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Pathways From Early Family Violence to Adolescent Reactive Aggression and Violence Victimization

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Abstract

Purpose—The current study examined how early childhood (EC) family violence and risk (i.e., maternal aggression, sibling aggression, environmental risk) predicted early adolescent (EA) reactive physical and relational aggression and violence victimization through middle childhood (MC) parenting (i.e., guilt induction, power assertive discipline).

Method—Mother-infant dyads (N = 216; 72% African American) were recruited as part of a larger longitudinal study on prenatal cocaine and other substance exposure. Observations, interviews, and maternal and child self-report measures were collected from dyads in early childhood (1 to 36 months), middle childhood (84 months), and early adolescence (12 to 15 years).

Results—A cascading path model was specified where current variables were regressed on variables from the preceding time point. Primary results showed that environmental risk and EC child physical aggression predicted higher levels of MC caregiver power assertive discipline, which subsequently predicted lower levels of EA reactive relational aggression. Maternal substance use in pregnancy and the child's continuous placement with biological caregivers predicted higher levels of reactive physical aggression in EA. Finally, MC physical aggression and EA reactive relational aggression predicted higher levels of EA violence victimization.

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Conclusion—There were a series of direct paths from early childhood family violence and demographic factors to reactive aggression and violence victimization. The current study underscores the importance of evaluating multiple facets of family violence and risk when evaluating aggressive behavior and victimization.

Keywords

reactive aggression; physical aggression; relational aggression; violence victimization; sibling aggression; parent aggression; parenting; prenatal substance exposure

Family violence is a common occurrence, with 37.3% of youth endorsing physical assault by peers or siblings and 15.2% endorsing maltreatment within the past year (Finkelhor, Turner, Shattuck, & Hamby, 2015). *Violence* has historically been defined as an extreme or severe form of aggression that has the potential to lead to serious injury or even death (Liu, Lewis, & Evans, 2013; Ostrov & Perry, 2018). Researchers have conceptualized acts such as child abuse or intimate partner violence (IPV) as family violence. More recently researchers have recognized the importance of other family relationships, such as siblings, in contributing to overall family violence and child outcomes (Button & Gealt, 2010; Tucker, Finkelhor, & Turner, 2018a).

There is overwhelming evidence that children who experience family violence are at a higher risk for experiencing maladaptive outcomes, such as internalizing and externalizing behavior problems, and are more likely to become victims and perpetrators of other types of violence (i.e., dating violence, gang violence, etc.; McKinney et al., 2009; Smith-Marek et al., 2015). The current study examined how early life family factors (i.e., parent aggression, sibling aggression, environmental risk) have cascading effects to early adolescent reactive aggression and violence victimization through middle childhood parenting practices. Specifically, the forms of *reactive aggression* (i.e., aggression defined by impulsive and retaliatory aggressive acts) were used to examine *reactive relational aggression* (i.e., intent to harm through the relationship or threat of the removal of the relationship) and *reactive physical aggression* (i.e., the intent to harm through physical acts or threat of physical acts).

An ecological model of development (Bronfenbrenner, 1979) is useful for understanding and studying aggression, victimization, and violence (Swearer & Hymel, 2015). This model focuses on individual characteristics and the multiple systems and environmental contexts that promote or prevent aggression and victimization. There is a dynamic interaction between the child and his or her family environment, such that the parent and child both influence one another's behavior, which impacts how they behave in other environments. An abundance of research has found that witnessing or experiencing violence or aggression within the family can generalize to other domains, such as the child exhibiting or being the recipient of violence or aggression with peers (e.g., Espelage, Low, Rao, Hong, Little, 2014). The goal of the current study was to examine a conceptual model predicting adolescent aggression and violence victimization spanning from the prenatal period to early adolescence using a diverse sample at high risk due to maternal substance use in pregnancy and associated risk factors. The model included several factors within the family environment that likely contribute to the overall context of family violence, such as child

aggression, sibling aggression, and parenting aggression. In addition, environmental risk (i.e., maternal depression, maternal exposure to violence, caregiving instability) was included in the model, as these factors are predictors of violence in the home. Given the high risk nature of the sample, maternal substance use in pregnancy and demographic risk (i.e., a child in non-biological care, socioeconomic status adversity, and the number of maternal children) variables were considered as covariates as these factors have emerged as important precursors to overall violence in the home and children's aggressive behavior (Hentges, Shaw, & Wang, 2018; Min et al., 2018).

