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Paternal antisociality and growth in child delinquent behaviors: Moderating effects of child sex and respiratory sinus arrhythmia

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Abstract

Children of fathers with antisocial personality disorder (ASPD) are at risk for developing delinquency, and both biological and environmental mechanisms contribute. In this study, we test parasympathetic nervous system (PNS) function as a vulnerability/sensitivity attribute in predicting intergenerational associations between fathers' antisociality and children's delinquency scores. We followed 207 children (ages 8–12 years at intake; 139 boys) across three annual assessments. Fathers' antisociality was measured via maternal reports on the Family Interview for Genetic Studies (FIGS). At Year 1, children's resting respiratory sinus arrhythmia (RSA) was measured. At Years 1, 2, and 3, child delinquent behaviors were assessed using the delinquency subscale of the Youth Self-Report. At age 8, boys' delinquency scores were associated weakly with paternal antisocial behaviors. However, boys' delinquency scores increased steeply thereafter specifically for those who had fathers with higher antisocial symptoms. In addition, associations between delinquency and paternal antisociality were largest for boys with higher resting RSA. For girls, growth in delinquency was unrelated to both father antisociality and resting RSA. These findings (a) suggest moderating effects of children's age, sex, and PNS function on associations between father antisocial behavior and offspring delinquency; and (b) provide insights into differential vulnerability among children of fathers with ASPD.

KEYWORDS

child delinquent behaviors, child resting RSA, developmental trajectory, father antisocial behaviors

1 | INTRODUCTION

During the past decade, approximately 1 in every 100 youth ages 12–17 years in the U.S. was involved in at least one serious violent crime (Bureau of Justice Statistics, 2017). In 2017 alone, 215, 000 serious violent crimes (14.2% of all serious violent crimes) were committed by youth in this age range (Bureau of Justice Statistics, 2017). Although many antisocial behaviors are committed by a small subgroup of early-starting offenders, the proportion of new criminal offenders grows quickly from childhood to adolescence (Moffitt, 1993). Based on this timing of onset, two subtypes of conduct disorder (CD) are recognized: childhood-onset type, in which symptoms emerge before age 10, and adolescent-onset type, in which symptoms emerge thereafter (American Psychiatric Association, 2013). Whereas some degree of adolescent-onset conduct disturbance is age-normative, childhood-onset CD often portends more severe externalizing behaviors across the lifespan, including delinquency, criminality, and substance use problems (see Beauchaine et al., 2009, 2017; Moffitt, 1993). Given the high costs of childhood-onset conduct problems (CPs) to individuals, local communities, and society,

there is an urgent need for better understanding of associated risk, protective, and resiliency factors.

In this study, we take a multiple-levels-of-analysis approach to understanding child CPs by considering biological vulnerability and environmental risk—particularly family risk (see also Beauchaine & Hinshaw, 2016), consistent with the developmental psychopathology perspective (e.g., Beauchaine & Gatzke-Kopp, 2012; Hinshaw, 2018). We extend our previous research on child CPs by examining interactions between (a) fathers' antisocial behaviors, a potent risk factor for children's externalizing progression (e.g., Hicks et al., 2004; Kopp & Beauchaine, 2007), and (b) parasympathetic nervous system (PNS) function, an established neurobiological vulnerability to externalizing behavior (e.g., Beauchaine et al., 2007; El-Sheikh et al., 2009).

1.1 | Intergenerational transmission of antisocial and delinquent behavior

1.1.1 | Heritable influences and gene–environment correlation

Children inherit many predispositions from their parents, including temperamental attributes such as impulsivity and negative affectivity, which can confer vulnerability to delinquency—particularly in contexts of environmental risk (see e.g., Beauchaine et al., 2017; Meier et al., 2008). Early in life, environmental risk is concentrated within families, who serve as children's first and primary socialization agents. Genetic vulnerabilities and family adversity therefore often overlap to increase risk, a phenomenon known as passive gene–environment correlation (see Plomin et al., 1977; Rutter, 2015). Disentangling the relative contributions of heritable and environmental influences is therefore impossible without complex twin studies. Thankfully, such studies have been conducted and demonstrate *both* heritability effects and rearing environment effects on child and adolescent delinquency (e.g., Boisvert et al., 2012; Burt et al., 2008; Hicks et al., 2004; Taylor et al., 2000). In this paper, we evaluate associations between fathers' histories of antisocial behavior and children's delinquency scores, including moderating effects of children's sex and resting state PNS function. First, however, we discuss characteristics of families with a father with antisocial traits.

1.1.2 | Effects of paternal antisociality on family function

Historically, fathers served largely as breadwinners and moral teachers for their children (Pleck & Pleck, 1997). Over the past 50 years, however, father roles have changed. In most U.S. subcultures, fathers are expected to coparent and therefore share childcare responsibilities with mothers. Thus, father involvement with their children has increased for many families (Cabrera et al., 2000; Pleck & Masciadrelli, 2004). A growing literature documents the important roles that fathers play in child development. For example, greater father involvement predicts better social and cognitive development (Lee & Schoppe-Sullivan, 2017; Sarkadi et al., 2008; Tamis- LeMonda et al., 2013; Yan et al., 2018). In contrast, poor father–child relationship quality portends child behavior problems and worse mental health among children of varying ages (Branje et al., 2010; Yan et al., 2019). Developmental psychologists, child clinical psychologists, community practitioners, and the public are now aware of the critical role that fathers play—for better or worse—in children's socioemotional development (Cabrera et al., 2014; Lamb & Lewis, 2010; Pleck, 2010; Yan et al., 2019).

Many family characteristics associated with child CPs are concentrated in households with a father with antisocial personality disorder (ASPD). Examples include psychopathology more broadly (Ellis & Hoskin, 2018), marital conflict (El-Sheikh et al., 2011), family instability (Fomby & Osborne, 2017), and ineffective parenting, such as coercion, lack of support, authoritarian control, and inconsistent discipline (Chung & Steinberg, 2006; Hoeve et al., 2009; Patterson et al., 2000; Simons et al., 2007). In studies that compare mother and father influences, fathers' behaviors yield larger effect sizes in explaining both current and future offspring delinquency (Loukas et al., 2001; Thornberry et al., 2003). Ironically, however, variability in father influences on family function are less understood (Hoeve et al., 2009). Given their low participation rates, fathers with antisocial traits are underrepresented in developmental psychopathology research (Parent et al., 2017; Phares et al., 2005).

Several studies describe fathering correlates of child delinquency. Fathers' but not mothers' alcohol use disorder is associated with both violent and non-violent offspring delinquency (Grekin et al., 2005). In contrast, mother and father involvement are associated with lower rates of delinquency (Cookston & Finlay, 2006). Additionally, father–child trust and communication are associated with fewer delinquent behaviors among low-income youth (Yoder et al., 2016). Children's perceptions of their father's knowledge of children's whereabouts and high-quality father–child relationships are associated with lower levels of delinquency among adolescents (Walters, 2019). In contrast, fathers' absence predicts higher levels of adolescent delinquency (Markowitz & Ryan, 2016). Non-resident fathers' involvement is associated with less delinquency among low-income, primarily minority adolescents (Coley & Medeiros, 2007).

