

# Investigation of a developmental pathway from infant anger reactivity to childhood inhibitory control and ADHD symptoms: interactive effects of early maternal caregiving

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**Background:** ADHD is a neurodevelopmental disorder with a complex pathogenesis. Individual differences in temperamental reactivity – in particular, anger reactivity – are predictive of ADHD. The goal of this study was to examine the moderating (maternal caregiving behaviors; MCB) and mediating (inhibitory control) variables of reactivity using a 9-year multimethod prospective longitudinal design. **Methods:** Participants included 291 children (135 male; 156 female) who participated in a larger study of temperament and social-emotional development. Anger reactivity was assessed by observation of facial anger during an arm restraint task, and MCB were observed during a series of semi-structured mother–infant tasks, both at 9 months of age. Inhibitory control was assessed by performance on a go/no-go task at 5 years of age. ADHD symptoms were assessed by parent and teacher report questionnaires at 7 and 9 years, respectively. **Results:** Anger reactivity and poor inhibitory control were predictive of later ADHD symptoms. Results supported a moderated mediation model, in which the indirect effects of anger reactivity on ADHD symptoms through inhibitory control were conditional on quality of early MCB. Inhibitory control mediated the effect of anger reactivity on ADHD symptoms, but only among children exposed to lower-quality MCB. **Conclusions:** Infant anger reactivity exerts a direct effect on later ADHD from infancy, suggesting anger reactivity as a very early indicator of ADHD risk. Higher-quality caregiving did not buffer against the direct risk of anger reactivity on ADHD but did buffer against the indirect risk by reducing the negative effect of anger reactivity on inhibitory control. Thus, in the developmental pathway from anger reactivity to ADHD, more sensitive, less intrusive parenting supports the development of protective mechanisms (i.e. inhibitory control) to remediate ADHD risk. **Keywords:** ADHD; temperament; anger reactivity; inhibitory control; parenting; infancy.

## Introduction

Attention-deficit/hyperactivity disorder (ADHD) is a neurodevelopmental disorder that can be highly impairing and exert an enduring negative impact on the quality of life of affected children and their families (Barkley, Murphy, & Fischer, 2008; Johnston & Chronis-Tuscano, 2015). Evidence from behavior genetics studies demonstrates that ADHD, as a *DSM-5* or *ICD 10/11* diagnosis, is an extreme manifestation of symptoms that occur on a continuum within the population and that ADHD symptom severity is a function of the strength of a constellation of etiological factors (Larsson, Anckarsater, Rastam, Chang, & Lichtenstein, 2012; Middeldorp et al., 2016; Salum et al., 2014). Early identification and intervention efforts targeting these factors have the potential to reduce symptom severity and improve the quality of life for at-risk children and their families. To do this, we need to understand how ADHD develops. However, the pathogenesis of the disorder is complex – involving multiple causal

pathways that are shaped by many interacting biological and environmental factors – and, as such, it has been challenging to chart clear developmental pathways linking biological liability to symptom emergence (Nigg, 2006; Sonuga-Barke & Halperin, 2010). Large prospective longitudinal studies that measure early indicators of ADHD liability and follow children from infancy through childhood are rare yet ideally suited to elucidating developmental pathways leading to elevated ADHD symptoms. An obstacle to this work has been identifying robust early indicators of ADHD liability due to the heterogeneity of developmental pathways for ADHD (Steinhausen, 2009). Nonetheless, one class of indicators with potential is individual differences in temperament.

Temperament describes constitutionally based individual differences in reactivity to stimuli (i.e. physiological and behavioral responses to the environment) and the regulation of that reactivity (Rothbart & Derryberry, 1981). Understanding the temperamental origins of ADHD has been a fruitful avenue for conceptualizing early ADHD pathways and identifying early temperament-based indicators of ADHD liability (Nigg, Goldsmith, & Sachek, 2004).

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In particular, there is reasonable evidence linking temperamental anger reactivity in infancy and toddlerhood (i.e. responding with negative affect to constraining or frustrating events) to familial risk for ADHD (Auerbach, Atzaba-Poria, Berger, & Landau, 2004; Sullivan et al., 2015) and later childhood ADHD symptoms (e.g. Gurevitz, Geva, Varon, & Leitner, 2014; Willoughby, Gottfredson, & Stifter, 2017). Conversely, infants' and toddlers' distress or negative affect in response to novel or strange stimuli is not as strongly predictive later childhood ADHD symptoms, suggesting potential specificity of anger reactivity as a liability indicator for ADHD symptoms (Miller, Degnan, Hane, Fox, & Chronis-Tuscano, 2019; Willoughby et al., 2017). Thus, evidence of an anger reactivity-to-ADHD pathway is consistent; however, we know little about how this early infant reactivity-related risk translates into childhood ADHD symptoms. In other words: What are the *mediators* and *moderators* of these anger reactivity effects?

The other component of temperament, regulation, also is likely involved in developmental pathways leading to ADHD and a potential mediator of reactivity-related effects (Nigg et al., 2004). Beginning in infancy, there are ongoing reciprocal and transactional relations between reactivity and regulation (Derryberry & Rothbart, 1997). Regulatory skills involve the modulation of reactivity-related behavioral and emotional responses to stimulus events (Lawson & Ruff, 2004); these skills are developed and strengthened by experience (Blair, 2002). For example, infants learn to modulate negative emotions by shifting their gaze away from upsetting stimuli and this strategy serves as a building block for developing more complex self-soothing techniques (Mangelsdorf, Shapiro, & Marzolf, 1995). High levels of anger reactivity can disrupt the development of regulatory skills by interfering with the quantity and quality of experience infants and children have with modulating reactivity (Rothbart, Posner, & Rosicky, 1994). For instance, intense negative emotions can make it difficult to initiate a regulatory skill (i.e. high-intensity emotions are distracting) or successfully implement a regulatory skill (i.e. high-intensity emotions are more difficult to modulate than lower intensity emotions).

