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Sociodemographic risk, parenting, and inhibitory control in early childhood: The role of Respiratory Sinus Arrhythmia

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Abstract

Background—Deficits of inhibitory control in early childhood are linked to externalizing behaviors and attention problems. While environmental factors and physiological processes are associated with its etiology, few studies have examined how they jointly predict inhibitory control. This study examined whether respiratory sinus arrhythmia (RSA) functioned as a mediator or moderator of both cumulative sociodemographic risk and parenting behaviors on inhibitory control during early childhood.

Methods—The sample included 206 children and their biological mothers. At 24, 30, and 36 months of child age dyads participated in a series of laboratory visits in which sociodemographic, parenting, and baseline RSA (RSAB) data were collected. Inhibitory control was assessed at 36 months using a gift-wrap delay task.

Results—A series of structural equation models yielded no evidence that RSAB mediated the relations of risk or parenting on inhibitory control. RSAB moderated the effects of risk, such that high-risk children with low RSAB performed more poorly on tasks of inhibitory control, while high-risk children with high RSAB did not.

Conclusions—These results suggest that higher levels of RSAB may mitigate the influence of environmental risk in early childhood.

Keywords

risk factors; parenting; self-control; RSA; vagal tone

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Supporting information

Additional Supporting Information may be found in the online version of this article:

Introduction

Inhibitory control is the ability to inhibit a dominant behavioral response (Kochanska, Coy, & Murray, 2001). *Ecological-systems models* (e.g., Bronfenbrenner & Morris, 2006) emphasize the influence of both distal and proximal environmental factors on child development, while *family stress models* (Conger & Elder, 1994) suggest that the influence of distal environmental factors (e.g., cumulative sociodemographic risk) on child outcomes may be mediated by proximal factors (e.g., parenting behaviors). However, whether neurophysiological processes should be treated as links in a mediational chain connecting distal and proximal environmental factors to developmental outcomes or as moderators of environmental influence remains unclear. Understanding the etiology of inhibitory control has important implications for clinicians, given that deficits of inhibitory control have been linked to externalizing and attention problems (Schoemaker et al., 2012). In this paper we explore different conceptualizations of the role of one physiological process – the function of the parasympathetic nervous system – in relation to environmental influences on inhibitory control.

Inhibitory control: environmental influences in early childhood

Performance on behavioral measures of inhibitory control improves during early childhood (Carlson, 2005), with rapid gains from 24 to 36 months on tasks with minimal working memory demands, such as delay of gratification tasks (e.g., Kochanska, Murray, & Harlan, 2000; Gagne & Saudino, 2016). Deficits in inhibitory control during this period are strongly associated with symptoms of attention deficit hyperactivity disorder (ADHD; Pauli-Pott & Becker, 2011), and these deficits may be traced, in part, to adverse environmental influence. For example, higher levels of poverty are linked to lower levels of inhibitory control (assessed via delay tasks) among preschoolers (e.g., Evans & Rosenbaum, 2008) and higher levels of cumulative sociodemographic risk are associated with poorer performance on delay tasks among school-aged children (e.g, Noble, Norman, & Farah, 2005).

Family stress models, however, suggest that distal environmental influences on inhibitory control are mediated by proximal processes, such as parenting (Conger & Elder, 1994). Maternal responsiveness (Kochanska et al., 2000) and the degree of dyadic connectedness (i.e., mutual engagement, shared pleasure, and reciprocity) between mothers and their children predicts better performance on a delay tasks (Li-Grining, 2007), whereas harsh parenting is associated with lower levels of compliance (Karreman, van Tuijl, van Aken, & Deković, 2006) and self-regulation (Colman, Hardy, Albert, Raffaelli, & Crockett, 2006) among preschoolers and school-age children. This, together with research indicating that high levels of cumulative sociodemographic risk undermine positive parenting behaviors while promoting negative ones (Kochanska, Aksan, Penney & Boldt, 2007; Popp, Spinrad, & Smith, 2008), outlines a model in which parenting behaviors mediate the effects of risk on inhibitory control. However, evidence supporting this model is sparse: to date only Lengua and colleagues (2007) have reported that the effects of risk on inhibitory control (at 40 months) were mediated by maternal limit-setting and scaffolding.