1.1.3 | Moderating factors and biological vulnerability hypotheses

A growing literature on relations between fathers' parenting and children's socioemotional development identifies child temperament and other biologically based individual differences as moderators of relations between fathers' parenting and children's behavioral adjustment. For example, infant reactivity and sex moderate relations between father involvement in parenting and children's socioemotional adjustment (Ramchandani et al., 2010). Similarly, children's skin conductance—a biomarker of

trait anxiety—moderates associations between parenting quality and child externalizing behavior (Kochanska et al., 2015). Among children with fathers with antisocial traits, high skin conductance confers protection from child delinquency (Shannon et al., 2007). Given the limited number of such studies, additional research is warranted.

The studies discussed immediately above notwithstanding, most research conducted to date evaluates bivariate associations between fathering attributes and child delinquency, without considering mediators or moderators of child outcomes and without considering longitudinal growth in children's delinquent behaviors. Child sex, age, and physiological regulation are all potential moderators of associations between family risk and child delinquency (Beauchaine, 2001; Beauchaine et al., 2007, 2017; Hoeve et al., 2009; Kopp & Beauchaine, 2007; Pang & Beauchaine, 2013; Shannon et al., 2007; Tyrell et al., 2019; Yan et al., 2017). In this study, we evaluate each.

Child sex

Sex differences in antisociality are among the largest effects seen in psychological research (Eme, 2016). Links between children's externalizing behaviors and PNS function are moderated by sex, with stronger associations for boys (Beauchaine et al., 2008; El-Sheikh et al., 2011). Such findings may suggest different mechanisms underlying externalizing behavior by sex. Other evidence suggests that boys' externalizing behaviors are associated more strongly with fathers' un-involvement, harsh parenting, and physical punishment (Gryczkowski et al., 2010; Kerr et al., 2004). We therefore evaluate moderating effects of sex.

Child age

We also examine child age as a likely moderator of growth in delinquency given clear developmental changes in CPs and across middle childhood and adolescence (see above; Abar et al., 2014; Hoeve et al., 2008; Keijsers et al., 2012; Moffitt, 1993). In early childhood, opportunities to engage in delinquent behaviors are limited (Richers & Cicchetti, 1993). As such opportunities increase in later childhood, associations between parenting quality and child delinquency may strengthen (Hoeve et al., 2009). One contributing factor may be decreases in father–child closeness that emerge during middle childhood (Yan et al., 2018).

Parasympathetic nervous system function

Respiratory sinus arrhythmia (RSA) captures the high-frequency component of heart rate variability associated with breathing (see Berntson et al., 1993; Shader et al., 2018). Under certain stimulus conditions, RSA indexes PNS function (Berntson et al., 1993, 1997; Zisner & Beauchaine, 2016). Resting RSA correlates consistently with self-regulation, socioemotional adjustment, and social engagement (Beauchaine, 2001, 2015; Calkins et al., 2007; Patriquin et al., 2013). Resting RSA is also associated with executive function and effortful control among typically developing preschoolers, suggesting stronger self-regulation capacity (Marcovitch et al., 2010; Taylor et al., 2015). In contrast, low resting RSA is associated with both externalizing and internalizing psychopathology (Beauchaine, 2015; Beauchaine et al., 2001, 2007; Calkins & Dedmon, 2000; Shader et al., 2018). Although RSA is shaped by environment early in life (Beauchaine et al., 2007; Bell et al., 2018), it may take on trait-like qualities in later childhood and mark biological sensitivity to environmental influences on socioemotional function (Obradović, 2012). Biological sensitivity refers to situations in which those with certain traits—in this case low PNS tone—are vulnerable in high-risk environments but thrive in enriched environments (Baião et al., 2020; Belsky & Pluess, 2009).

Evidence for the biological sensitivity to context hypothesis is mixed, and may depend on both sex and severity of externalizing behavior (e.g., Shader et al., 2018; Zisner & Beauchaine, 2016). Among samples comprised of mostly male children, adolescents, and adults with clinical levels of CPs, aggression, and related behaviors, low resting RSA is typically observed (see immediately above). Thus, RSA appears to mark poor self- and emotion regulation (e.g., Beauchaine et al., 2001, 2007; Mezzacappa et al., 1997; Pang & Beauchaine, 2013; Rukmani et al., 2016). In an era of non-replication, such findings stand out for their consistency, and support a vulnerability interpretation. However, low resting RSA may not characterize girls with clinical levels of delinquency and aggression (Beauchaine et al., 2008), findings that further support the need to evaluate sex effects (see above).

In contrast, studies of normative developmental, community, and high-risk samples—where only fractions of participants score in clinical ranges on externalizing conduct—yield less consistent findings (for recent discussions, see Shader et al., 2018; Zisner & Beauchaine, 2016). Some studies appear to support differential susceptibility (see below), whereas others do not (Wagner et al., 2018; Zhang et al., 2017). Sturge-Apple et al. (2016) found that higher RSA was associated with longer delay of gratification among children from middle-class families, but with shorter delay of gratification among children from environments with more limited resources. Others report susceptibility to a range of environmental risks, including low SES, interparental relationship problems, parenting stress, maternal depression/anxiety, and traumatic events among children with higher resting RSA (Blandon et al., 2008; Davis et al., 2017; Eisenberg et al., 2012; Gray et al., 2017; Mammen et al., 2017; Peltola et al., 2016). In apparent contrast are reports that *lower* resting RSA is associated with susceptibility to parenting insensitivity, harshness, and emotion socialization practices in community samples (Gueron-Sela et al., 2017; Hastings & De, 2008; Hastings et al., 2008; Hinnant et al., 2015). Thus, further studies are needed to determine both (a) whether a differential susceptibility model holds, and (b) which direction of effects is supported. It is scientifically untenable that findings with different directions of effect are being cited as evidence of biological sensitivity to context (see Zisner & Beauchaine, 2016). It should also be noted that RSA shows plasticity across development in response to environmental influences—both positive and negative (e.g., Bell et al., 2018)—and that longitudinal research is therefore needed to disentangle these and related questions.

1.2 | The current study

In this study, we test associations between (a) mother reports of fathers' antisociality and (b) growth in children's self-reported delinquent behaviors across ages 8–15 years in a mixed normative and clinical sample. We also evaluate moderating effects of age, sex, and resting RSA on these associations. Significant growth in delinquency is expected in this age range (Moffitt, 1993), and has been reported previously with this sample (McDonough-Caplan et al., 2018). Consistent with the literature outlined above, we expected growth in delinquency among children to be predicted by histories of paternal antisociality and sex, with stronger effects for boys (see e.g., Hoeve et al., 2009; Moffitt et al., 2001).