One aspect of regulation, inhibitory control, describes the ability to inhibit a dominant or prepotent response in order to initiate a more planful, appropriate one (Rothbart & Bates, 2006). Inhibitory control deficits frequently co-occur with ADHD symptoms and may share common etiologic factors (Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005). For instance, inhibitory control deficits during preschool have been found to mediate the relation between family history of ADHD and child ADHD symptoms (Pauli-Pott, Dalir, Mingebach, Roller, & Becker, 2013). Furthermore, unaffected relatives of children with ADHD tend to have poorer inhibitory

control compared to controls, suggesting inhibitory control deficits are associated with genetic risk for ADHD (Bidwell, Willcutt, DeFries, & Pennington, 2007; Rommelse et al., 2008). Taken together, evidence suggests inhibitory control as a potential early intermediary linking an underlying biological liability to ADHD symptoms.

Rabinovitz, O'Neill, Rajendran, and Halperin (2016) were the first to examine a pathway from anger reactivity to ADHD symptoms with inhibitory control as a mediator. They found higher levels of anger reactivity at age 3–4 years predicted ADHD symptoms at 7 years, and this effect was partially mediated by executive functions, including inhibitory control, at 6 years (measured through performance on a combination of working memory, and set-shifting/inhibitory control tasks). These results suggest anger reactivity as an early indicator of ADHD liability and provide preliminary evidence that this liability contributes to later ADHD through its negative effects on executive function, including inhibitory control. A limitation, however, is that measures of reactivity were obtained at 3–4 years, when executive functions are already developing (Zelazo, Carter, Reznick, & Frye, 1997) and ADHD symptoms can manifest (Lavigne, LeBailly, Hopkins, Gouze, & Binns, 2009). Earlier measures of anger reactivity are therefore needed to clearly establish the antecedent effects of temperamental reactivity on the later development of inhibitory control and ADHD symptoms.

Thus, the first goal of this paper was to replicate in an independent data set the findings of the Rabinovitz study using measures of anger reactivity in infancy. We tested these relations using a 9-year prospective longitudinal design with observational measures of anger reactivity at 9 months, task-performance measures of inhibitory control at 5 years, and parent and teacher report of ADHD symptoms at 7 and 9 years. We hypothesized inhibitory control would mediate the relation between infant anger reactivity and childhood ADHD symptoms: Higher levels of anger reactivity would predict worse inhibitory control, which would, in turn, predict higher levels of childhood ADHD symptoms. Given that child behavioral problems (e.g. oppositional behaviors and conduct problems) also are related to infant anger reactivity and ADHD (Lahey et al., 2008), we included behavioral problems as a covariate to isolate the unique effects of anger reactivity on ADHD.

In addition, as a second aim we examined the early caregiving environment as a potential moderator of the effects of infant anger reactivity on both inhibitory control and ADHD symptoms. As previously mentioned, high levels of reactivity can interfere with the development of regulatory skills (i.e. it is challenging to learn self-soothing skills in a state of intense negative emotional arousal; Rothbart et al., 1994). Sensitive, responsive caregiving can buffer

these interfering effects by providing infants with a source of external regulation that can reduce their negative emotional arousal to a level where they can successfully self-regulate (Kopp, 1989). Empirically, there are several examples of interactive effects of caregiving and reactivity contributing to the development of self-regulation, with lower-quality caregiving behaviors (e.g. less sensitive, more intrusive) increasing the likelihood that higher levels of reactivity translate into poor regulation (e.g. Poehlmann et al., 2012).

Caregiving also has been found to modulate the effects of infant reactivity on ADHD symptoms. In particular, our group found that 4-month reactivity (measured through motor activity and positive affect in response novelty) not only predicted higher levels of parent/teacher reported ADHD symptoms at lower quality of maternal caregiving during infancy, but also predicted lower levels of ADHD symptoms at higher quality of maternal caregiving; these results suggest that, depending on caregiving, reactivity to novelty may exert risk or protective effects on later ADHD symptoms (Miller et al., 2019). Taken together, results of these prior studies suggest a worthwhile extension to the simple mediation model reported by Rabinovitz et al. (2016) in which the transaction between temperament and caregiving predicted both inhibitory control and ADHD. Therefore, the second goal of this paper was to examine whether early maternal caregiving (measured at 9 months through observation) *moderated* the effects of infant anger reactivity on either inhibitory control or ADHD symptoms. To do this, we tested a moderated mediation model. We predicted that the strength of the infant anger reactivity effects on both later inhibitory control and ADHD symptoms would be strongest at lower-quality maternal caregiving.