Developmental cascade models allow researchers to empirically examine the distal and proximal determinants and interactions in systems that alter the course of a child's development and lead to outcomes across contexts (Dodge, Greenberg, Malone, & Conduct Problems Prevention Research Group, 2008; Masten & Cicchetti, 2010). Cascade models are particularly useful in examining developmental processes spanning long periods of time such as from prenatal risk to adolescent outcomes. Dodge and colleagues (2008) found support for a developmental cascade model of adolescent violence, such that an early disadvantaged environment beginning in the prenatal period led to adolescent violence through factors such as early harsh-inconsistent parenting, diminished cognitive and social skills, and less parental monitoring in adolescence. The current study used a developmental cascade model grounded in ecological theoretical frameworks, positing that early family processes would predict future child aggression and community violence victimization through modeling and interactions with the parent and sibling. Developmental cascade models are specified in a structural equation modeling (SEM) framework by allowing for variables at one time point to be regressed on variables from the prior time point (e.g., Dodge et al., 2008; Eiden et al., 2016; see Figure 1).

Sibling Aggression and Child Aggression

Sibling relationships are an important training ground for subsequent peer relationships and for many children they serve as the first horizontal relationship system (Dunn, 1993). Moreover, sibling violence is the most common form of family violence and is more prevalent in families high in other aspects of family violence and adversity, such as in families where children experience corporal punishment, physical abuse, or instability (Button & Gealt, 2010; Tucker, Finkelhor, & Turner, 2018a). Additionally, children who experience high levels of sibling victimization are more likely to experience peer victimization, providing evidence for the generalizability of victimization across contexts (Tucker et al., 2018b).

Environmental Risk and Child Aggression

A large body of literature has demonstrated a robust link between environmental risk factors and both aggression and victimization among adolescents. In the current study, environmental risk is defined by family contextual variables such as maternal depression, maternal exposure to violence, and caregiving instability. These variables may be particularly predictive of adolescent aggression. For example, numerous studies have demonstrated an association between aspects of maternal psychopathology, such as

depression and hostility, and externalizing behaviors in children and adolescents (e.g., Allen, Manning, & Meyer, 2010). Another salient contextual risk that has been linked to increased aggression in middle childhood and adolescence is maternal exposure to violence both within and outside of the home (Deater-Deckard, Dodge, Bates, & Pettit, 1998; Vu, Jouriles, McDonald, & Rosenfield, 2016). In addition, disruptions to the parent-child relationship as a result of experiences such as separations from the parents (Averdijk, Malti, Eisner, & Ribeaud, 2012) or absence of father-involvement in child rearing (Deater-Deckard et al., 1998; Aimé, Paquette, Déry, & Verlaan, 2018) have been linked to aggressive and antisocial behavior in both children and adolescents.

Parenting and Child Aggression

In the current study parenting was theorized to have an initial and intermediate effect on the development of aggressive behavior and victimization in adolescence. Specifically, early childhood parent aggression was theorized to predict middle childhood parental psychological control and power assertive discipline, which in turn would predict adolescent outcomes. Harsh, ineffective parenting has been consistently and robustly related to child aggression (Labella & Masten, 2018). In particular, aggression within parent-child interactions in early development is associated with later child aggression with peers (e.g., Olson, Lopez-Duran, Lunkenheimer, Chang, & Sameroff, 2011), and with parents' use of less adaptive parenting practices (e.g., Rodriguez, 2010). Therefore, parents who are high on aggression within the parent-child relationship in early childhood demonstrate higher levels of dysfunctional parenting (i.e., guilt induction and power assertive discipline) in middle childhood (Rodriguez, 2010). In middle childhood and adolescence, specific types of parenting, such as psychological control, have been found to be uniquely related to relational aggression whereas physical punishment or harsh discipline have been found to be uniquely associated with physical aggression (e.g., Kuppens, Grietens, Onghena, & Michiels, 2009; Kuppens, Laurent, Heyvaert, & Onghena, 2013), suggesting that there may be specificity of effects from parenting practices to reactive relational and physical aggression.

Reactive Aggression and Violence Victimization

To date, the focus of research on reactive functions of aggression has been on peer victimization (e.g., Renouf et al., 2010), and other forms of victimization, such as *violence victimization* (i.e., more serious acts of victimization within the community such as being a victim of burglary or sexual assault) have been understudied. This is surprising as reactive aggression may also be associated with these types of victimization during adolescence. Reactive aggression and associated feelings of retaliation may lead some adolescents to believe that fighting is necessary (Bettencourt & Farrell, 2013) and this in turn may reinforce their victimization status by increasing exposure to violence at school and in the community. A study of undergraduates revealed that reactive functions of aggression were concurrently associated with multiple types of stressful life events (e.g., war zone exposure, interpersonal victimization, and sexual victimization) that were experienced or witnessed (Brown, Fite, DiPierro, & Bortolato, 2017). However, there is limited research on pathways from reactive aggression to violence victimization. Thus, it is conceivable that reactive aggression subtypes may be associated with more serious forms of victimization in an at-risk sample.