Given inconsistencies across studies for tests of moderating effects of children's resting RSA on their externalizing behaviors and few evaluations of sex effects, we do not offer directional hypotheses. Rather, our analyses are intended to clarify a murky literature in which opposite directions of effect have been used as evidence for biological sensitivity to context.

2 | METHOD

2.1 | Participants

Data were drawn from a longitudinal study of CPs, depression (DEP), and heterotypic comorbidity (CMB) among 207 children (139 boys, 65.6%), ages 8–12 years (M = 9.80, SD = 1.53) at the first of three annual assessments. Recruitment included advertisements in local newspapers, community publications, city buses, clinics and community centers, radio spots, and school newsletters. Three clinical groups (CPs, DEP, CMB) and one non-psychiatric control group (CTR) were targeted separately in advertisements. For the clinical groups, advertisements described characteristics of DEP and CPs.

If an interested parent had a child who fit the description of a clinical group, s/he was asked to complete a 30-min computerized, structured phone screen to assess potential child psychopathology. The telephone interview included portions of the Child Symptom Inventory (CSI; Gadow & Sprafkin, 1997) and the Child Behavior Checklist (CBCL; Achenbach & Edelbrock, 1991), two commonly used measures of child psychopathology with good reliability. The CSI provides dimensional scores and diagnostic cutoffs for DSM disorders. Each diagnostic criterion is rated on a 4-point scale (0 = never, 1 = sometimes, 2 = often, 3 = very often). Ratings ≥2 are considered positive for each diagnostic criterion. For purposes of the phone screen, CSI scales included attention-deficit/hyperactivity disorder (ADHD), CD, oppositional defiant disorder (ODD), major depressive disorder (MDD), and dysthymia (DYS). Internal consistencies for these scales range from a (Sprafkin et al., 2002). Administered subscales of the CBCL included aggression, attention problems, and anxiety/depression. Internal consistencies for these scales range from .73 to .84 (Achenbach & Edelbrock, 1991).

Among 445 families who expressed interest, 212 were recruited and placed into one of the four groups based on results from the telephone interview. Among these families, 207 provided usable data. Children were placed into either a control group (n = 69) or one of three clinical groups (CD, n = 30; DEP, n = 28; comorbid, n = 80). Control group participants were excluded if they met criteria for any disorder on the CSI and/or scored $T \geq 70$ on any CBCL scale. Children who were placed into the clinical groups were required to meet CSI criteria for their respective disorder(s). In addition, children in the CD group had to score ≥95th percentile on the CBCL aggression subscale, children in the DEP groups had to score ≥85th percentile on the CBCL anxious/depressed subscale, and children in the comorbid group were required to meet criteria on both the aggression and anxious/depressed subscales. Extensive details regarding recruitment and eligibility appear elsewhere (McDonough-Caplan et al., 2018; Pang & Beauchaine, 2013).

Consistent with sociodemographic data in the metropolitan location, 62.3% of children identified as Caucasian, 11.8% identified as African American, 9.9% identified as Hispanic, and 16.0% identified as other races. Among mothers, 42.0% held bachelor's degrees, 13.7% held advanced degrees, and 2.8% did not finish high school. Among fathers, 35.4% held bachelor's degrees, 11.3% held advanced degrees, and 9.0% did not finish high school. Approximately half of children (52%) lived with their biological father at Year 1 (Y1).

2.2 | Procedures

At Y1, the target child and a parent (204 mothers) completed a lab visit in which extensive interviews and autonomic assessments were conducted. Interviews assessed family function and both parent and child psychopathology, among other outcomes (see McDonough-Caplan et al., 2018; Pang & Beauchaine, 2013). Children's RSA was measured during a 5-min rest period while they sat alone in a stimulus-free room. They were allowed to speak with lab personnel if needed via a microphone (e.g., for comforting), but almost none did. Participating parents reported on antisocial behaviors of the other biological parent using the Family Interview for Genetic Studies (FIGS; Maxwell, 1992). At all three time points (Y1, Y2, and Y3), children reported their delinquent behaviors on the Youth Self-Report (YSR; Achenbach & Rescorla, 2001). Self-reports were used given that parents are unaware of many of their children's delinquent activities and therefore underreport (Laird et al., 2003).

2.3 | Measures

2.3.1 | Baseline respiratory sinus arrhythmia (Year 1)

Electrocardiographic signals were obtained using a HIC 2000 impedance cardiograph. Spot electrodes were placed in a modified Lead II configuration to minimize movement artifacts (Qu et al., 1986). Missing and extra beats were interpolated and removed, respectively,

by trained research assistants. RSA was indexed using autoregressive spectral analysis, which provides more accurate estimates than other spectral-analytic approaches (Shader et al., 2018). To ensure children reached a relaxed state, RSA was computed for the last min of the 3-min baseline. Accurate RSA assessment using spectral analysis requires at least 10 respiratory cycles (Berntson et al., 1997). Over 95% of children and adolescents in this age range breathe at 11 or more cycles per min (see Wallis et al., 2005).

We used Kubios HRV software (version 2.2) to compute RSA (Tarvainen et al., 2014). As a preliminary step, we applied a medium artifact correction and used smoothing priors to decrease distortion of data end points, and to eliminate very low frequency trend components (Tarvainen et al., 2002). Because autoregressive spectral analysis identifies respiratory frequency peaks for each individual empirically, it corrects for developmental changes in respiration from ages 8–15. This contrasts with typical analyses, which use single respiratory cutoffs for all participants regardless of age. Single respiratory cutoffs often overestimate RSA among younger children (Shader et al., 2018). All spectral density functions were inspected visually to ensure that respiratory peaks and their surrounding distributional dispersion were captured.

2.3.2 | Father antisocial behaviors (Year 1)

Fathers' antisocial behaviors were indexed via maternal reports on the FIGS (Maxwell, 1992), a semi-structured interview that measures family history of psychiatric disorders. The FIGS assesses lifetime depression, mania, substance use, and schizophrenia, as well as schizoid, schizotypal, paranoid, and antisocial personality disorders. Only ASPD was measured for this study. When mothers were not the participating parent ($n = 8$), reports of paternal antisocial behaviors were coded as missing. In total, 49 fathers reached diagnostic threshold for ASPD. As expected, group differences were observed in both ASPD symptom severity and ASPD diagnoses (see Kopp & Beauchaine, 2007 for details). Children with CPs (CMB) had (a) fathers with higher symptoms of ASPD ($M = 2.35$, $SD = 2.8$) than other children ($M = 1.4$, $SD = 2.55$), $t(205) = 2.54$, $p = .01$, and (b) fathers with more diagnoses of ASPD than expected by chance ($n = 33$ versus. $n = 16$), $\chi^2(3) = 9.6$, $p = .037$.

2.3.3 | Child delinquent behaviors (Years 1, 2, and 3)

Children's delinquent behaviors were measured using the delinquency subscale of the YSR (Achenbach & Rescorla, 2001), which includes 11 items (e.g., "I don't feel guilty after doing something I shouldn't", "I lie or cheat", "I hang around with kids who get in trouble"). Internal consistency and concurrent validity of YSR among younger youth are established by previous research (e.g., Ebesutani et al., 2011). Cronbach's alphas do not differ for younger versus older youth (.70 and .78, respectively). In addition, younger youths' reports on the DSM-oriented externalizing scales of the YSR correspond with DSM diagnoses and parent reports at a comparable level with older youth (Ebesutani et al., 2011).