Inhibitory control and the activity of the parasympathetic nervous system

The parasympathetic nervous system (PNS) is one branch of the autonomic nervous system, and its activity is commonly indexed by respiratory sinus arrhythmia (RSA; Lewis, Furman, McCool, & Porges, 2012). According to polyvagal theory (e.g., Porges & Furman, 2011), high resting or "basal" levels of RSA (RSAB) reflect greater sensitivity to environmental influence and an enhanced capacity to regulate behavior in response to shifting environmental demands (Beauchaine, 2001). *Bioecological models* (e.g., Bronfenbrenner & Morris, 2006) stipulate that RSA functions as a mediator of environmental influence. There is evidence that both risk (for a review, see Propper, 2012) and parenting (Kennedy, Rubin, Hastings, & Maisel, 2004; Moore, 2010) influence RSAB, and that higher RSAB is associated with better performance on measures of sustained attention (Suess, Porges, & Plude, 1994), effortful control (Taylor, Eisenberg, & Spinrad, 2015), and executive function (Marcovitch et al., 2010). A recent meta-analysis found that higher RSAB is modestly associated with better performance on multiple measures of self-regulation (Holzman & Bridgett, 2017). Alternatively, the theory of *biological sensitivity to context* (BSC; Ellis & Boyce, 2008) suggests that RSA moderates the effects of environment on inhibitory control. Recent studies found that RSAB moderated the effects of socioeconomic status (SES) on delay-task performance (Sturge-Apple et al., 2016) and the influence of parenting on executive functions (which encompasses inhibitory control; Gueron-Sela et al., 2016), though the findings diverged as to whether low (Gueron-Sela et al., 2016) or high (Sturge-Apple et al., 2016) RSAB exacerbated the influence of low SES. These findings are not necessarily contrary to those reporting a direct link between RSAB and self-regulation: in studies conducted with sociodemographically diverse samples, no direct effect of RSAB on inhibitory control was observed (Sturge-Apple et al., 2016), while in studies conducted with low-income samples, high RSAB predicted *poorer* performance on tasks of behavioral self-regulation (e.g., Davies, Sturge-Apple, Cicchetti, Manning, & Zale, 2009).

Current study

This study addresses how sociodemographic risk, parenting, and RSAB may influence the development of inhibitory control by examining whether RSAB functions as a mediator or moderator of these environmental influences. Given the conflicting literature, we make no hypothesis as to which function for RSAB is more likely. If RSAB functions as a mediator, we anticipate that higher levels of risk and negative parenting behaviors will be associated with lower RSAB, which will in turn be associated with poorer inhibitory control, while higher levels of positive parenting behaviors will be associated with higher RSAB and better inhibitory control. If RSAB functions as a moderator, we expect that the effects of risk and negative parenting behaviors on inhibitory control will be exacerbated for children with higher RSAB and that the effects of positive parenting behaviors will be amplified.

Methods

Participants

Participants were healthy full-term infants recruited for a longitudinal study at 3 months of age. The sample included 206 children (48.5% female) and their biological mothers, over half of whom were African American (n = 121, 58.7%; European American, n = 85, 41.3%).

At 3 months (the first age of data collection) 25.9% of families were below 100% of the federal poverty level (Mean income-to-needs ratio = 2.69, *SD* = 2.42). For additional details about sample demographics, see Holochwost et al., 2016. During the first three years 27 participants (13.1% of the initial sample) were lost to attrition. Likelihood of attrition was unrelated to child gender ($p = .970$), ethnicity ($p = .676$), or cumulative risk ($p = .526$).

Procedures

Children and their mothers participated in home and/or laboratory visits at 24, 30, and 36 months of child age. Visits included a four-minute period for the collection of resting cardiac data and a ten-minute period of free play in which the mother played with her child "like she would if she had free time during the day." Free-play sessions were videotaped and subsequently coded (see below). At each visit mothers completed questionnaires.

During the 36-month visit children participated in the gift-wrap task (Kochanska et al., 2000). In this task an experimenter brings a gift and wrapping supplies into a room where the child is waiting. During the wrapping phase, the child is told to sit with his or her back to the experimenter for one minute without peeking while the gift is wrapped. The experimenter then leaves the room to retrieve a bow, thus initiating the bow phase, returning after two minutes. During this time the child is told not to open the gift. The child's behavior was videotaped and subsequently coded (see below).

Ethical considerations

Mothers provided informed consent and permission for the children to participate, and all procedures were approved by the Institutional Review Board at the University of North Carolina Chapel Hill.

Measures

Cumulative risk (30 and 36 months)—Mothers provided data regarding their marital status, highest degree/certification, number of children in their household (age ≤ 18 years), annual pre-tax household income, and whether they had given birth while a teenager. Responses were dichotomized to indicate sociodemographic risk using thresholds based on previous research (e.g., Burchinal, Roberts, Hooper, & Zeisel, 2000) and cumulative risk scores at each age were calculated as the proportion of risk factors to the number of risk factors for which data were provided (Mistry, Benner, Biesanz, Clark, & Howes, 2010). Cumulative risk scores were highly correlated ($r(176) = .84$, $p < .001$) at 30 ($M = .23$, $SD = .26$) and 36 months ($M = .24$, $SD = .26$). Therefore, a composite risk score was calculated as the mean of these proportion scores for cases where scores were available for at least one age ($\alpha = .91$; see Holochwost et al., 2016).