Child Biological Sex as a Moderator

Prior researchers have called for the inclusion of gender informed models when examining the precipitants and outcomes of aggressive behavior (e.g., Ostrov & Perry, 2018). However, prior work has found limited evidence of differential effects in developmental cascade models of adolescent violence for males and females (e.g., Dodge et al., 2008) and a recent meta-analysis found no gender differences (i.e., Pinquart, 2017). Nonetheless, there have been gender differences found in relations between psychological control, related to guilt induction in the current study, and relational aggression, where the effect between parent psychological control and relational aggression was larger for girls (Kawabata et al., 2011). Finally, there is evidence of between group gender differences (i.e., boys display more physical aggression) and within group gender differences (i.e., girls are more likely to display relational than physical aggression) in aggressive behavior (Card, Stucky, Sawalani, & Little, 2008). In the current study, biological sex is considered as a moderator in lieu of gender, since this variable was collected at birth. Given the limits of sample size, only significant cascading paths in the current model were tested for moderation by sex instead of all the significant pathways in the final model for the full sample.

Current Study (Hypotheses)

The present study builds upon this prior work by examining a longitudinal developmental cascade model across several developmental periods in an at-risk sample using a comprehensive set of family violence predictors while also evaluating the continuity of negative parenting practices. Moreover, one common criticism of prior work examining aggressive behavior is a failure to parse apart the different subtypes of aggression. This work addresses this limitation by examining reactive relational and physical aggression. Finally, a more severe type of victimization, violence victimization, is examined. Few studies of reactive aggression and violence victimization have included such a long developmental span or included prenatal risk variables (e.g., maternal substance use and socioeconomic adversity in pregnancy) as significant predictors of cascading effects across time. Substance using families may be particularly important to include in studies of violence victimization given the co-occurrence of illicit substance use and violence and potential child exposure to community and family violence (Ackerman, Riggins, & Black; 2010; Eiden, Peterson, & Coleman, 1999; Veira et al., 2014).

Consistent with a developmental cascade model and prior studies of prenatal risks such as substance exposure and socioeconomic adversity (e.g. Eiden et al., 2016), it was postulated that negative family factors in early childhood (i.e., sibling aggression, parenting aggression, child aggression) and environmental risk would predict early adolescent reactive relational and physical aggression through the cascading associations with child aggression and parenting practices in middle childhood. Furthermore, it was hypothesized that within early adolescence reactive physical and relational aggression would predict violence victimization. The hypothesized model is shown in Figure 1.

Method

Participants and Procedure

The sample consisted of 216 mother–infant dyads recruited after delivery from two area hospitals serving a predominantly low-income population for a longitudinal study on the effects of prenatal cocaine exposure (PCE) that almost always occurs in the context of maternal use of multiple substances. A final sample of 216 dyads were recruited (116 cocaine exposed, 100 non-cocaine exposed (but majority using substances other than cocaine), 106 biologically male; see Eiden, Godleski, Schuetze, & Colder, 2015 for more details on recruitment).

The procedures were approved by the university IRB. Informed written consent was obtained from all recruited participants, and they were compensated for their time. Data collected from mothers and children in early childhood (at about 4–8 weeks, 7, 13, 18, 24, 30, and 36 months of child ages), in middle childhood (when the children were in 2nd grade), and again in early adolescence (adolescent age ranged from 12 to 15 years, *M* age = 13.26 years, *SD* = 0.88 years) were used in the current analyses. Children born at or before 37 weeks gestation were scheduled for their appointments at chronological age corrected for prematurity until the 24-month assessment. By the 36-month assessment, 50 children had been or currently were in non-biological parent care that included kinship care and foster care. At the early adolescent assessment, an additional 5 children had been placed in non-biological parent care and 11 children who had been in non-biological parent care at 36-months were now in biological parent care. All assessments were conducted with the primary caregiver of the child at that time, although for ease of presentation the terms mother and maternal are used throughout when referring to the primary caregiver. Biological mothers were interviewed at the 4- to 8-week assessment in addition to the foster mothers in order to obtain accurate information about prenatal substance use. The primary caregiver was identified as the adult who had legal guardianship of the child and accompanied the child at all appointments.

Biological mothers ranged from 18 to 42 years old (*M* = 29.53; *SD* = 6.06) at recruitment. The majority (76%) were receiving Temporary Assistance for Needy Families, 66% were single, 69% were on Medicaid, 27% were unemployed, and 71% had high school or below education. About 72% of mothers were African-American, 8% Hispanic, 19% Caucasian, and 1% other. Mothers who participated (vs. those eligible but not enrolled) were more likely to be (a) 18–25 years old, (b) have high school or below education, and (c) in the PCE group.