2.4 | Analytic plan

First, we examined descriptive statistics and quantified missing data. Of the 207 participants at Y1, 178 (86%) returned at Y2, and 159 (76%) returned at Y3. Missing data were imputed using the *Mice* package in R, with 50 imputations, which were pooled using Rubin's rule (Buuren & Groothuis-Oudshoorn, 2010). We specify complete degrees of freedom ($df = 178$) for pooled results using the *testEstimates()* function in the *mitml* package (Grund et al., 2019). This adjusts degrees of freedom for smaller samples using a Bayesian paradigm (Barnard & Rubin, 1999). Multilevel models (MLMs) with restricted maximum likelihood (REML) estimation were performed in the *lme4* package in R to simultaneously examine both intra- and interindividual changes in children's delinquent behaviors over time.

First, we fit an unconditional model to examine initial levels of and growth in child delinquent behaviors (Model 1). Intercepts and slopes were modeled as random effects and therefore varied freely. As a result, covariance of intercepts and slopes also varied freely. Second, we added (a) *Child Age* (grand mean-centered) × *Father Antisocial* (grand mean-centered) × *Child Sex* interaction terms, and (b) a *Child RSA* (grand mean-centered) × *Father Antisocial* (grand mean-centered) × *Child Sex* interaction terms to examine whether and to what extent age and resting RSA moderate associations between fathers' antisocial behaviors and trajectories of child delinquent behaviors, differentially by sex (Model 2). We recognize that power to detect three-way interactions, particularly slope effects, is a concern with the sample size (see Heo & Leon, 2010). However, given theoretical justification for all terms, we include them in our models. Variables were grand mean-centered to alleviate possible collinearity of interactions. For significant continuous moderators (child age and RSA), follow-up tests of significance were conducted at 1.0 *SD* below and 1.0 *SD* above the grand mean.

Finally, we modeled race, education, income, and fathers' residence status at study entry to determine whether findings from Model 2 differed in whole or in part as a result of demographic influences (Model 3).[1] Race was dummy-coded with Caucasian as the reference group. MLM equations were as follows:

2.4.1 | Level 1

$$\text{Child Delinquency} = \beta_{0i} + \beta_{1i} \times t$$

2.4.2 | Level 2 (Model 1)

$$\beta_{0i} = \gamma_{00} + \mathrm{u}_{0i}$$

TABLE 1 Descriptive statistics, missing rates, and bivariate correlations among study variables and continuous covariates

Variables	*M*	*SD*	Min.	Max.	Missing rate (%)	1	2	3	4	5	6	7	8	9	10
1. Child sex (1 = girls, 0 = boys)	0.34	0.48	0	1	0										
2. Child age (years)	9.80	1.53	7	12	0	−.12									
3. Household income (thousands)	50.12	42.64	0	440	0	−.02	.00								
4. Mother education (years)	14.84	1.98	8	20	0	.24[b]	−.09	.27[b]							
5. Father education (years)	14.01	2.54	8	20	0	.18[b]	−.14[a]	.32[b]	.42[b]						
6. Child resting RSA Y1 (log(beats/min^2/Hz))	7.06	1.10	4.52	10.12	16	.06	−.02	−.02	.09	.07					
7. Father residency (1 = resident)	0.52	0.50	0	1	0	−.02	−.06	.31[b]	.16[a]	.32[b]	−.00				
8. Father antisocial behaviors (FIGS) Y1	1.39	2.16	0	6	3	.02	.06	−.30[b]	−.14	−.32[b]	−.01	−.44[b]			
9. CBCL child delinquency score Y1	52.62	4.99	50	75	1	−.16[a]	.11	−.06	−.12	−.19[b]	−.03	−.09	.24[b]		
10. CBCL child delinquency score Y2	52.48	4.95	50	86	16	−.04	.11	−.17[a]	−.04	−.19[b]	.07	−.13	.21[b]	.28[b]	
11. CBCL child delinquency score Y3	53.17	5.72	50	88	25	−.06	.15	−.17[a]	−.12	−.22[b]	−.01	−.14	.24[b]	.29[b]	.44[b]

FIGS, Family Interview for Genetic Studies; CBCL, Child Behavior Checklist. Child delinquency is reported as *T*-scores for ease of interpretation. Raw scores, however, were used in analyses.

[a]$p < .05$

[b]$p < .01$.

$$\beta_{1i} = \gamma_{10} + u_{1i}$$

2.4.3 | Level 2 (Model 2)

$$\beta_{0i} = \gamma_{00} + \gamma_{01} \times \text{Child Age}_i \times \text{Father Antisocial}_i \times \text{Child Sex}_i + \gamma_{02} \times \text{Child Resting RSA}_i \times \text{Father Antisocial}_i \times \text{Child Sex}_i + \text{second-order interaction terms} + u_{0i}$$

$$\beta_{1i} = \gamma_{10} + \gamma_{11} \times \text{Child Age}_i \times \text{Father Antisocial}_i \times \text{Child Sex}_i + \gamma_{12} \times \text{Child Resting RSA}_i \times \text{Father Antisocial}_i \times \text{Child Sex}_i + \text{second-order interaction terms} + u_{1i}$$

2.4.4 | Level 2 (Model 3)

$$\beta_{0i} = \gamma_{00} + (\text{Model 2 predictors}) + \gamma_{04} \times \text{Race(African American)}_i + \gamma_{05} \times \text{Race(Hispanic)}_i + \gamma_{06} \times \text{Race(Other)}_i + \gamma_{07} \times \text{Mother Educ.}_i + \gamma_{08} \times \text{Father Educ.}_i + \gamma_{09} \text{Household Income}_i + \gamma_{010} \times \text{Father Residency}_i + u_{01}$$

$$\beta_{1i} = \gamma_{10} + (\text{Model 2 predictors}) + \gamma_{14} \times \text{Race(African American)}_i + \gamma_{15} \times \text{Race(Hispanic)}_i + \gamma_{16} \times \text{Race(Other)}_i + \gamma_{17} \times \text{Mother Educ.}_i + \gamma_{18} \times \text{Father Educ.}_i + \gamma_{19} \text{Household Income}_i + \gamma_{10} \times \text{Father Residency}_i + u_{11}$$

3 | RESULTS

3.1 | Preliminary analyses

Table 1 presents means, *SD*s, minimum, maximum, missing data rates, and zero-order correlations for all variables. Tables 2 and 3 provide descriptive statistics and correlation matrices, respectively, separately by child sex. Little's missing completely at random (MCAR) test did not reject the null hypothesis of MCAR, $\chi^2(126) = 150.77$, $p = .07$. As noted above, missing data were imputed. As expected, children's delinquency scores were (a) autocorrelated across Years 1–3 (all *r*s ≥ .28, all *p*s < .01), and (b) correlated with fathers' antisocial behaviors (measured at Y1) across Years 1–3 (all *r*s ≥ .21, all *p*s < .01).