Parenting behaviors (24 and 36 months)—Maternal behaviors during free-play were rated by two observers on seven scales (NICHD Early Child Care Research Network, 1997). Average inter-rater reliability was .90 (24 months) and .92 (36 months). Sensitivity scores were calculated as the mean of the five scale scores that loaded on a sensitivity factor ($\alpha = .90$ and .84 for the 24- and 36-month scores, respectively), while negative-intrusiveness scores were calculated as the mean of the two scores that loaded on a negative-intrusiveness

factor (α = .82 at both ages). Scores at 24- and 36-months were positively correlated (sensitivity: $r(152) = .51$, $p < .001$, negative-intrusiveness: $r(152) = .60$, $p < .001$) and therefore cross-age composites were calculated (after Propper, Willoughby, Halpern, Carbone, & Cox, 2007). For further detail, see Mills-Koonce, Propper, and Barnett (2012).

Respiratory Sinus Arrhythmia (24, 30, and 36 months)—Cardiac data were collected during a 4-min period of rest using a pair of pediatric electrodes affixed to the child's chest. Output was amplified and sent to a heart inter-beat interval (IBI) monitor (Mini Logger; Mini-Mitter/Respironics, Bend, OR) for R-wave detection. Files containing IBI data were edited for movement artifacts using MXEdit software (Delta Biometrics, Bethesda, MD), and files requiring editing of more than 10% of data were excluded from subsequent analyses. Measures of RSA were extracted using Porges' (1985) moving 21-polynomial method (band-pass filter set to 0.24 – 1.04 Hz). Mean RSA was calculated for every 15-s epoch during the 2-min period of rest (after Huffman et al., 1998), and RSAB was calculated as the mean of these epochs. As values of RSAB were significantly inter-correlated ($r(41, 82) = [.38, .49]$, $p = [< .001, .014]$), a cross-age composite measure of RSAB was calculated for all children who had RSAB values for at least two ages ($N = 112$, $M = 5.16$, $SD = 1.26$; results reported below were robust to the inclusion of children with RSAB values at only one age).

Inhibitory control (36 months)—The gift-wrap task developed by Kochanska and her colleagues (2000) is a simple delay task that is appropriate for children ages two to four (Spinrad et al., 2007). The task yields several latency scores for the two-minute bow phase: latency to touch ($M = 54.07$ s, $SD = 52.86$ s), lift ($M = 79.52$, $SD = 51.90$), and open the gift ($M = 105.19$, $SD = 36.32$), all of which are calculated as time elapsed from the start of the phase. Following Kochanska et al. (2000), we calculated a composite score for the bow phase as the mean of the latency to touch, lift, and open the gift (α = .79; $M = 79.59$, $SD = 40.06$). The composite score for the bow phase was used as our measure of inhibitory control, given that performance on delay tasks in early childhood is most predictive of outcomes when it is assessed absent adult guidance (Mischel, Shoda, & Rodriguez, 1989).

Missing data

Data could be missing due to attrition or because a mother or child did not complete a measure during a lab visit. Cumulative risk scores were available for 183 children, parenting behaviors for 176 mother-child dyads (i.e., behaviors were available at either 24 months, 36 months, or both ages), RSAB for 112 children (with measures at two or three ages), and gift-wrap scores for 164 children. Missing data for these measures were regressed on all other measures and child gender and ethnicity. African American children were more likely to be missing RSAB data at a rate approaching significance ($B = 0.72$, Wald (1) = 3.67, $p = .056$). Missingness of RSAB was classified as missing at random (MAR) and ethnicity was included as a covariate in subsequent models (Alison, 2009).

Data analysis

Our hypotheses were evaluated via a series of structural equation models. The first set of models established relations among risk, parenting, and inhibitory control (see Figure 1a).

The next set of models (Models 1 and 2) specified RSAB as a mediator of the effects of both risk and parenting on inhibitory control (see Figure 1b), while the third and final set of models (Models 3 and 4) specified RSAB as a moderator (see Figure 1c). Separate models were run for each dimension of parenting (sensitivity and negative-intrusiveness) to maximize the likelihood of obtaining reliable parameter estimates in a small sample (Bollen, 1989) and to prevent attenuating the estimates of parenting's effects on inhibitory control (Preacher & Hayes, 2008). Risk, parenting, and RSAB were measured as cross-age composites. Therefore, we examined an alternate set of models in which parenting and RSAB at 36 months mediated the effects of risk at 30 months. While sacrificing the more stable measures offered by the cross-age composites, this alternate establishes the temporal precedence of risk.

All models were tested in MPlus 7.0 (Muthén & Muthén, 2015) using full-information maximum likelihood estimation. Each model was assessed using multiple fit indices, with the Akaike (AIC) and Bayesian Information Criteria (BIC) used to compare the relative fit of each pair of models specifying RSAB as either a mediator or moderator of risk and each dimension of parenting. Specific indirect effects and their standard errors were estimated using the products of coefficients approach and the delta method, respectively (Bollen, 1989). Moderation was assessed via interaction terms between mean-centered variables and significant interactions were probed using online utilities (Preacher, Curran, & Bauer, 2006).