Measures

Control variables

Maternal substance use in pregnancy: Four sources were used to measure prenatal substance use: health screener, detailed self-report, maternal and infant urine, and maternal hair. Urine toxicology results were available for 90% of the families in the no prenatal cocaine exposure (NCE) group and 92% of the families in the PCE group, and hair samples were collected from all mothers. Urine toxicologies consisted of standard urine screening for

drug level or metabolites of cocaine, opiates, benzodiazepines, and tetrahydrocannabinol. Urine was rated as positive if the quantity of drug or metabolite was greater than 300 g/ml. Hair samples were screened for cocaine, followed by a gas chromatography/mass spectrometry (GC/MS) confirmation for positive cocaine screens. The Timeline Follow-Back Interview (TLFB; Sobell, Sobell, Klajner, Pavan, & Basian, 1986) was used to assess maternal substance use during pregnancy and is established as a reliable and valid method of obtaining longitudinal data on substance use patterns, has good test–retest reliability, and is correlated with other measures (Brown et al., 1998). Mothers also completed the Fagerstrom Test for Nicotine Dependence (FTND) at the first appointment (Heatherton, Kozlowski, Frecker, & Fagerstrom, 1991).

The cumulative prenatal risk composite was created by computing a count variable, which included all of these substance use measures within each trimester and then summed across trimesters. Participants were given a score of one within each trimester using the following cut-offs: maternal cigarette use per week of 7 or higher (high probability of daily smokers) as reported on the TLFB or a nicotine dependence score of 1 or more on the FTND; any prenatal alcohol use as indicated on the health screener, TLFB, or positive urine screen; smoking 1 joint per week or more during pregnancy on the health screener or the TLFB, or if mother/infant urine was positive for marijuana metabolites reflecting persistent use in pregnancy; any indication of maternal cocaine use in health screener, TLFB, maternal/infant urine, or maternal hair. The scores for cumulative prenatal risk could range from 0 to 15, and ranged from 0 to 13 within this sample.

Socioeconomic adversity: SES adversity was assessed at 1-month of child age as a composite variable consisting of maternal education, family structure, and family income. For each component, higher scores indicated greater SES adversity. An average of all three indicators was computed (Moran et al., 2016).

Non-biological parent care: Child's care status (in biological parent care, kin care, foster care, adoptive care, etc.) was assessed at every time point. Given small cell sizes, care status was dichotomized at each time point (in biological vs. ever been in non-biological parent care) and a composite variable reflecting *0 = never been in out of home care* (n = 161) or *1= ever been in non-biological parent care* (n = 55) from birth to early adolescence was used as a covariate.

Maternal children: At one month of age, mothers were asked: "How many children do you have?" These children could be biological or nonbiological children of the mothers. Values ranged from 0 to 13 with an average of 3.65 (SD = 2.06).

Early childhood variables

Environmental risk: A composite caregiving environmental risk score was computed and was comprised of maternal psychopathology, maternal exposure to violence, and caregiving instability. Specifically, maternal psychopathology was assessed using a total score of the Brief Symptom Inventory (BSI; Derogatis, 1993) at 1, 7, 13, 24, and 36 months. Maternal exposure to violence was assessed at each time point (i.e., 1, 7, 13, 18, 24, 30, and 36

months) using the TLFB (Sobell et al., 1986). Women were asked about their exposure to violence using a daily calendar. The total number of days women witnessed, experienced, or perpetrated violence were summed within each time point. Caregiving instability was assessed at 1, 7, 13, 24, and 36 months using the Structured Clinical Interview (SCI; Platzman, Coles, Lynch, Bard, & Brown, 2001) which was administered to the child's caregiver by a trained examiner.

The cumulative caregiving environmental risk variable was created by computing a count variable across time periods, which included BSI, exposure to violence, and caregiving instability. Specifically, a point was granted when a person had a score in the upper quartile for total BSI for each time point that it was assessed, when a person received a score of 1 for the dummy-coded variable for maternal exposure to violence for each time point that it was assessed, and when a person received a score of 1 for the dummy-coded variable for caregiving instability for each time point that it was assessed. For more details see Eiden et al., 2015.

Child physical aggression: At 18, 24, and 36 months mothers completed the Child Behavior Checklist (CBCL; Achenbach & Rescorala, 2001). Mothers were asked how often a behavior occurred over the past two months and rated items on a three point Likert scale ranging from *0 (not true)* to *2 (very true or often true)*. A 6-item composite of overt physical aggression (e.g., NICHD ECCRN, 2004) was used. A weighted sum was used to account for missing data, which was then averaged across time points to get an index of early childhood aggression (Cronbach's αs ranged from .75–.81).