3.2 | Unconditional growth model

The intercept of the unconditional growth model (Model 1) was significant, $SE = 0.23$, $t = 8.06$, $p < .001$, indicating an average initial delinquency score of 1.86. Delinquent behaviors, on average, also increased over time, as indicated by a significant unconditional slope, $SE = 0.11$, $t = 2.11$, $p = .035$. On average, YSR delinquency scores increased 0.23 points per year. The unconditional growth model fit better than an intercept-only model, $\chi^2(2) = 7.32$, $p = .026$. Significant variation in both intercepts, $p < .001$, and slopes, $p = .026$, was observed, indicating variance left to be explained.

TABLE 2 Descriptive statistics and missing rates for study variables and continuous covariates by child sex

Child Sex	Girls					Boys				
Variables	*M*	*SD*	Min.	Max.	Missing rate (%)	*M*	*SD*	Min.	Max.	Missing rate (%)
Child age (years)	9.55	1.52	7	12	0	9.93	1.53	8	12	0
Household income (thousands)	48.85	34.01	0	160	0	50.79	46.67	0	440	1
Mother education (years)	15.51	1.91	12	20	0	14.49	1.94	8	20	0
Father education (years)	13.63	2.60	8	20	0	13.68	2.46	8	20	0
Child resting RSA Y1 (log(beats/min^2/Hz))	7.15	1.06	4.52	9.82	11	7.01	1.12	4.56	10.12	19
Father residency (1 = resident)	0.51	0.50	0	1	0	0.53	0.50	0	1	0
FIGS father antisocial behaviors Y1	1.44	2.19	0	6	0	1.36	2.15	0	6	4
CBCL child delinquency score Y1	51.54	3.82	50	67	1	53.19	5.43	50	75	1
CBCL Child Delinquency Score Y2	52.25	5.32	50	86	8	52.61	4.74	50	70	20
CBCL child delinquency score Y3	52.76	5.02	50	69	19	53.41	6.11	50	88	29

FIGS, Family Interview for Genetic Studies; CBCL, Child Behavior Checklist. Child delinquency is reported as *T*-scores for ease of interpretation. Raw scores, however, were used in analyses.

[a] $p < .05$.

[b] $p < .01$.

TABLE 3 Bivariate correlations among study variables and continuous covariates by child sex for boys (above diagonal) and girls (below diagonal)

Variables	1	2	3	4	5	6	7	8	9	10
1. Child age (years)		.00	−.06	−.11	−.03	−.14	.12	.10	.13	.12
2. Household income (thousands)	−.02		.17[a]	.25[b]	.10	.21[a]	−.25[b]	−.03	−.19[a]	−.13
3. Mother education (years)	−.09	.59[b]		.35[b]	.13	.06	−.01	−.02	−.05	−.01
4. Father education (years)	−.13	.55[b]	.49[b]		.12	.27[b]	−.32[b]	−.14	−.23[a]	−.14
5. Child resting RSA Y1 (log(beats/min2/Hz))	.01	−.24	−.01	−.04		.07	−.14	−.04	.09	−.01
6. Father residency (1 = resident)	.07	.59[b]	.38[b]	.45[b]	−.13		−.37[b]	−.03	−.13	−.15
7. FIGS Father antisocial behaviors Y1	−.04	−.44[b]	−.39[b]	−.34[b]	.25[a]	−.56[b]		.19[a]	.21[a]	.28[b]
8. CBCL Child delinquency score Y1	.06	−.19	−.28[a]	−.26[a]	.05	−.30[a]	.38[b]		.32[b]	.31[b]
9. CBCL Child delinquency score Y2	.09	−.13	−.02	−.12	.04	−.14	.21	.21		.42[b]
10. CBCL Child delinquency score Y3	.20	−.24	−.32[a]	−.36[b]	.01	−.12	.21	.24	.52[b]	

FIGS, Family Interview for Genetic Studies; CBCL, Child Behavior Checklist. Child Delinquent Behaviors are reported as *T*-scores for ease of interpretation. Raw scores, however, were used in the analyses.

[a]$p < .05$.

[b]$p < .01$.

TABLE 4 Hierarchical linear regression coefficients for associations between father antisocial behaviors and slopes and intercepts of child delinquent behaviors, moderated by child sex, child age, and resting RSA (Model 2)

	Intercept of delinquent behaviors					Slope of delinquent behaviors				
	B	*SE*	*t*	*df*	*p*	*B*	*SE*	*t*	*df*	*p*
Intercept	2.424	0.188	12.922	167.1	<.001	0.231	0.138	1.677	131.7	.096
Father antisocial behaviors Y1	0.192	0.209	0.917	148.3	.361	0.275	0.152	1.809	122.8	.073
Child age	0.145	0.122	1.188	169.0	.237	0.007	0.090	0.078	131.7	.938
Child sex (1 = girls, 0 = boys)	−1.117	0.321	−3.473	165.7	.001	0.177	0.235	0.752	132.9	.454
Child resting RSA Y1	−0.031	0.209	−0.148	108.0	.882	0.165	0.142	1.161	120.8	.248
Age × Antisocial	0.374	0.128	2.926	161.5	.004	−0.224	0.093	−2.396	131.5	.018
Child sex × Antisocial	0.223	0.341	0.656	159.8	.513	−0.185	0.241	−0.766	146.9	.445
Child sex × Child Age	−0.059	0.205	−0.285	168.5	.776	0.029	0.151	0.192	132.4	.848
RSA × Antisocial	−0.056	0.239	−0.236	106.7	.814	0.330	0.167	1.971	110.4	.051
Child SEX × RSA	0.096	0.340	0.281	137.8	.779	−0.226	0.241	−0.935	132.1	.351
Child sex × Child age × Antisocial	−0.386	0.205	−1.887	169.5	.061	0.251	0.147	1.703	146.1	.091
Child sex × Resting RSA × Antisocial	0.221	0.349	0.635	127.4	.527	−0.509	0.246	−2.068	131.3	.041

3.3 | Conditional growth models

Coefficients for Model 2 appear in Table 4. Model 2 explained 25.54% of the variance in intercepts and 17.48% of the variance in slopes of child delinquent behaviors.

3.4 | Father antisocial behaviors and child age at study entry

For both intercepts, $B_{\text{Child Sex} \times \text{Child Age} \times \text{Father Antisocial}} = -0.39$, $SE = 0.21$, $t = -1.89$, $p = .061$, and slopes, $B_{\text{Child Sex} \times \text{Child Age} \times \text{Father Antisocial}} = 0.25$, $SE = 0.15$, $t = 1.70$, $p = .091$, in child delinquency scores, the Child Sex × Child Age × Father Antisocial three-way interactions approached significance. Ordinarily, we would not decompose/interpret marginal effects. However, we do so here given substantial literature showing stronger effects of paternal antisociality on boys' delinquency (as reviewed above), and limited power to detect sex effects.