Results

Preliminary Analyses

Table 1 presents descriptive statistics and bivariate correlations among study variables. Female (r (164) = −.22, p = .004) and European-American (r (164) = .30, p < .001) participants performed better on the gift-wrap task; therefore, these were covariates in all models. European-American ethnicity was associated with lower levels of cumulative risk (r (183) = −.39, p < .001), higher levels of sensitivity (r (176) = .38, p < .001), lower levels of negative-intrusiveness (r (176) = −.46, p < .001), and lower levels of RSAB (r (112) = .24, p = .012). Cumulative risk was associated with lower levels of sensitivity (r (174) = −.48, p < .001) and higher levels of negative-intrusiveness (r (174) = .54, p < .001), while higher levels of sensitivity (r (164) = .32, p < .001) and lower levels of negative-intrusiveness (r (164) = −.48, p < .001) were associated with better performance on the gift-wrap task. The orthogonal relation of RSAB and performance on the gift-wrap task raises questions about the veracity of models in which RSAB is specified as a mediator.

Model Specification and Testing

Preliminary models—Results of our preliminary models are presented in the Supplementary Materials. In brief, higher levels of cumulative risk predicted lower levels of maternal sensitivity (B = −.39, p < .001) and inhibitory control (B = −.24, p = .003). Maternal sensitivity was not related to inhibitory control (p = .131) and did not mediate the effects of risk on inhibitory control (specific indirect effect: p = .143). In contrast, higher levels of risk predicted higher negative intrusiveness (B = .44, p < .001), which predicted

lower levels of inhibitory control ($B = -.33$, $p < .001$). Negative-intrusiveness mediated the effects of risk on inhibitory control (specific indirect effect: $B = .14$, $p < .001$).

RSAB as a mediator: Models 1 and 2—Based on these results, models 1 and 2 were specified differently: in model 1, RSAB and sensitivity were allowed to mediate the effects of risk on inhibitory control in parallel, whereas in model 2 RSAB was allowed to mediate the effects of negative-intrusiveness on inhibitory control, which in turn mediated the effects of risk. Model 1 was a good fit to the data (see Table 2). While there was a significant direct effect of risk on both sensitivity ($B = -.39$, $p < .001$) and inhibitory control ($B = -.25$, $p = .001$), neither the effect of sensitivity ($B = .13$, $p = .102$) nor RSAB on inhibitory control was significant ($B = .15$, $p = .091$; see Figure 2a). The test of the specific indirect effect of risk on inhibitory control through RSAB was also not significant ($p = .317$).

Model 2 was an acceptable fit to the data (see Table 2). Higher levels of cumulative risk were associated with higher levels of negative-intrusiveness ($B = .44$, $p < .001$) and lower levels of inhibitory control ($B = -.18$, $p = .007$); higher levels of negative-intrusiveness were associated with lower levels of inhibitory control ($B = -.36$, $p < .001$; see Figure 2b). However, negative-intrusiveness was not significantly related to RSAB ($B = -.15$, $p = .203$), nor was RSAB associated with inhibitory control ($B = .05$, $p = .498$). The test of the specific indirect effect from risk to inhibitory control through negative-intrusiveness and then RSAB was not significant ($p = .552$). The alternative models used to establish temporal precedence yielded similar results (see Supplementary Material).

RSAB as a moderator: Models 3 and 4—In model 3 RSAB was allowed to moderate the effects of risk and sensitivity on inhibitory control. This model was a good fit to the data (see Table 2). The interaction between RSAB and risk was significant ($B = .22$, $p = .017$), while the interaction between RSAB and sensitivity was not ($B = .07$, $p = .455$; see Figure 3a). The smaller values of both the AIC and BIC for this model indicated that it was a superior fit to the data (i.e., the divergence from the true model is smaller) than model 1, in which RSAB mediated the effects of risk. Moreover, while adding RSAB as a mediator uniquely accounted only for 2% of variance in inhibitory control ($\Delta R^2 = .02$), including RSAB as a moderator uniquely accounted for 7% of the variance, or nearly a third of the proportion collectively accounted for by risk, ethnicity, gender, and sensitivity.

In model 4 RSAB was allowed to moderate the effects of risk and negative-intrusiveness on inhibitory control. The model was an acceptable fit to the data (see Table 2). The AIC and BIC indicated that this model was a better fit to the data than model 2. The results were similar to those produced by the moderation model including sensitivity: RSAB moderated the effects of risk ($B = .24$, $p = .011$), but not negative-intrusiveness ($B = -.17$, $p = .089$; see Figure 3b).