Sibling physical aggression: At 24 months, mothers completed reports of the CBCL for up to six of the child's siblings and at 36 months they completed reports of the CBCL for one of the child's siblings. The same aforementioned physical aggression composite was computed for siblings and was reliable at 24 months (Cronbach's α = .75). At 24 months, the siblings' aggression scores were averaged to get an indication of sibling aggression. Twenty-four month scores were used in lieu of 36 month scores because there was more sibling data at the 24-month time point, therefore, offering a better indication of sibling aggression in the home. If a child had a missing sibling aggression score at 24 months, then the 36-month aggression score was used.

Parent aggression: Maternal aggression or harshness was coded during specific segments of the 24 and 36 month observational assessments. These included a 10-minute mother–child free play paradigm (i.e., no stress), a 10-minute clean-up (i.e., moderate stress), 8-minute structured play, 10-minute eating a snack, and 5-minute emotion regulation paradigm (i.e., high levels of stress; Keenan & Shaw, 1994). Aggression/harshness was scored on the basis of codes developed in previous studies (Keenan & Shaw, 1994; Eiden et al., 2011). Coders were blind to group status. This included physical aggression directed toward a person (mother to child); physical aggression directed toward an object; verbal aggression that consists of cursing; and verbal aggression that consists of threats. Each aggressive episode was coded for duration or length of time that episode lasted, the highest rating of aggression during that episode, and an overall intensity of maternal aggression coded along a

4-point scale ranging from 1 = “no aggression” to 4 = “severely aggressive”. This intensity rating was used in model testing.

Middle childhood variables

Child physical aggression: At 84 months, overt physical aggression was measured using the same composite that was used in early childhood (NICHD ECCRN, 2004). This composite demonstrated acceptable internal consistency (Cronbach’s α = .75).

Parenting: Parent guilt induction and power assertive discipline were measured at 84 months using maternal reports of the Parental Responses to Child Misbehavior Questionnaire (Holden, Coleman, & Schmidt, 1995). This 25-item scale assesses four dimensions of parent responses: guilt induction, power assertive discipline, positive reinforcement, and age appropriate discipline. The parent guilt induction subscale (five items) and the power assertive discipline subscale (nine items) were used in the current study. The internal consistency of both scales was good (Cronbach’s α*’s* = .83 and .84 respectively).

Early adolescent variables

Reactive and proactive aggression: The Forms and Functions of Aggression Questionnaire (FFQ), a self-report measure of aggression (Little, Henrich, Jones, & Hawley, 2003), is comprised of 36 questions and was used to assess reactive relational aggression, reactive physical aggression, and proactive functions of aggression. Participants were asked how true each item is of them (*0 = not at all true* to *4 =completely true*). The subscales for reactive physical aggression and reactive relational aggression were internally consistent in the present sample (Cronbach’s α*’s* > .74). The proactive physical and proactive relational subscales were heavily skewed (kurtosis > 20.9 skew > 4.76). Therefore, the two-proactive physical and relational subscales were averaged to obtain an overall proactive aggression control variable, which had adequate reliability (Cronbach’s α = .89).

Violence victimization: The Survey of Exposure to Community Violence Checklist (CECV) is a 35 item self-report measure (Richters & Saltzman, 1990). The CECV measures the child’s exposure to several types of severe violence such as experiencing or witnessing assault, stabbings, or robberies using a 5 point Likert scale (*0 = never* to *4 = more than 10 times*). The measure includes questions such as “I have been sexually molested or raped,” and “I have been chased by gangs or gang members.” A weighted sum was used to examine the child’s total exposure to violence, which was internally consistent (Cronbach’s α = .89).

Data Analysis Plan

First, descriptive data and correlations of the measures were obtained (see Table 1). Outliers were modified by adjusting the value to +/− three standard deviations from the mean (Kline, 2011). A square root transformation was used on the early and middle childhood physical aggression variables because they were positively skewed. For other key study variables, skew values ranged from .15 to 2.03 and kurtosis values ranged from −0.74 to 3.65, which are within accepted ranges for normally distributed variables (Kline, 2011).

As with all longitudinal studies and especially due to the high-risk nature of the sample, missing data was expected. In regards to key study variables there was incomplete data for 15.7% of participants in early childhood, 24.5% of participants in middle childhood, and 30.1% of participants in early adolescence. Little's (1988) Missing Completely At Random (MCAR) test was used to examine whether the data was MCAR. All control and target variables were examined for MCAR. The MCAR test demonstrated that the data was missing completely at random [$\chi^2(267) = 300.87$, $p = .08$].