For boys, age at study entry moderated effects of fathers' antisociality on both initial levels of intercepts, $B_{\text{Child Age} \times \text{Father Antisocial}} = 0.37$, $SE = 0.13$, $t = 2.93$, $p = .004$, and increases in slopes of, $B_{\text{Child Age} \times \text{Father Antisocial}} = -0.22$, $SE = 0.09$, $t = -2.40$, $p = .018$, children's delinquent behaviors increased over time. For girls, effects of fathers' antisociality on both intercepts, $B_{\text{Child Age} \times \text{Father Antisocial}} = -0.01$, $SE = 0.16$, $t = -0.087$, $p = .940$, and slopes, $B_{\text{Child Age} \times \text{Father Antisocial}} = 0.03$, $SE = 0.11$, $t = 0.24$, $p = .813$, of delinquent behaviors were unrelated to age at study entry. Thus, moderating effects of age at study entry on relations between fathers' antisociality and initial rates of and growth in delinquency were restricted to boys.

We followed-up this finding by examining effects of fathers' antisociality on growth in delinquency specifically for boys, conditioned on age at study entry. For this analysis, we evaluated intercept and slope effects for boys at −1.0 *SD* (~8.5 years) and +1.0 *SD* (~11.5 years) of mean age at study entry. Fathers' antisocial behaviors predicted initial levels of delinquency for older boys (intercepts), $B_{\text{Father Antisocial}} = 0.75$, $SE = 0.24$, $t = 3.11$, $p = .002$, but not younger boys, $B_{\text{Father Antisocial}} = -0.74$, $SE = 0.43$, $t = -1.74$, $p = .082$. Slope effects indicated that fathers' antisocial behaviors predicted increases in delinquency among younger, $B_{\text{Father Antisocial}} = 0.83$, $SE = 0.31$, $t = 2.73$, $p = .007$, but not older boys, $B_{\text{Father Antisocial}} = -0.06$, $SE = 0.18$, $t = -0.33$, $p = .742$. These findings, which we summarize in Figure 1, suggest that fathers antisociality exerts potentiating effects on development of delinquent behaviors in middle childhood (age 8.5), which then "fan out" across development (age 11.5). We discuss these findings in greater detail below.

3.5 | Father antisocial behaviors and child resting RSA

For slopes, the Child Sex × Child RSA × Father Antisocial three-way interaction was significant, $B_{\text{Child Sex} \times \text{Child RSA} \times \text{Father Antisocial}} = -0.51$, $SE = 0.25$, $t = -2.07$, $p = .041$. The three-way intercept interaction effect was not, $B_{\text{Child Sex} \times \text{Child RSA} \times \text{Father Antisocial}} = 0.22$, $SE = 0.35$, $t = 0.64$, $p = .527$. We followed-up the slope effect by examining RSA × Father Antisocial two-way interactions by sex. For boys, resting RSA moderated the association between fathers' antisociality and increases in delinquent behaviors, $B_{\text{Child RSA} \times \text{Father Antisocial}} = 0.33$, $SE = 0.17$, $t = 1.97$, $p = .051$. For girls, no such effect was found, $B_{\text{Child RSA} \times \text{Father Antisocial}} = -0.18$, $SE = 0.18$, $t = -0.98$, $p = .328$.

Next, we evaluated slope effects for boys at −1.0 *SD* (~6.0 [log(beats/min^2/Hz)]) and + 1.0 *SD* (~8.15[log(beats/min^2/Hz)]) of mean resting RSA at Y1. Fathers' antisocial behaviors were associated with increases in delinquency for boys with high resting RSA ($B_{\text{Father Antisocial}} = 0.61$, $SE = 0.25$, $t = 2.38$, $p = .018$), but not for boys with low resting RSA ($B_{\text{Father Antisocial}} = -0.06$, $SE = 0.19$, $t = -0.28$, $p = .778$). Taken together, these findings, which we summarize in Figure 2, suggest that father antisociality exerts potentiating effects on development of delinquent behaviors for boys with higher resting RSA.

Finally, we compared coefficients across Models 2 and 3 to determine whether patterns of findings or statistical significance changed when modeling demographics. No appreciable changes were found. We therefore report specific coefficients only for Model 2.

4 | DISCUSSION

We sought to better understand effects of fathers' antisociality on children's emerging, self-reported delinquency across middle childhood and adolescence. Rather than evaluating only main effects, we assessed theoretically informed moderators, including child sex, age, and resting RSA. As expected, associations between paternal antisocial behaviors and children's self-reported delinquency were moderated by sex. For girls, both initial rates of and growth in self-reported delinquent behaviors were unrelated to parental antisociality. Girls' delinquency scores were also unrelated to age at study entry and resting RSA. We found a significant Child Sex × Child RSA × Father Antisocial three-way interaction on rates of increase in delinquent behaviors, and marginal Child Sex × Child Age × Father Antisocial three-way interactions on both initial levels of and rates of change in delinquent behaviors. Given null findings on all measures for girls, we focus most of our remaining discussion on outcomes for boys. We note that two-way interactions were probed based on *marginal* findings from three-way interactions, and should therefore be interpreted with caution.

For boys, relations between father antisociality and self-reported delinquency were moderated by both age at study entry and resting RSA. Although boys' delinquency scores were unrelated to paternal antisocial behaviors at age 8, growth in boys' delinquency increased steeply thereafter specifically for those who had fathers with higher antisocial symptoms. These findings are consistent with previous reports of stronger effects of fathers' antisociality and harshness on boys' versus girls' externalizing trajectories (e.g., Gryczkowski et al., 2010; Kerr et al., 2004). In addition, when fathers