Post-hoc analyses

Given the similar results across models 3 and 4, the model including sensitivity was selected for post-hoc probing. Simple slopes for the relation between risk and inhibitory control were calculated at low (−1 *SD*) and high (+1 *SD*) levels of RSAB. Among children with low RSAB, higher levels of risk were associated with significantly lower levels of inhibitory

control (B = −.81.7 (SE = 22.3), t = −3.66, p < .001). Among children with high RSAB, no significant decrement in inhibitory control was observed at high levels of risk (p = .571).

Discussion

This study examined whether RSAB functioned as a mediator or moderator of environmental influence on inhibitory control. While our preliminary models indicated that negative-intrusiveness fully mediated the effects of risk on inhibitory control, there was no evidence that RSAB mediated the effects of risk or either dimension of parenting on inhibitory control. This finding is consistent with results reported by Sturge-Apple et al. (2016, study 2), and underscore the possibility that in socioeconomically-diverse samples a direct relation between RSAB and inhibitory control may not be observed. In contrast, our models indicated that RSAB moderated the effects of risk on inhibitory control: for children with low RSAB, there was a significant, negative relation between higher risk and poorer performance on the gift-wrap task; for children with high RSAB, this relation was not observed. Although this finding is consistent with previous studies that found high RSAB to be a protective factor (e.g., Gueron-Sela et al., 2016), it contradicts our hypothesis that high RSAB would exacerbate the effects of risk. This hypothesis was based, in part, on studies reporting high RSAB to be a susceptibility factor (Conradt et al., 2013) and Sturge-Apple et al. (2016)'s finding that children from low-income backgrounds with high RSAB exhibited *poorer* performance on a snack delay task than low-income children with low RSAB.

Some of these differences may be explained by the fact that the role of RSAB was examined with respect to different outcomes (as in the case of Conradt et al., 2013; Gueron-Sela et al., 2016), as well as differences in study design, including the precise constructs measured (risk versus poverty), the ages at which measurement occurred, and the measures used. For example, Sturge-Apple et al. (2016) used a snack delay task in which children were given the choice to eat a small amount of candy now or a larger amount later. The authors argued that for children reared in poverty, consuming a small amount of candy now would be adaptive. We would add that consuming the candy immediately does not entail a violation of a rule or instruction. This is in contrast to the gift-wrap task, in which touching, lifting, or unwrapping the present during the bow phase violates a rule. For children from high-risk environments, rule violation is maladaptive, given the severe consequences relative to those experienced by children from more affluent backgrounds (e.g., Hannon, DeFina, & Bruch, 2013). Thus, in the context of the gift-wrap task, high levels of RSAB support the adaptive behavior of delaying or abstaining from violating the rule among children from high-risk environments.

Conclusion

Clinical Implications and Future Directions

Our results have implications for clinicians who work with young children and high-risk families. Consistent with prior research (e.g., Noble et al., 2005), we found that higher levels of sociodemographic risk predict lower levels of inhibitory control, as do negative-intrusive parenting behaviors. However, our results also suggest that certain patterns of neurophysiological activity can buffer the effects of risk. These findings, together with the

fact that deficits of inhibitory control are implicated in the etiology of externalizing disorders (Pauli-Pott & Becker, 2011; Schoemaker et al., 2012), suggest two courses for intervention designed to reduce the prevalence and severity of these disorders, one reducing negative parenting behaviors by bolstering parents' ability to facilitate their child's exploration while fostering their autonomy, and a second identifying children who are most likely to develop deficits of inhibitory control based on their neurophysiological activity.

While this study focused on RSAB, future research should examine the mediating or moderating role of RSA withdrawal, given that the degree to which children recruit parasympathetic resources is also linked to inhibitory control (Holzmann & Bridgett, 2017). Moreover, while we found evidence that RSA functions as a moderator during the third year of life, physiological systems may be more open to environmental influence earlier in development, and therefore may be more likely to function as mediators. As the activity of physiological systems becomes increasingly consolidated, this activity may be more likely to function as a moderator. Understanding how the role of physiological systems change over the course of early childhood – and how that heterochronicity varies with respect to different developmental outcomes – is an important direction for future research.