All models were estimated in Mplus version 8.2 (Muthén & Muthén, 2018). Maximum likelihood estimation with robust standard errors (MLR) was used due to a slight skew in some of the variables. Missing data was accommodated by using full information maximum likelihood (FIML). The initial model included only the paths supported by a cascade model of risk (a → b → c → d). In the present study, all direct paths were not included in our a priori model given the complexity of the cascade model and limits of sample size. Given our sample size, and the complexity of the model particularly for the multigroup analysis (i.e., $n = 106$ for boys and $n = 110$ for girls), reduced power was a concern. Therefore, direct effects across longer time periods were treated in an exploratory manner, where modification indices (MI) were examined to determine whether there were relations between maternal substance use in pregnancy, or other early life family factors, on early adolescent aggression or victimization. A modification index of 3.84 or higher (Whittaker, 2012) was used to determine whether adding a path would lead to a significant ($p < .05$) reduction in the chi-square value. Paths were added only if they were consistent with theory. Several covariates were considered, but were only included in the model if they were related to outcomes at $p < .10$. Early childhood outcomes and proactive aggression were regressed on control variables. Reactive physical and relational aggression were regressed on proactive aggression.

Multiple group analyses were used to examine if significant cascading pathways varied by sex. First, a model was tested in which a significant cascading path was free to vary. Second, a model was tested in which the regression paths were constrained to equivalence. This was repeated for other significant cascading paths. The MLR fit index uses a scaling factor to adjust the chi-square test statistic and therefore, methods developed by Satorra and Bentler (2010) were used to calculate the chi-square difference test statistic. If there was a significant difference, MI were used to determine which parameters should be sequentially freed in accordance with procedures outlined by Yoon and Millsap (2007).

A likelihood ratio χ^2 test was used to test overall model fit where $p > .05$ indicates good model fit. The following alternative fit indices were also considered: (a) comparative fit index (CFI), where values greater than .95 suggest good fit, (b) standardized root mean-square residual (SRMR) where values less than .08 represent mediocre fit, and values less than .05 indicate close fit (Hu & Bentler, 1999), and (c) root mean square error of approximation (RMSEA; Steiger, 1990), where values less than .08 suggest mediocre fit, and values less than .05 indicate close fit (Browne & Cudeck, 1992; MacCallum, Browne, & Sugawara, 1996).

Results

First, a cascading path model was specified. All within time point covariances were included. This model was not identified. Removing the within time point covariances between non-biological parent care and other control variables led to a model which provided an adequate fit to the data [$\chi^2(43) = 72.19$, $p = .004$, CFI = .89, SRMR = .05, RMSEA = .06].

The results of model testing suggested two additional theoretically justifiable paths that were added one at a time. The paths were from maternal substance use in pregnancy to early adolescent reactive physical aggression and from middle childhood physical aggression to early adolescent violence victimization. A model with these two paths added provided a good fit to the data [$\chi^2(41) = 56.61$, $p = .05$, CFI = .94, SRMR = .05, RMSEA = .04]. No remaining modification indices reached a threshold of 3.84 or were theoretically meaningful.

All significant paths for this model are shown in Figure 2. Primary results showed that early childhood environmental risk ($\beta = .20$, $p = .01$) and physical aggression ($\beta = .17$, $p = .02$) predicted higher levels of middle childhood power assertive discipline. In turn, middle childhood power assertive discipline predicted lower levels of reactive relational aggression ($\beta = -.19$, $p = .04$). There were also direct effects from maternal substance use in pregnancy ($\beta = .25$, $p < .01$) to early adolescent reactive physical aggression. Non-biological caregiver status in early childhood or adolescence, predicted lower levels of sibling aggression in early childhood ($\beta = -.19$, $p = .02$), lower levels of maternal guilt induction in middle childhood ($\beta = -.14$, $p < .01$), and lower levels of child reactive physical aggression in early adolescence ($\beta = -.20$, $p < .01$). Finally, middle childhood physical aggression ($\beta = .17$, $p = .049$) and early adolescent reactive relational aggression ($\beta = .19$, $p = .02$) predicted higher levels of violence victimization.

Post-hoc, the significance of indirect cascading effects were examined for longitudinal cascading paths. The indirect path from early childhood environmental risk to early adolescent reactive relational aggression through middle childhood power assertive discipline was not significant [$\beta = -.04$, $p = .13$, 95% CI (−.09, .01)]. Second, the indirect path from early physical aggression to early adolescent reactive relational aggression through middle childhood power assertive discipline was not significant [$\beta = -.03$, $p = .15$, 95% CI (−.08, .01)], suggesting that even though there are significant individual pathways, the sum of the cascading pathway does not exert a significant effect. It is important to note that direct effects were not accounted for (i.e., environmental risk to reactive relational aggression and early childhood physical aggression to reactive relational aggression), and bootstrapping was not available with the MLR estimator. Paths with reactive relational aggression as an indirect effect to violence victimization were not examined, given the cross-sectional nature of the two variables.