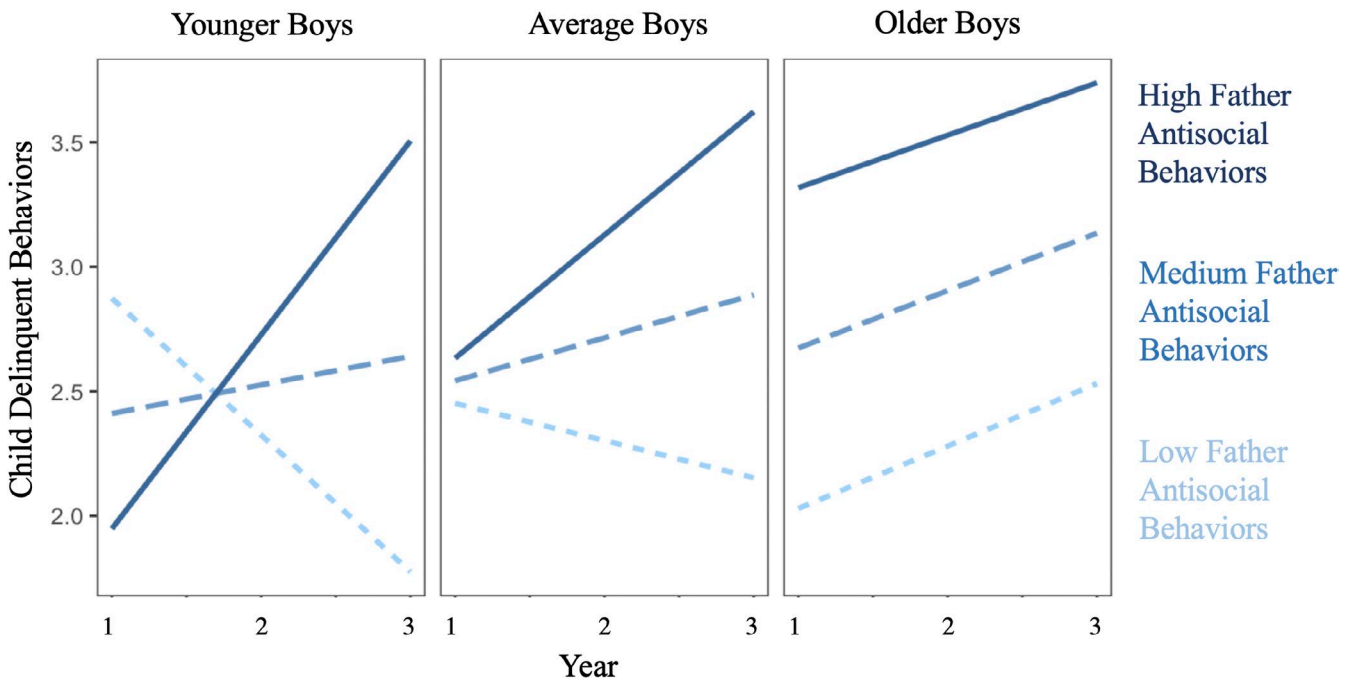

FIGURE 1 Intercepts and growth trajectories for boys' delinquent behaviors based on age at study entry and father antisociality. The left panel depicts boys at 1.0 *SD* below the sample mean of age at study entry (~8.5 years at Y1), the middle panel depicts boys at the sample mean of age at study entry (~10.0 years at Y1), and the right panel depicts boys at 1.0 *SD* above the sample mean of age at study entry (~11.5 years at Y1), at three levels of father antisociality. See text for additional details

scored high on antisociality, boys with higher resting RSA showed steep increases in delinquency, whereas boys with lower resting RSA did not. Taken together, findings for boys highlight the importance of considering conjoint effects of fathers' mental health and children's individual differences (here, sex, and PNS function) when studying socioemotional adjustment during middle childhood and early adolescence.

In disaggregating age effects, we identified (a) no relation between father antisociality and boys' delinquency at study entry (intercepts), (b) strong effects of father antisociality on growth in boys' delinquency only among younger participants (slopes), and (c) significant effects of father antisociality on boys' delinquency scores only among older participants (intercepts). Collectively, these findings suggest both potentiating (early) and maintaining (later) effects of paternal antisociality on boys' delinquency (see Figure 1). We note, however, that our findings are descriptive, and do not support inferences about causal mechanisms. Although it might be tempting to infer a sensitive period in the development of delinquency, this would go beyond the data at hand, which are limited by self-reports, and by potential floor effects for delinquency early in development given limited opportunities for children to engage in criterion behaviors (e.g., Richers & Cicchetti, 1993). In future studies, specific risk and protective mechanisms (e.g., mothers' parenting, peer group influences, etc.), potential neuroaffective mechanisms, and different forms of father antisociality should be evaluated (Beauchaine et al., 2017; Moffitt, 1993; Nelson et al., 2017; Pfeifer et al., 2011; Raine, 2018).

In disaggregating RSA effects, we found that higher resting RSA was associated with increases in delinquency scores specifically for sons of fathers with higher antisocial symptoms. Typically, low resting RSA marks poorer self-regulation in clinical samples (Beauchaine et al., 2001, 2007; Mezzacappa et al., 1997; Pang & Beauchaine, 2013; Rukmani et al., 2016). Indeed, *main effects of low resting RSA on other externalizing measures have been reported in this very sample* when examining parent-reports of child behaviors. (Beauchaine et al., 2007, 2008; Shannon et al., 2007). Here, we assessed child reports and found no association between RSA and delinquency. Moreover, still other papers from this sample (Pang & Beauchaine, 2013) did not find associations between parent-reported CD and resting RSA. Thus, as is often the case, results from parent- and child-reports differ. Child-reports, considered herein, include information about delinquency that parents are unaware of. Our results suggest that a differential vulnerability model may fit child-report but not parent-report data. Findings suggest a potentiating effect of father antisociality on boys' delinquency for those with high resting RSA. One possible interpretation is that of biological sensitivity to context– boys with high resting RSA may be more sensitive to paternal antisociality than boys with low resting RSA, who tend to show higher externalizing behavior *regardless* of their fathers' antisociality scores (Shannon et al., 2007).