Our study makes several contributions to the literature. It is the first study of which we are aware to systematically examine whether RSAB functions as a mediator or moderator of both distal (i.e., cumulative risk) and proximal (parenting) factors on inhibitory control, and it addresses this question during a particularly sensitive period for the development of inhibitory control. It is also the first study that demonstrates that negative and intrusive parenting behaviors mediate the effects of risk on inhibitory control. These findings do not settle the question of how RSAB functions with respect to inhibitory control; rather, they raise new questions about the role of physiological function with respect to environment, how that role may change, and how efforts to prevent and treat externalizing and attention problems can best be informed by an understanding of physiological function in early childhood.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Key points

- Inhibitory control is a crucial developmental outcome of early childhood, and deficits in inhibitory control have been linked to externalizing problems.
- While both environment and physiological function are associated with the etiology of inhibitory control, few studies have systematically examined the role of the parasympathetic nervous system (PNS) with respect to environment and inhibitory control.
- This study demonstrated that baseline activity of the PNS, indexed by respiratory sinus arrhythmia (RSA), moderated, rather than mediated, the influence of risk on inhibitory control, such that children exposed to high levels of risk who exhibited high baseline RSA performed better on a gift-wrap delay task of inhibitory control.
- While elucidating the role of RSA with respect to inhibitory control during a sensitive period in its development, this study also underscores the need to consider how that role may change over time.

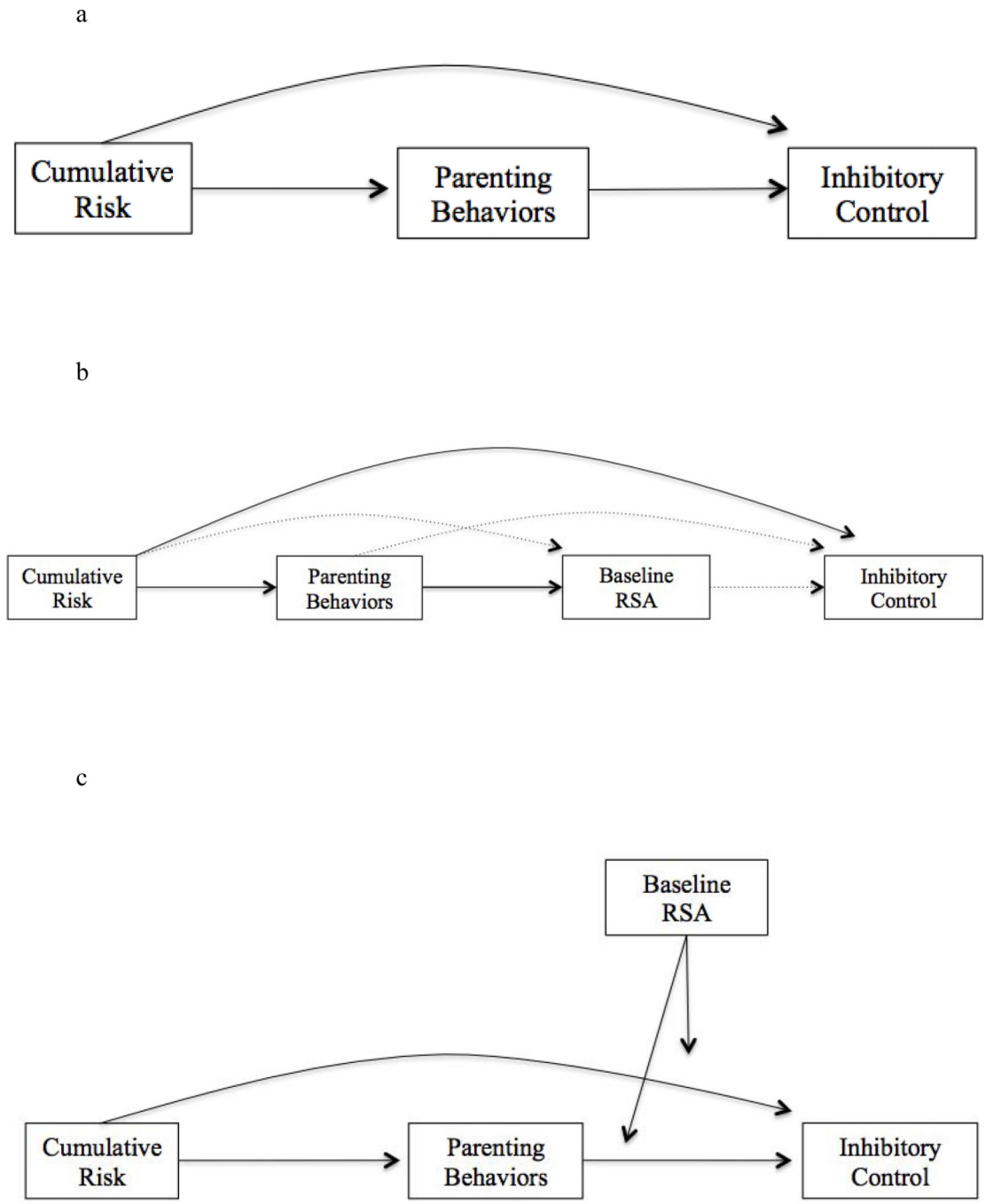


Figure 1.
Conceptual models of possible relations among risk, parenting, RSAB, and inhibitory control

