify more causal mechanisms in the association between early adversity and IC (e.g., Fox & Calkins, 2003). Recent studies by Lengua et al. (2007, 2014, 2015) have found that proximal parenting may function as a mediator between cumulative risk and its effects on child cognitive development; in other words, positive parenting may serve to attenuate the harmful impact of contextual risk on development.

Notably, the link between IC and physiological regulation as indexed by RSA reactivity was also moderated by cumulative risk. Specifically, the oft-reported finding that greater RSA withdrawal is linked to higher IC (Graziano & Derefinko, 2013; Holzman & Bridgett, 2017) was observed in our sample only for children with lower levels of risk. These results contribute to the growing body of literature that chronicles the varied associations between PNS function and behavior as a function of early life adversity seen in higher risk samples (Conradt et al., 2014, 2016; Skowron et al., 2014). Of particular interest, greater RSA withdrawal was not associated with better IC in children experiencing high levels of cumulative risk, raising the possibility that deficits in IC associated with cumulative risk are related to the effects of cumulative risk on children’s ability to effectively leverage parasympathetic activity during cognitive challenge. We suggest that the heterogeneous associations between RSA reactivity and IC, in particular the small effect sizes noted in meta-analyses (Graziano & Derefinko, 2013; Holzman & Bridgett, 2017; Zahn et al., 2016), may be attributable to the moderating effect of variables such as cumulative risk, or may be a consequence of a sampling bias in psychological research toward lower risk samples. It is also important to note that resting RSA was associated with IC scores for the sample overall, but did not interact with cumulative risk in predicting behavior, as seen with RSA reactivity. These findings are consistent with reports of resting RSA as a trait-like marker of self-control (Beauchaine & Thayer, 2015), as well as reports in much younger samples from whom RSA reactivity in the first months of life predict greater sensitivity to the negative effects of early adversity on later behavioral problems (Conradt et al., 2016).

A final notable aspect of results from our study is that the composite measure of cumulative risk that considered risk stemming from CM exposure, psychosocial, and socio-economic risk, was most...
robust, in terms of amount of variance in children's inhibitory control that was explained, relative to models that considered any single type of risk factor. Nonetheless, it should be noted that socio-economic forms of risk also showed unique associations with children's RSA withdrawal and IC, which was not the case for the other two types of risk factors comprising the cumulative risk battery (i.e., CM exposure and psychosocial risks). This pattern of results is consistent with other studies of cumulative risk, which have found that cumulative indices are generally more predictive of biobehavioral outcomes than single dimension risk indicators (Atkinson et al., 2015; Evans et al., 2013). A challenge in the cumulative risk literature is that widely varied conceptualizations of risk come with a potential tradeoff in terms of the specificity of theoretical mechanisms to be targeted by translational scientists (Evans et al., 2013). However, several researchers have noted that varied risks experienced early in life tend to cluster together (Evans, 2003; Masten & Coatsworth, 1998; Sameroff & Rosenblum, 2006), supporting the notion that cumulative risk more accurately represents the range of adversity children may experience. Yet, in terms of relative contributions of individual indicators, socio-economic risk often shows robust associations between cognitive performance and neurobiological measures (Farah et al., 2008; Stevens et al., 2009).

In terms of more-clinical applications, multiple interventions (e.g., Tools of the Mind, Kids in Transition to School) designed for children with early adversity exposure (low-income homes, maltreatment) have been documented to improve children's IC or closely related aspects of self-regulation (Blair & Diamond, 2008; Pears, Kim, & Fisher, 2012). Self-regulation-focused and parent-focused interventions, such as Parent–Child Interaction Therapy (PCIT), are also strong candidates for improving children's IC (e.g., Fisher & Skowron, 2016). Future research would do well to examine the pathways through which these interventions improve IC and how measuring children's underlying biology can help understand intervention-related IC improvements. One possibility is that interventions such as these strengthen the association between PNS function and IC for at-risk children, resulting in PNS–IC links that are similar in children from lower risk environments. Another possibility is that the extent to which PNS function is supportive of (or at least associated with) IC is "programmed" relatively early in life, and interventions help at-risk children develop more effective IC through alternate pathways, such as changes in goal-directed motivation supported by sympathetic nervous system engagement (Brenner & Beauchaine, 2011). Studies have increasingly reported a link between blunted sympathetic nervous system activity and reduced behavioral regulation during development (Beauchaine et al., 2013; Hinnant, Erath, Tu, & El-Sheikh, 2016). Interactions between the sympathetic nervous system and the PNS may be critical in that changes in sympathetic nervous system function might feed up a greater dynamic range for parasympathetic reactivity in terms of autonomic space (Berntson, Cacioppo, Quigley, & Fabro, 1994), thus enabling more dynamic physiological engagement in fluctuating environmental demands.

It is necessary to note several limitations of this study. First, the data are cross-sectional and cannot lend themselves to causal interpretations; while results support an inverse relationship between cumulative risk exposure and the degree of association between IC and RSA reactivity, it cannot be concluded that the former causes the latter. However, there is some evidence that adversity experienced during the prenatal environment has a causal effect on RSA reactivity (Kaplan, Evans, & Monk, 2008; Monk et al., 2004), and that a profile of greater RSA reactivity in infancy is associated with later deficits in self-regulation during early childhood (Conradt et al., 2016). In addition, the study sample was predominantly White, of lower socio-economic status, and from rural communities, so findings may be limited in their generalizability. In terms of cumulative risk, this study relied upon self-report measures by parents of their child's early environment, without including physical measures of household risks, that have been included in previous reports on cumulative risk (Evans & Kim, 2007). Thus, measures of cumulative risk in our study may have underestimated the extent of risk exposure for individual children. We also note that cumulative risk was not extremely high in the present sample, so results may not replicate in samples on the highest end of the cumulative risk continuum.

Taken together, these results replicate previous findings that exposure to early adversity is associated with deficits in children's self-regulation abilities and that successful child self-regulation is associated with parasympathetic reactivity (i.e., RSA withdrawal). We extended these findings by demonstrating that parasympathetic reactivity is not predictive of self-regulation abilities for children who have experienced a higher degree of exposure to an array of life stressors. This raises the possibility that deficits in childhood self-regulation associated with early risk exposure may have to do with the effectiveness with which children engage their PNS, and that this process might be altered by early exposure to a variety of risk factors. These results add to a growing evidence base for intervention and prevention science efforts aimed at improving childhood self-regulation in samples of children often exposed to higher levels of disadvantage early in life.

Acknowledgments

The author thank the members of the Penn State Family Systems Lab for assistance with data collection, transcription, and coding. This project was supported by National Institutes of Health Research grant R01 MH079328 to Elizabeth A. Skowron and funded by the National Institute of Mental Health and Administration for Children and Families/Children's Bureau of the Administration on Children, Youth, and Families as part of the Federal Child Neglect Research Consortium.

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How to cite this article: Giuliano RJ, Roos LE, Farrar JD, Skowron EA. Cumulative risk exposure moderates the association between parasympathetic reactivity and inhibitory control in preschool-age children. Developmental Psychology. 2018;60:324–332. https://doi.org/10.1002/dev.21608