## Method

### Participants and ethical considerations

Participants were part of a larger sample of 291 children (135 males, 156 females) recruited for a longitudinal study on child socioemotional development conducted in a large metropolitan mid-Atlantic region of the United States beginning in 2001. A total of 779 four-month-old infants, recruited from hospital birth records, participated in temperament screening tasks, and a subgroup of infants was selected to participate based on their reactivity to novelty (Hane, Fox, Henderson, & Marshall, 2008). Infant behaviors were coded for motor activity (frequencies of arm, leg, and body movements), negative affect (frequencies of fussing and crying), and positive affect (frequencies of smiles and positive vocalizations). Infants with higher levels of reactivity on these dimensions were oversampled to represent a wider range of reactivity compared to a randomly selected community sample. Reported when infants were four months old, the ethnic/racial breakdown for parents (mother/father) was as follows: 69.4%/68.7% Caucasian, 16.5%/18.6% African American, 7.2%/5.5% Hispanic, 3.1%/2.7% Asian, 3.4%/3.1% other, and 0.3%/1.4% missing. 16.2% of mothers had a high school education, 41.9% had a college

education, 35.7% had a graduate school education, 5.5% reported other forms of education, and 0.7% were missing.

There were missing data across assessment periods. Reasons for missing data included that participants were unavailable for that assessment period (e.g. moved away, difficulty scheduling) or measure-specific experimenter error (see Measures). Of the sample of 291 children, 155 had valid data for anger reactivity, 241 had valid data for maternal caregiving behaviors, 204 had valid data for inhibitory control, and 204 had valid data for ADHD symptoms (either by parent or teacher report). Excepting mother ethnicity, no key (i.e. anger reactivity, caregiving, inhibitory control, ADHD symptoms, behavioral problems) or demographic variables were related to missingness (missing vs. nonmissing) on either outcome variable (i.e. inhibitory control, ADHD symptoms;  $ps > .08$ ). Compared to children with Caucasian mothers, children with non-Caucasian mothers were more likely to have missing data for parent or teacher report of ADHD symptoms ( $ps < .02$ ), and inhibitory control ( $p = .05$ ). These results suggest that mother ethnicity explains some patterns of missingness in the sample. We retained this variable in our main analyses as an auxiliary (i.e. control) variable to support the assumption that the data were missing at random (Enders, 2010).

Informed consent was obtained from adult participants at each assessment, and procedures were approved by the University of Maryland Institutional Review Board.

### Measures

**Anger reactivity (9 months).** Anger reactivity was assessed during a laboratory visit through an arm restraint task designed to elicit anger (Provost & Gouin-Decarie, 1979). Infants were seated in a car seat placed on a table. Mothers stood behind the car seat and out of sight from infants. Mothers were instructed to gently press their infant's forearms down against their sides for three 30-second trials. The experimenter provided infants a toy to play with between trials to reduce carry over effects.

Infant anger reactivity was measured by facial coding of expressions of anger using the Facial Action Coding System (Ekman, Friesen, & Hager, 2002). Anger was coded by the simultaneous expression of at least two main action unit characteristics of anger prototypes. The main action units included the following: brow lower (AU4), upper lid raiser (AU5), lid tighten (AU7 or AU23), and lips part/jaw drop (AU25/AU26) (see He et al., 2013 for additional details). Coding was completed by two independent coders (21% overlap) using partial interval coding for 30 seconds of each trial with excellent reliability (ICC = 0.97). Trials were divided into 10-second epochs each scored for intensity of anger (0 = *absent*; 1 = *expression barely present or fleeting*, 2 = *clear and strong expression*). Anger reactivity was measured by the average of angry intensity prorated for number of epochs coded (number of epochs coded:  $M = 4.88$ ,  $SD = 2.92$ ). Number of epochs coded was negatively correlated with anger reactivity ( $r(153) = -.70$ ,  $p < .001$ ).

**Maternal caregiving behaviors (MCB; 9 months).** Maternal caregiving behaviors were measured through behavioral coding of maternal sensitivity and intrusiveness across seven semi-structured mother-infant tasks (e.g. feeding, toy-based play) completed during a home visit. Maternal sensitivity was coded using a modified version of Ainsworth's Maternal Care Behavior rating scales (Ainsworth, 1976; Hane & Fox, 2006). Raters provided a global rating (1 = *low*; 9 = *high*) of Acceptance, Sensitivity, Availability, Appropriateness, Delight, and Encouragement. Ratings were averaged for each task and combined into an omnibus sensitivity score. Maternal intrusiveness was coded using reverse-scored ratings of Cooperation from the Ainsworth's scales (1 = *interference*; 9 = *cooperation*) and Intrusiveness ratings

(1 = *not at all*; 4 = *highly*) from Park, Belsky, Putnam, and Crnic (1997). These were standardized and averaged to create an omnibus intrusiveness score. Omnibus sensitivity and intrusiveness scores were standardized. MCB were measured by a composite of standardized sensitivity and intrusiveness ratings calculated as sensitivity – intrusiveness, with higher scores represented sensitive, nonintrusive maternal caregiving. Coding was completed by two trained coders (14% overlap in addition to 6 training cases) who were blind to other data in the study, with good reliability (ICC for sensitivity = 0.69, ICC for intrusiveness = 0.82, and ICC for MCB = 0.85).