Next, a multi-group analysis was run to examine whether significant pathways were moderated by sex. Non-biological parent care status was used as a covariate. First, the path from environmental risk to violence victimization through power assertive discipline and reactive relational aggression was freed and provided a poor fit to the data [$\chi^2(130) =$

183.18, $p = .002$, CFI = .84, SRMR = .09, RMSEA = .06]. The constrained model, was also a poor fit to the data [$\chi^2(133) = 187.37$, $p = .001$, CFI = .84, SRMR = .09, RMSEA = .06] and there was no difference in model fit between the two models [$\Delta\chi^2(3) = 4.19$, $p = .24$]. Second, a model with the path freed from early childhood aggression to reactive relational aggression and violence victimization through power assertive discipline provided a poor fit to the data [$\chi^2(130) = 184.36$, $p = .001$, CFI = .84, SRMR = .09, RMSEA = .06] and there was no difference in model fit with the constrained model [$\Delta\chi^2(3) = 2.73$, $p = .43$]. Third, a model was specified where the path from early childhood aggression to middle childhood aggression to violence victimization was freed, which provided a poor fit to the data [$\chi^2(131) = 186.34$, $p = .001$, CFI = .84, SRMR = .09, RMSEA = .06] and provided no difference in model fit with the constrained model [$\Delta\chi^2(2) = 1.04$, $p = .60$].

Discussion

The aim of the current study was to examine cascading influences between prenatal/early life family (i.e., parent aggression, sibling aggression) and environmental risk to early adolescent reactive aggression and violence victimization through middle childhood parenting in an at-risk sample. As hypothesized there were a series of cascading direct effects from these early life family factors to later outcomes. Environmental risk and child physical aggression in early childhood predicted maternal power assertive discipline in middle childhood, which in turn predicted reactive relational aggression in early adolescence. There were direct effects from maternal substance use in pregnancy and early childhood non-biological parent care to early adolescent reactive physical aggression. Reactive relational aggression concurrently predicted violence victimization and middle childhood physical aggression predicted future victimization.

As hypothesized, there were a series of direct effects from early childhood to early adolescent reactive relational aggression through middle childhood parenting. Specifically, environmental risk and child physical aggression in early childhood were prospectively predictive of maternal power assertive discipline in middle childhood, which in turn accounted for unique variance in reactive relational aggression in early adolescence. This suggests that the child's behavior and environmental risk are predicting the mother's parenting behavior, which is then influencing the child's aggression, supporting transactional models of parent-child influences (Labella & Masten, 2018). Contrary to hypotheses, maternal use of power assertive discipline in middle childhood predicted less reactive relational aggression in early adolescence. The at-risk nature of the sample may create a context in which power assertive discipline is less likely to be associated with maladjustment (Deater-Deckard, Dodge, Bates, & Pettit, 1996).

In contrast to our hypotheses, there were not cascading effects to early adolescent reactive physical aggression. There were direct effects from maternal substance use in pregnancy and non-biological parent care. Children whose mothers had engaged in more prenatal substance use had higher reactive physical aggression scores in early adolescence. Maternal substance use in pregnancy was also positively related to early childhood environmental risk and parent aggression, suggesting that these may be mechanisms through which prenatal substance use may exert subsequent influence. This is consistent with prior research that has

found that illicit substance use such as cocaine is also associated with a higher risk for violence and exposure to violence (Eiden et al., 1999) and results are similar to previous samples selected for high rates of maternal cocaine and other substance use in pregnancy (e.g., Min et al., 2018). Moreover, mothers who engage in substance use in pregnancy exhibit more antisocial behavior and may confer both genetic risk and behavioral risk, by socializing their children to be more aggressive (Ruisch, Dietrich, Glennon, Buitelaar, & Hoekstra, 2018). Maternal substance use in pregnancy is a teratogenic risk factor for regulatory processes, such that these children have greater difficulty regulating their arousal in the context of stress (Schuetze, Eiden, & Danielewicz, 2009). Finally, these results suggest that the continuity of maternal substance use postpartum may also play a role, such that mothers had higher prenatal substance use may continue to use a higher level of substances postnatally, therefore exposing their children to more environmental risks (e.g., Parolin & Simonelli, 2016). These findings highlight the importance of considering early and prenatal risk factors when exploring trajectories to adolescent aggression.

Results also showed that placement in non-biological care predicted lower levels of reactive physical aggression in early adolescence. Non-biological parent care (foster or kinship) may serve as a protective factor by removing the child from a negative home environment or providing extended family support. This may be protective in this particular sample since the majority of the children entered non-biological care at birth and non-biological caregivers had lower levels of psychological risks (see Eiden, Foote, Schuetze, 2007).