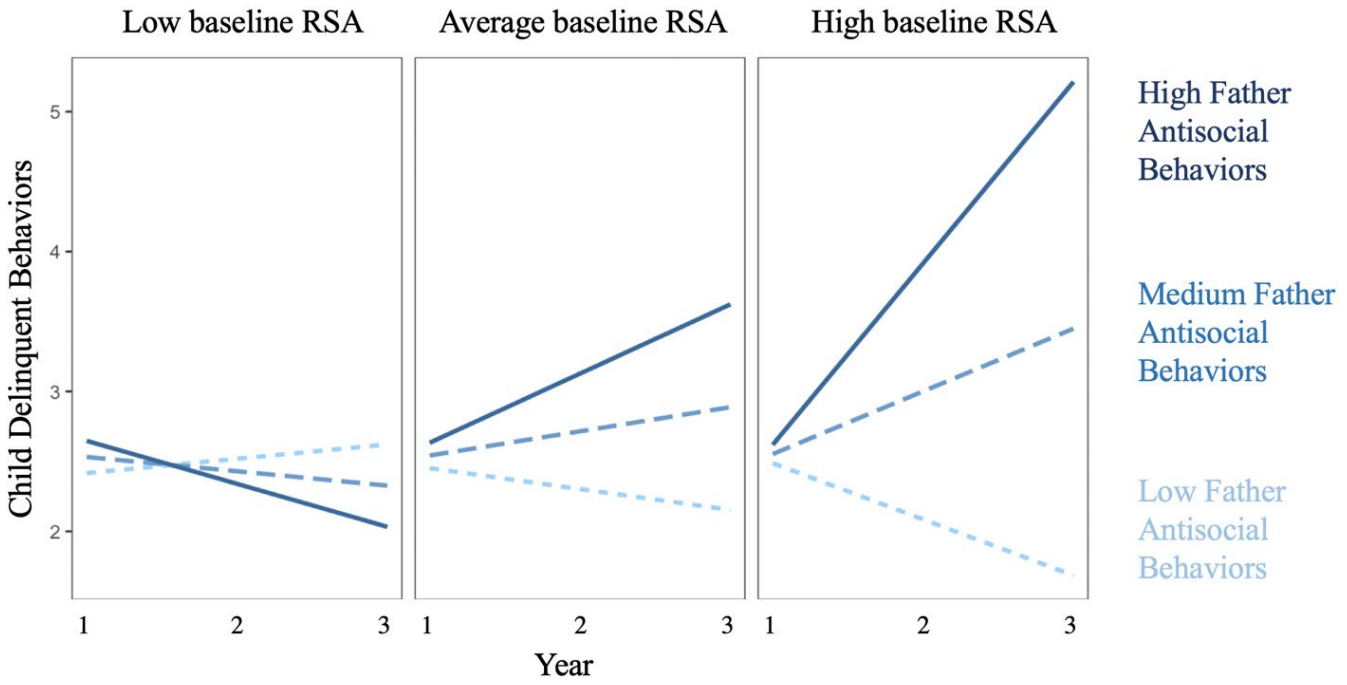

FIGURE 2 Intercepts and growth trajectories for boys' delinquent behaviors based on levels of resting RSA at study entry and father antisociality. The left panel depicts boys at 1.0 *SD* below the sample mean of resting RSA (~6.0 log[beats/min2/Hz]), the middle panel depicts boys at the sample mean of resting RSA at study entry (~7.05 log[beats/min2/Hz]), and the right panel depicts boys at 1.0 *SD* above the sample mean of resting RSA (~8.15 log[beats/min2/Hz]), at three levels of father antisociality. See text for additional details

In infancy, RSA correlates with higher levels of reactivity and engagement with environments (see e.g., Beauchaine, 2001), perhaps reflecting biological sensitivity (Perry et al., 2018). Our findings suggest that similar patterns may be found beyond infancy, such that higher resting RSA magnify effects of environmental adversity on emerging externalizing behavior. This is consistent with findings from prior research conducted in early and middle childhood (e.g., Sturge-Apple et al., 2016; Tabachnick et al., 2020). Whereas low resting RSA is a reliable marker of poor self-regulation in clinical samples (Beauchaine et al., 2001, 2007; Mezzacappa et al., 1997; Pang & Beauchaine, 2013; Rukmani et al., 2016), high resting RSA may mark plasticity to environmental adversity, including father antisociality. This is consistent with evolutionary-developmental accounts of children's biological sensitivity to context (Ellis et al., 2011). That said, given both (a) a limited sample size, and (b) mixed directions of effects for RSA as a biomarker of susceptibility in previous research (see above), strong conclusions are unwarranted, and more research is needed.

An additional question concerns the extent to which RSA is inherited versus shaped by environment. Research suggests both heritable and environmental effects on RSA, which, as outlined above, serves as a biomarker of self- and emotion regulation (Beauchaine et al., 2007; Beauchaine & Thayer, 2015). Children who are reared in emotionally labile, coercive families show poor emotion regulation and altered RSA (Beauchaine & Bell, 2020). Reducing negative parenting through intervention improves children's emotion regulation and normalizes their RSA (Bell et al., 2018). For this specific study, we cannot disentangle the extent to which heritable versus environmental influences contributed to children's RSA given that we have no data on fathers' involvement with participant children. Future research should address this question directly.

4.1 | Alternative Interpretations to the Findings

Intergenerational transmission of antisocial behaviors involves complex interactions and transactions among various biological vulnerabilities and environmental risk factors over time (see Beauchaine & Hinshaw, 2020; Beauchaine et al., 2017; Thornberry et al., 2003). No study, including this one, can capture or disentangle even a fraction much less all of these mechanisms. Because we did not have information on the quantity and quality of fathers' involvement, we do not know how often fathers exerted direct influences on their children, making it difficult to disentangle biological vulnerabilites versus environmental risk factors associated with paternal antisociality. Additionally, because fathers' lifetime antisocial behaviors were reported by mothers, the measure may be confounded with quality of father–mother relationships and mothers' characteristics.

Thus, several alternative interpretations could explain our findings. Among these, associations between fathers' higher levels of antisocial behaviors and boys' delinquent behaviors may be explained by shared genetic propensities of fathers and their male offspring (e.g., Silberg et al., 2012). It is also possible that this association reflects effects of problematic coparenting relationships or maternal distress on children (Schoppe-Sullivan & Fagan, 2020).

4.2 | Limitations and strengths

Several limitations should be mentioned. In this study, fathers' antisocial behaviors were reported by mothers. Although this reduces biases associated with fathers' underreporting and unwillingness to participate in research, it can introduce other confounds, including but not limited to retrospective reporting biases by mothers (especially for those who no longer associate with their children's father) and halo effects. Mother reports may also be systematically biased based on the length and quality of their relationship with the child's father, mother–father relational dynamics, and maternal distress. We were not able to accurately assess reliability and validity of mothers' reports of fathers' lifetime antisocial behaviors without information on these factors. Considering the important roles that fathers play in their children's socioemotional development, more father data are needed (Barker et al., 2017). Of note, given recent advances in missing data techniques, the field may be able to gain valuable insight about father influences even when only subsets of samples include father reports (Cabrera et al., 2018; Enders, 2010; Young & Johnson, 2015). Here, however, there were far too few fathers for such approaches.

In addition, the sample contained a relatively high proportion of Caucasian children, which limits generalizability to the broader population. It should also be emphasized that about half of children were recruited into the study based on externalizing behaviors—a sampling strategy that likely affected results (Munafò et al., 2018). We also oversampled children with depression and heterotypic comorbidity, which could result in overestimation of strengths of effects. Additionally, we used raw scores of children's self-reported delinquent behaviors instead of *T*-scores. Although this is recommended in many contexts (Achenbach & Edelbrock, 1991), mixing boys' and girls' raw scores for analysis can reduce sensitivity of analyses to girls' behaviors given large mean differences between sexes on virtually all externalizing outcomes (see Eme, 2016). We decided to use raw scores because *T*-scores are normed by age, which obscures the very patterns of growth we sought to assess. We also relied on a single informant—children/adolescents. Although child-/adolescent-reports of delinquency have advantages over parent-reports because parents are unaware of many delinquent behaviors (especially in adolescence; see Laird et al., 2003), self-reports also have drawbacks. These include systematic underreporting and difficulties eliciting honest responses (e.g., Krohn et al., 2010). Future research would ideally replicate findings across multiple reporters of child delinquency.

Finally, we had limited power to test high-order interactions. Findings should be interpreted in this context, and future research with larger samples should be conducted. Despite these limitations, our study underscores both (a) the importance of considering interactive contributions of multiple factors toward understanding the development of externalizing behavior from middle childhood to early adolescence, and (b) the need for prevention and early intervention given rapid growth in self-reported delinquency among boys at an early age.

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DATA AVAILABILITY STATEMENT

The data used in this study are available on request from the corresponding author. These data are not available publicly due to privacy/ethical restrictions.

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ENDNOTE

[1] Unfortunately, we do not have information on the amount of father contact or involvement.

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