**Inhibitory control (5 years).** Children completed the Zoo Game, a modified go/no-go task (Lamm, White, McDer-mott, & Fox, 2012). The objective of the Zoo Game was to help the zookeeper capture animals that had escaped from the zoo, but not to capture the monkeys because they were the zoo keeper's assistant. Children were instructed to press the button on a button box as fast as they could when they saw an animal (go trials) but not to press the button when they saw a monkey (no-go trials). Children were administered 12 practice trials and 120 experimental trials distributed over two blocks each containing 45 go trials and 15 no-go trials. For each trial, a stimulus appeared for 700 ms, followed by a blank screen for 2,300 ms or until the child responded. The intertrial interval was 500 ms. Trials were screened for anticipatory responses and removed if response time was <200 ms. All participants achieved at least 50% accuracy on go trials, indicating they were not randomly responding. The go/no-go task was administered to 209 of the 269 children from the study's sample. Thirteen children were administered a different quantity of go ( $M = 106$ ;  $SD = 7$ ) and no-go ( $M = 14$ ;  $SD = 7$ ) trials. Of these, we removed cases who were administered 10 or fewer no-go trials ( $n = 5$ ) leaving valid data for 204 children. No-go trial accuracy (i.e. % correct) was used as a measure of inhibitory control. The split-half reliability for this task was excellent (using two random halves; Spearman–Brown correlation,  $r = .94$ ).

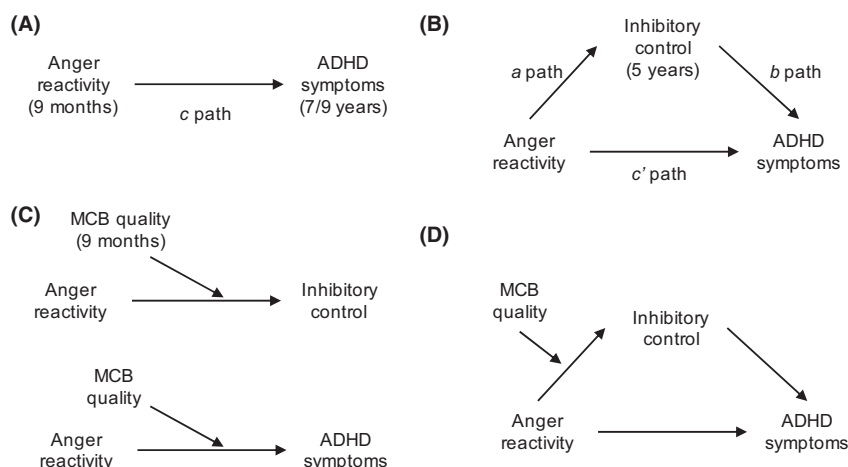
**ADHD symptoms (7 and 9 years).** ADHD symptoms were assessed using parent and teacher report on the Swanson, Nolan, and Pelham-IV (SNAP-IV; Swanson, 1992) 9-item inattention and hyperactivity/impulsivity scales. Raters reported how well each item described the child (1 = *not at all*; 4 = *very much*), and scores were calculated as the average

of items within the scales. Due to constraints of the larger study, parent report data were collected during the seven-year assessment period ( $n = 193$ ) and teacher report data were collected for a subset of participants ( $n = 72$ ) at the nine-year assessment. Sixty-one participants had data for both parent- and teacher report, 132 had data for parent report only, and 11 had only teacher report data. Reliability was excellent for parent and teacher report ( $\alpha s > .93$ ). We have previously used a latent variable of parent and teacher report with inattention and hyperactivity/impulsivity as indicators (Miller et al., 2019). Therefore, to be consistent with our previous study and to maximize information from all available data, ADHD symptoms were measured using a latent variable of parent- and teacher report of inattention and hyperactivity/impulsivity. We also report results using parent report only of ADHD as sensitivity analyses.

The initial measurement model had poor fit (Comparative Fit Index (CFI) = 0.87, Root Mean Square Error of Approximation (RMSEA) = 0.23,  $p = .003$ ). Guided by modification indices, we allowed teacher report residuals to covary. The final measurement model had good fit ( $\chi^2(1) = 2.50$ ,  $p = .11$ , CFI = 0.99, and RMSEA = 0.09,  $p = .20$ ). Relative to parent report inattention, loadings were  $\lambda = 1.00$  (teacher report inattention),  $\lambda = 0.69$  (parent report hyperactivity/impulsivity), and  $\lambda = 0.23$  (teacher report hyperactivity/impulsivity). Measurement invariance testing indicated that loadings did not significantly differ by child sex ( $\chi^2_{diff}(3) = 1.04$ ,  $p = .79$ ).

**Behavioral problems (7 years).** Behavioral problems were assessed using 16 items reflecting oppositional behavior and conduct problems from the SNAP-IV parent report. A four-point Likert scale was used for item ratings. Reliability for this scale was excellent ( $\alpha = .89$ ). Behavioral problems were used in the main analyses as a covariate predictor of ADHD symptoms in order to isolate the unique effects of key predictors on later ADHD symptoms.

**Data analytic plan.** Structural equation modeling with *lavaan* in RStudio (version 1.0.136) was used to test the study's main hypotheses. Figure 1 describes our analytic approach. First, we examined the total effect of infant anger reactivity on childhood ADHD symptoms (Figure 1A). Second, we examined a simple mediation model with 5-year inhibitory control explaining the relation between anger reactivity and later ADHD symptoms (Figure 1B). Next, we tested whether



**Figure 1** Conceptual summary of analytic models. (A) Total effect model; (B) simple mediation model; (C) moderation models; (D) moderated mediation model

quality of MCB was a moderator of anger reactivity effects on either inhibitory control or ADHD symptoms in separate models (Figure 1C) and included significant moderated pathways in a moderated mediation model (Figure 1D).