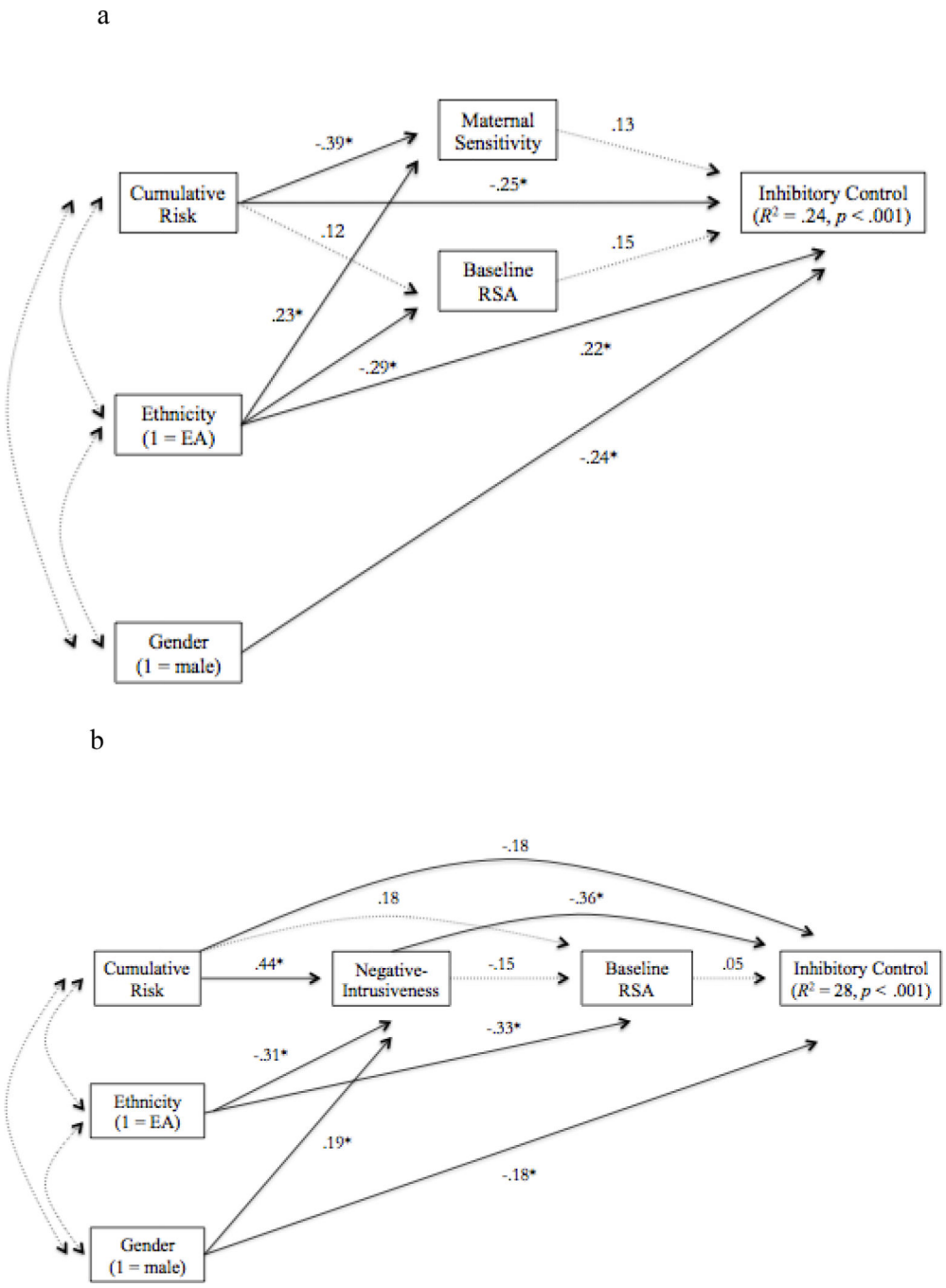


Figure 2.

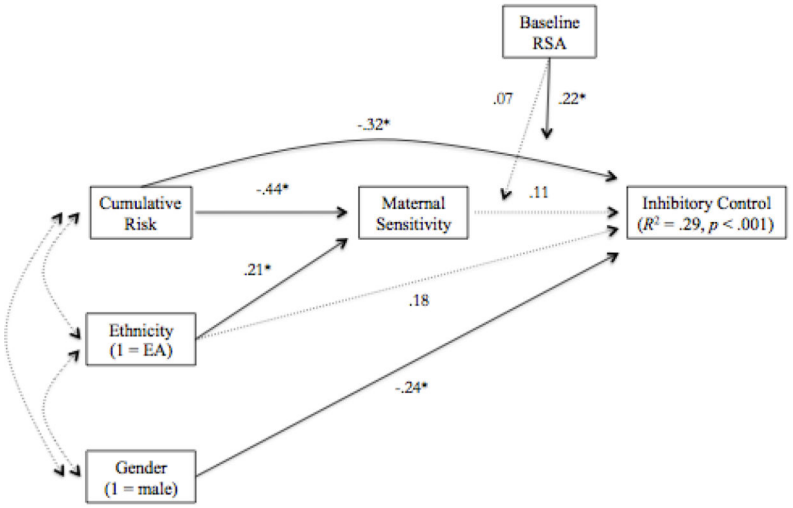
Model 1: RSAB as a mediator of the effects of risk on inhibitory control