To test mediation effects, we used the delta method which tests the significance of the product of standard errors for the  $a$  and  $b$  paths, providing a conservative estimate of mediation (Sobel, 1982). We also calculated Monte Carlo confidence intervals for the indirect effect using an interactive online tool with 20,000 repetitions (Preacher & Selig, 2012; Selig & Preacher, 2008). We examined the indirect effect at one standard deviation above and below the MCB mean to understand the nature of the moderated mediation effect (Preacher, Rucker, & Hayes, 2007). In the moderation models, predictors were mean-centered and the interaction term was created by the product of the mean-centered predictors.

A full information maximum-likelihood estimator was used to account for missing data, which provides parameter estimates using all available data and is the recommended method for handling missing data when data meet the assumption of missing at random (Enders, 2010; Kline, 2010). Robust standard errors were used to account for skew and kurtosis among our key variables. We examined the Comparative Fit Index (CFI) and Root Mean Square Error of Approximation (RMSEA) fit indices to determine model fit; CFI values  $\geq 0.95$  and RMSEA values  $\leq 0.05$  are indicative of excellent fit (Tabachnick & Fidell, 2013).

As previously mentioned, mother ethnicity (Caucasian vs. non-Caucasian) was included as an auxiliary variable in the models. Other demographic variables, such as maternal education, were included as covariate predictors in the model if they were significantly correlated with mediator or outcome variables. Covariate pathways among predictor and demographic variables were modeled when they were significantly correlated. Demographic covariates were free to covary with one another.

Given the sex differences in reactivity-related ADHD liability found in our previous work (Miller et al., 2019), we examined the moderating role of child sex by modeling sex as a grouping variable for each analytic model. We assessed for sex differences by comparing the chi-square value from analyses where key variable regression coefficients were constrained to be equal for both sex groups to a chi-square value from analyses allowing separate regression estimates. A significant difference between chi-square values is evidence of moderation by sex.

## Results

### Descriptive statistics

Descriptive statistics and bivariate associations for the key variables are shown in Table 1. Inhibitory control was negatively associated with most measures of ADHD symptoms, and parent and teacher reports of ADHD symptoms were positively associated. We also examined for group differences in children above and below the DSM-5 clinical threshold for ADHD (i.e. six or more symptoms from parent or teacher report on SNAP-IV endorsed at quite a bit or very much; Swanson et al., 2001). Compared to children below the symptom threshold ( $n = 174$ ), children above the threshold ( $n = 30$ ) had higher levels of anger reactivity ( $t(112) = -2.50$ ,  $p = .01$ , Hedges'  $g = .63$ ) and lower levels of inhibitory control ( $t(174) = 2.91$ ,  $p = .004$ , Hedges'  $g = .65$ ). Demographic variables were related to at least one of the key variables (Table 1). Chi-square difference testing indicated child sex was not a moderator of regression coefficient strength in any of the

analytic models ( $ps > .05$ ; see Table 2). Therefore, child sex, along with father ethnicity and maternal education, was retained as covariates.

### Total, direct, and indirect effects of anger reactivity

To address the first aim of the study – inhibitory control would mediate the effects of infant anger reactivity on childhood ADHD symptoms – these analyses examined the total effect of anger reactivity on ADHD symptoms (Figure 1A), as well as the direct and indirect effect of anger reactivity on ADHD symptoms with inhibitory control as a mediator in a simple mediation model (Figure 1B). As described in Table 2, in the total effect model, infant anger reactivity was positively related to childhood ADHD symptoms (i.e.  $c$  path).

In the simple mediation model, infant anger reactivity was not a predictor of either inhibitory control (i.e.  $a$  path) or childhood ADHD symptoms (i.e.  $c'$  path). However, inhibitory control was negatively related to ADHD symptoms (i.e.  $b$  path). Contrary to prediction and previous findings, the indirect effect of anger reactivity on ADHD symptoms through inhibitory control was nonsignificant ( $b = .007$ ,  $SE = .01$ ,  $p = .48$ , 95% CI  $[-0.01, 0.03]$ ).

### Moderated mediation model

To address the second aim of the study – whether higher-quality MCB in infancy could attenuate the effects of anger reactivity – these analyses examined the moderating effect of MCB quality on direct effects, as well as on the indirect effect, between anger reactivity and ADHD symptoms via inhibitory control. Preliminary analyses of separate moderation models revealed that MCB quality moderated the effects of anger reactivity on inhibitory control but not ADHD symptoms (Figure 1C; Table 2). Simple slope analyses examining the relation between anger reactivity and inhibitory control at lower and higher-quality MCB revealed that anger reactivity was negatively related to inhibitory control at lower-quality MCB (i.e.  $-1$  SD;  $b = -.11$ ,  $SE = 0.04$ ,  $p = .005$ ,  $B = -.30$ ), but not significantly related to inhibitory control at higher-quality MCB (i.e.  $+1$  SD;  $b = .05$ ,  $SE = 0.04$ ,  $p = .29$ ,  $B = .12$ ).

Given the findings from our moderation analyses, we examined the moderating effect of MCB quality on the relation between anger reactivity and inhibitory control (i.e. the  $a$  path) in a moderated mediation model (Figure 1D; Table 2). Results revealed that the indirect effects of anger reactivity on ADHD symptoms through inhibitory control were conditional on quality of MCB (interaction indirect effect:  $b = -.01$ ,  $SE = 0.007$ ,  $p = .05$ , 95% CI  $[-0.03, -0.001]$ ). Follow-up analyses examining the indirect effect of anger reactivity across lower, average, and higher-quality MCB (Table 3) revealed an indirect effect at lower-quality MCB (i.e.  $-1$  SD) and no indirect effect

**Table 1** Descriptive statistic and bivariate associations for key and demographic variables

	1. Anger	2. MCB	3. IC	4. IA-P	5. HI-P	6. IA-T	7. HI-T	8. BP
1. Anger reactivity (9 months; observed)	0.10							
2. MCB quality (9 months; observed)	-0.06	0.07						
3. Inhibitory control (5 years; task)	0.07	-0.05	-0.25***					
4. Inattention (7 years; P)	0.19 <sup>†</sup>	-0.07	-0.17*	0.66***				
5. Hyperactivity/impulsivity (7 years; P)	0.06	-0.16	-0.46***	0.66***	0.52***			
6. Inattention (9 years; T)	0.06	0.10	-0.15	0.20	0.27*	0.46***		
7. Hyperactivity/impulsivity (9 years; T)	-0.03	0.003	-0.03	0.52***	0.60***	0.38**	0.07	
8. Behavioral problems (7 years; P)	-0.02	0.05	-0.21**	0.18*	0.27***	0.33**	0.15	0.25***
Male child	0.20*	0.35***	0.20**	0.03	0.001	-0.21 <sup>†</sup>	0.14	
Mother Caucasian	0.17*	0.36***	0.17*	-0.09	-0.08	-0.31**	0.12	-0.06
Father Caucasian	-0.10	-0.24***	-0.05	0.11	0.09	0.12	0.07	0.10
Mother high school education	-0.05	0.18**	0.08	-0.02	-0.02	0.05	-0.10	-0.05
Mother graduate school education	155	241	204	193	193	72	71	193
<i>M (SD)</i>	1.11 (0.76)	0.00 (1.79)	0.50 (0.28)	1.77 (0.56)	1.69 (0.58)	1.81(0.83)	1.46 (0.57)	1.36 (0.34)
Min	0.00	-6.00	0.00	1.00	1.00	1.00	1.00	1.00
Max	2.00	5.14	0.97	3.78	3.78	4.00	3.89	2.63
Skew	-0.11	-0.75	-0.41	1.00	1.02	1.04	2.04	1.11
Kurtosis	-1.53	0.81	-0.95	1.30	0.69	0.11	5.04	0.89

BP, behavioral problems; HI, hyperactivity/impulsivity; IA, inattention; IC, inhibitory control; MCB, maternal caregiving behaviors; P, parent report; T, teacher report.  
<sup>†</sup>*p* < .10; \**p* < .05; \*\**p* < .01; \*\*\**p* < .001.

**Table 2** Summary of results from the analytic models (*n* = 291)

	Total effect		Simple mediation		Moderation: inhibitory control		Moderation: ADHD symptoms		Moderated mediation	
	<i>b</i> ( <i>SE</i> )	<i>B</i>	<i>b</i> ( <i>SE</i> )	<i>B</i>	<i>b</i> ( <i>SE</i> )	<i>B</i>	<i>b</i> ( <i>SE</i> )	<i>B</i>	<i>b</i> ( <i>SE</i> )	<i>B</i>
<b>Effects on ADHD symptoms</b>										
Anger reactivity	.10* (0.05)	.17	.08† (0.05)	.14			.10* (0.05)	.17	.08† (0.05)	.14
MCB							-.01 (0.02)	-.05		
Anger reactivity × MCB							.03 (0.02)	.11		
Inhibitory control			-.30* (0.13)	-.19					-.30* (0.13)	-.19
Behavioral problems	.84*** (0.12)	.14	.86*** (0.11)	.66			.85*** (0.11)	.65	.86*** (0.11)	.66
Male child	.12* (0.06)		.09 (0.06)	.10			.12* (0.06)	.14	.09† (0.06)	.10
Father Caucasian	-.09 (0.07)	-.10	-.06 (0.07)	-.07			-.06 (0.07)	-.06	-.06 (0.07)	-.07
<b>Effects on inhibitory control</b>										
Anger reactivity			-.02 (0.03)	-.06	-.03 (0.03)	-.09			-.03 (0.03)	-.08
MCB					.02 (0.01)	.11			.02 (0.01)	.10
Anger reactivity × MCB					.04** (0.02)	.23			.04** (0.02)	.23
Male child			-.10** (0.04)	-.18	-.11** (0.04)	-.19			-.10** (0.04)	-.18
Father Caucasian			.10* (0.05)	.16	.09† (0.05)	.14			.09† (0.05)	.15
<b>Model fit</b>										
$\chi^2$	$\chi^2(16) = 29.24, p = .02$	.02	$\chi^2(34) = 48.73, p = .05$	.05	$\chi^2(7) = 4.24, p = .75$	.75	$\chi^2(38) = 40.59, p = .36$	.36	$\chi^2(46) = 61.93, p = .06$	.06
CFI	0.98		0.97		1.00		0.99		0.97	
RMSEA	0.05, <i>p</i> = .39		0.04, <i>p</i> = .78		0.00, <i>p</i> = .95		0.02, <i>p</i> = .98		0.04, <i>p</i> = .89	
<i>R</i> <sup>2</sup> ADHD	.53		.56		.54		.54		.56	
<i>R</i> <sup>2</sup> Inhibitory control			.07		.12				.12	
$\chi^2_{diff}$ (test of moderation by child sex)	$\chi^2(3) = 4.03, p = .26$	.26	$\chi^2(6) = 5.79, p = .45$	.45	$\chi^2(4) = 0.37, p = .98^a$	.98 <sup>a</sup>	$\chi^2(5) = 4.79, p = .44$	.44	$\chi^2(8) = 5.93, p = .66$	.66

Mother ethnicity (Caucasian vs. non-Caucasian) modeled as an auxiliary variable. Anger reactivity covaried with father ethnicity. MCB and interaction covaried with father ethnicity and mother education, and behavioral problems covaried with child sex. Anger reactivity, MCB, and their interaction were free to covary. Demographic covariates were free to covary. CFI, Comparative Fit Index (adjusted for auxiliary variable); *df*, degrees of freedom; MCB, maternal caregiving behaviors; RMSEA, Root Mean Square of Approximation.