Model 2: RSAB as a mediator of the effects of risk and negative-intrusiveness on inhibitory control

Note: Risk was assessed at 30 and 36 months, parenting at 24 and 36 months, RSAB at 24, 30, and 36 months, and inhibitory control at 36 months. All coefficients represent fully-standardized parameter estimates. Solid lines and coefficients with asterisks denote statistically-significant relationships (p < .05), while dashed lines denote relationships that

were not significant. Curved double-headed arrows correspond to correlations among the exogenous variables.

a



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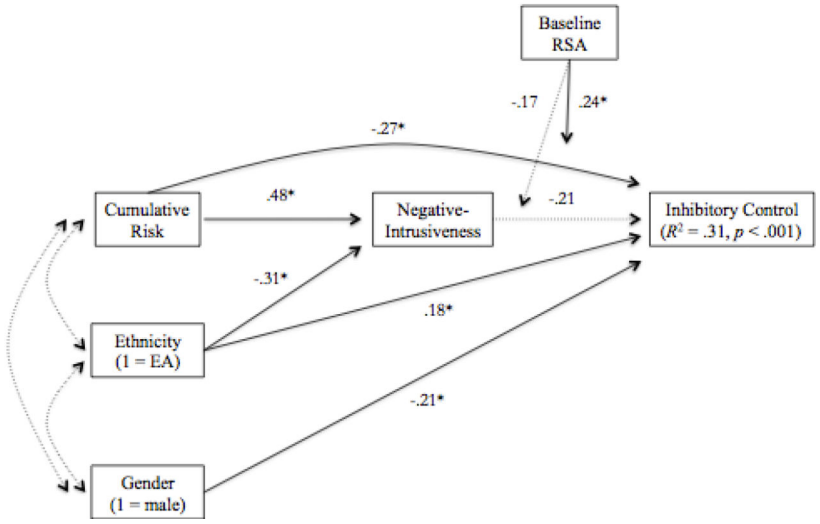

Figure 3.

Model 3: RSAB as a moderator of the effects of risk and sensitivity on inhibitory control

Model 4: RSAB as a moderator of the effects of risk and negative-intrusiveness on inhibitory control

See Figure 2 note.

Table 1:

Pearson correlations among and descriptives for demographics, cumulative risk, parenting, RSAB, and inhibitory control

	1.	2.	3.	4.	5.	6.	7.
1. Gender (male)	---						
2. Ethnicity (EA)	.06	---					
3. Cumulative Risk (30 & 36 mos.)	−.04	−.39**	---				
4. Sensitivity (24 & 36 mos.)	−.05	.38**	−.48**	---			
5. Negative-Intrusiveness (24 & 36 mos.)	.13	−.46**	.54**	−.63**	---		
6. RSAB (24 – 36 mos.)	−.03	−.33**	.24*	−.24*	.13	---	
7. Inhibitory control (36 mos.)	.22**	.30**	−.36**	.32**	−.48**	0	---
N	206	206	183	176	176	112	164
M	.51	.41	.24	4.45	2.78	5.16	79.59
SD	.50	.49	.25	1.07	1.26	1.26	40.06

Note: For pairwise correlations N = [109, 206]. EA = European American.

* $p < .05$

** $p < .01$

Table 2:

Indices of model fit

	Model 1 (Figure 3a)	**Model 2 (Figure 3b)**	**Model 3 (Figure 4a)**	**Model 4 (Figure 4b)**
Parenting behavior	Sensitivity	Negative-Intrusiveness	Sensitivity	Negative-Intrusiveness
Putative role of RSA	Mediator	Mediator	Moderator	Moderator
X^2 (*df*), *p*	3.12 (3), .373	3.24 (2), .198	5.13 (8), .744	10.39 (7), .168
RMSEA, 90% CI	.015, [0, .130]	.060, [0, .173]	0, [0, .080]	.070 [.014, .169]
CFI	.999	.992	1.0	.988
TLI	.995	.950	1.0	.941
SRMR	.023	.019	.030	.040
AIC	2475.1	2492.1	1767.1	1766.8
BIC	2522.5	2542.6	1810.6	1813.0

Note: RMSEA = Root mean square error of approximation; CI = confidence interval; CFI = Confirmatory fit index; TLI = Tucker-Lewis index; SRMR = Standardized root mean square residual; AIC = Akaike Information Criterion; BIC