<sup>a</sup>Chi-square difference test conducted without the auxiliary variable because models would not converge with the auxiliary variable.

† *p* < .10; \* *p* < .05; \*\* *p* < .01; \*\*\* *p* < .001.

**Table 3** Indirect effect of infant anger reactivity on childhood ADHD symptoms through 5-year inhibitory control as a function of quality of maternal caregiving behaviors (MCB)

Quality of maternal caregiving behaviors	<i>b</i>	<i>SE</i>	95% CI
Lower MCB quality (−1 <i>SD</i> )	.03 <sup>a</sup>	0.02	0.001, 0.07
Mean MCB quality	.01	0.01	−0.008, 0.04
Higher MCB quality (+1 <i>SD</i> )	−.03	0.02	−0.05, 0.01

<sup>a</sup>*p* = .07 using the Delta method, 95% CI = 95% Monte Carlo confidence intervals.

at average or higher-quality MCB (i.e. +1 *SD*). Consistent with our moderated mediation hypothesis, inhibitory control mediated the relation between infant anger reactivity and childhood ADHD symptoms, but only among children exposed to lower-quality MCB in infancy.

### Sensitivity analyses

**Missing data.** Maximum-likelihood estimation is the recommended procedure for handling missing data under conditions of missing at random (Enders, 2010). As previously discussed, there were missing data in our original sample of 291 participants and we used this procedure to account for these missing data in our analyses. However, in longitudinal mediation analyses it is common practice to use listwise deletion to exclude cases with missing data within the mediation pathway (Zhang & Wang, 2013). To be consistent with these practices, we re-ran our analyses using a sample of participants excluding those with missing data for at least two of three values in mediation pathway (anger reactivity, inhibitory control, ADHD symptoms (included if valid data for either parent or teacher report)). This exclusion resulted in a sample of 207 participants. The results of these analyses were consistent with results from our analyses using the original sample of 291 participants and are reported in the Supporting Information (Tables S1 and S2).

**Parent report of ADHD as outcome.** We chose to measure ADHD using a latent variable of parent and teacher report to maximize use of available data, both to remain consistent with our prior research (Miller et al., 2019) and because multi-informant assessment of ADHD is recommended (Pelham, Fabiano, & Massetti, 2005). However, these measures were taken at different assessment periods and teacher report was only collected for a subsample of participants. Therefore, to ease interpretation of our results, we re-ran our analyses using parent report of ADHD only (measured using the mean of inattention and hyperactivity/impulsivity subscale scores). Results were consistent with analytic models using the latent variable. In the moderated mediation model using parent report only, indirect effects of anger reactivity through inhibitory control were

conditional on MCB quality (interaction indirect effect:  $b = -.01$ ,  $SE = 0.006$ ,  $p = .04$ , 95% CI [−0.03, −0.001]). Follow-up analyses indicated that this indirect effect was significant at lower-quality MCB ( $b = .03$ ,  $SE = 0.02$ ,  $p = .06$ , 95% CI [0.003, 0.07]), but not at higher-quality MCB ( $b = -.02$ ,  $SE = 0.01$ ,  $p = .29$ , 95% CI [−0.05, 0.01]).

### Discussion

Growing evidence suggests anger reactivity as an early life predictor of childhood ADHD symptoms, yet little is known about how infant anger reactivity translates into childhood symptoms. The goal of this study was to elucidate this pathway by examining the *mediating* role of 5-year inhibitory control on the relation between 9-month anger reactivity and childhood ADHD symptoms, as well as the *moderating* role of early caregiving (at 9 months) on these reactivity effects. Study findings supported a moderated mediation model in which the indirect effects of anger reactivity on ADHD symptoms were a function of MCB quality: Inhibitory control mediated the relation between anger reactivity and ADHD symptoms when children experienced lower-quality MCB. These findings were robust and we found the same effects across our sensitivity analyses.

Our findings indicated that both 9-month anger reactivity and 5-year inhibitory control were predictive of childhood ADHD symptoms (at ages 7–9 years). Contrary to expectation, anger reactivity was not related to inhibitory control (i.e. the *a* path), and inhibitory control was not a mediator of the relation between anger reactivity and ADHD symptoms in the simple mediation model. These findings were not consistent with Rabinovitz et al.'s (2016) mediation results and, given the methodological similarities across our studies, suggest developmental differences (i.e. age when anger reactivity measured) may account for these divergent findings. In particular, these results suggest the processes linking anger reactivity to inhibitory control become stronger or more stable with increasing age – or conversely, may be more mutable at younger ages. Results from our moderated mediation analyses also demonstrate the mutability of anger reactivity effects in infancy.

Within the moderated mediation model, our hypothesis that the effects of anger reactivity on ADHD symptoms would be mediated by inhibitory control was supported; however, the significance of this pathway was conditional on maternal caregiving quality at 9 months. At lower-quality caregiving, inhibitory control significantly mediated the relation between infant anger reactivity and childhood ADHD symptoms. However, at average and higher-quality maternal caregiving, there was no mediation of anger reactivity. These differential indirect effects were due to a moderating effect of maternal caregiving on the relation between infant anger reactivity and 5-year



inhibitory control (i.e. the strength of the  $a$  path depended on maternal caregiving quality). In support of the hypothesis that higher levels of temperamental anger reactivity would negatively impact the development of inhibitory control, anger reactivity predicted lower levels of inhibitory control – but only in the context of lower-quality caregiving. These results, consistent with past studies (e.g. Poehlmann et al., 2012), suggest that higher-quality caregiving can buffer against the risk that higher levels of anger reactivity pose for the development of poor inhibitory control. For instance, sensitive, attuned parenting can provide a stable, calming source of external regulation to a highly reactive infant, thereby allowing opportunities for the infant to develop self-regulation when s/he might otherwise be too overwhelmed by negative emotion (Gilliom, Shaw, Beck, Schonberg, & Lukon, 2002).

Our hypothesis that maternal caregiving would moderate the relation between infant anger reactivity and childhood ADHD symptoms was not supported. Instead, anger reactivity predicted higher levels of ADHD symptoms, and this relation was not moderated by caregiving. These results are in contrast to our previous work finding caregiving quality moderated the effects of 4-month-old's reactivity to novelty (motor activity and positive affect) on childhood ADHD symptoms (Miller et al., 2019). Conversely, the current study findings suggest that anger reactivity is a risk factor for ADHD symptoms that is not directly mutable by early caregiving. Together, these results highlight the heterogeneity of temperament-related pathways for ADHD and one way in which surgent (i.e. higher motor activity and positive affect) and anger reactivity pathways to ADHD may differ (Karalunas et al., 2014).

It is curious that anger reactivity effects on 5-year inhibitory control were mutable by early caregiving, yet anger reactivity effects on 7–9 year ADHD symptoms were not. These seemingly discordant results fit well with Halperin and Schulz's (2006) recovery model of ADHD and may provide additional evidence for understanding ADHD pathogenesis via anger reactivity. They propose that ADHD symptoms are due to etiological factors that manifest early in life and remain static throughout the lifespan, but that ADHD symptom severity can be remediated through development of executive functions (e.g. regulatory strategies develop to compensate for the effects of reactivity). Our results demonstrate both the static effect of anger reactivity on ADHD symptoms and the development of a protective pathway through the transactional effects of caregiving and reactivity on the development of inhibitory control.

Strengths of this study include its 9-year prospective longitudinal design beginning in infancy with independent measures of all key variables allowing for a robust test of the mediation and moderated mediation pathways. Furthermore, we were able to identify unique variance associated with ADHD

symptoms by controlling for childhood behavioral problems (i.e. oppositional behaviors and conduct problems). Generalizability of our results is a potential limitation because the majority of parents in the sample were well-educated and Caucasian. Parent and teacher report of ADHD symptoms were collected at different assessment periods and teacher report data were collected for a subsample of participants. Finally, our longitudinal study did not collect data on parental or familial ADHD symptoms or diagnoses; therefore, we were unable to control for shared genetic liability in our analyses. Given that maternal ADHD symptoms are associated with less sensitive and more intrusive caregiving behaviors (Park, Hudec, & Johnston, 2017), it is possible that the moderating effect of maternal caregiving on anger reactivity-to-inhibitory control pathway was a proxy for the moderating effects of the child's genetic liability for ADHD. Future research is needed to clarify the unique, and likely interactive, contributions of parenting versus familial risk on ADHD pathogenesis.

## Conclusion

This study examined one potential pathway for the development of childhood ADHD symptoms beginning in infancy. Our results converge with previous studies and suggest that infant anger reactivity is an early indicator of risk for ADHD. In addition to the direct risk anger reactivity poses for the development of ADHD, anger reactivity, when combined with lower-quality caregiving, may undermine the development of a potential protective pathway against ADHD by negatively impacting inhibitory control. Taken together, these findings underscore the importance of the transactional relations between infant and parent for understanding ADHD pathogenesis (Johnston & Chronis-Tuscano, 2015) and point to the potential of early parenting interventions, especially among anger-prone infants, for mitigating risk for ADHD in childhood.

## Supporting information

Additional supporting information may be found online in the Supporting Information section at the end of the article:

**Table S1.** Summary of results from the analytic models ( $n = 207$ ).

**Table S2.** Indirect effect of infant anger reactivity on childhood ADHD symptoms through 5-year inhibitory control as a function of quality of maternal caregiving behaviors (MCB) ( $n = 207$ ).

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## Key points

- Understanding the developmental pathways for ADHD is crucial for identifying potential points of early intervention with most clinical impact.
- We find infant anger reactivity and poor inhibitory control at age 5 predicted ADHD symptom severity at age 7–9.
- This is the first study to demonstrate an indirect effect of infant anger reactivity on ADHD symptoms through inhibitory control, but only among children exposed to lower-quality caregiving in infancy.
- Higher-quality early caregiving may buffer reactivity-related risk for ADHD by disrupting the negative impact of anger reactivity on the development of inhibitory control.
- Anger reactivity may be a useful early indicator of ADHD risk. Interventions targeting very early parenting could help prevent development of ADHD symptoms via supporting better inhibitory control in anger-prone infants.

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