Interestingly, early adolescent reactive relational aggression and middle childhood physical aggression, but not early adolescent reactive physical aggression, were related to violence victimization in early adolescence. Consistent with ecological theory, there is a transactional relation between a child and their community, such that the child's aggression may influence how they operate and their status within the broader community, which may then influence their behavior. The use of aggression may lower the social status of an individual within a community, putting them at risk for more serious victimization. Meta-analytic work has found that aggression is related to reduced peer status, such as less peer acceptance, increased peer rejection (Card, Stucky, Sawalani, & Little, 2008) and more detached relationships with teachers (Madill, Gest, & Rodkin, 2014). However, victimization and reactive aggression were assessed concurrently so directionality cannot be inferred from the current study.

Limitations and Future Directions

This study had several limitations. First, the sample was selected for maternal cocaine and other substance use in pregnancy and many of the control group mothers used substances other than cocaine. It is possible that the parameter estimates are not generalizable to non-substance using mothers and their families. Second, a few of our measures were broad, such as our measure of maternal exposure to violence, which did not uniquely refer to the home context. Child reactive aggression and violence-victimization were all measured concurrently in early adolescence, thus limiting conclusions regarding causality. Finally, given the limited sample size, we were unable to examine whether non-biological care status served as a moderator.

Future research should continue to study relations between harsh and power assertive parenting and child aggression within diverse contexts. It is also likely that other factors not currently examined (e.g., peer influences, temperament, autonomic regulation) predict reactive aggression. Although the family context is critical, in early adolescence peer relations become increasingly important (Nickerson & Nagle, 2005), and the selection and influence of peers contribute to aggression (Sijtsema et al., 2010). Future research should test models accounting for both family and peer interactions and processes. Finally, prospective models may mitigate the limitation of the concurrent measurement of reactive aggression and violence-victimization.

Conclusions

Consistent with a developmental cascade model, the current study found evidence for the effect of prenatal and early family risk on reactive physical and relational aggression. Early childhood environmental risk, and child physical aggression predicted maternal power assertive discipline in middle childhood, which successively predicted reactive relational aggression. Additionally, there were direct effects from prenatal substance use and non-biological parent care to early adolescent reactive physical aggression. In turn, early adolescent reactive relational aggression and middle childhood physical aggression predicted violence victimization. Results suggest that practitioners should be aware of the complex bidirectional interactions between child aggression and parenting behavior. Additionally, practitioners may benefit from taking a family systems perspective to children's aggression and exposure to violence, as many family factors contribute to the development of the child's behavior from prenatal factors to the current family environment. Overall, results underscore the transactional nature of the early family environment on children's later aggression and victimization experiences.

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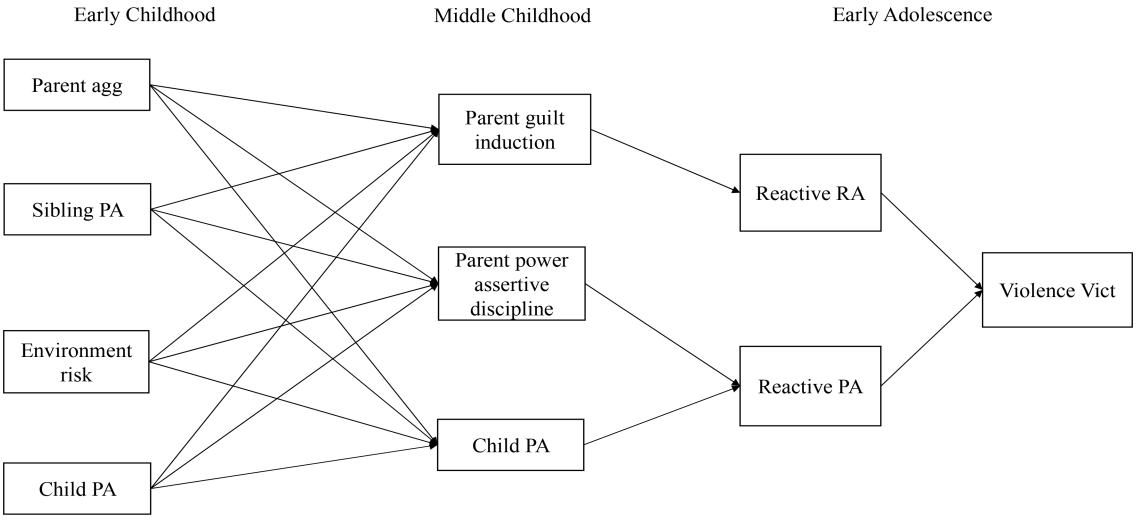

Figure 1.

Hypothesized developmental cascade structural model. This figure illustrates a developmental cascade model of early adolescent aggression and violence victimization. agg = aggression, PA = physical aggression, RA = relational aggression, Vict = victimization. Within time point covariances were also included in the model but are not shown for ease of interpretation. Maternal substance use in pregnancy, non-biological caregiver status, maternal children (i.e., the number of children the mother has when the child is one month old), SES adversity, and proactive aggression were controlled.

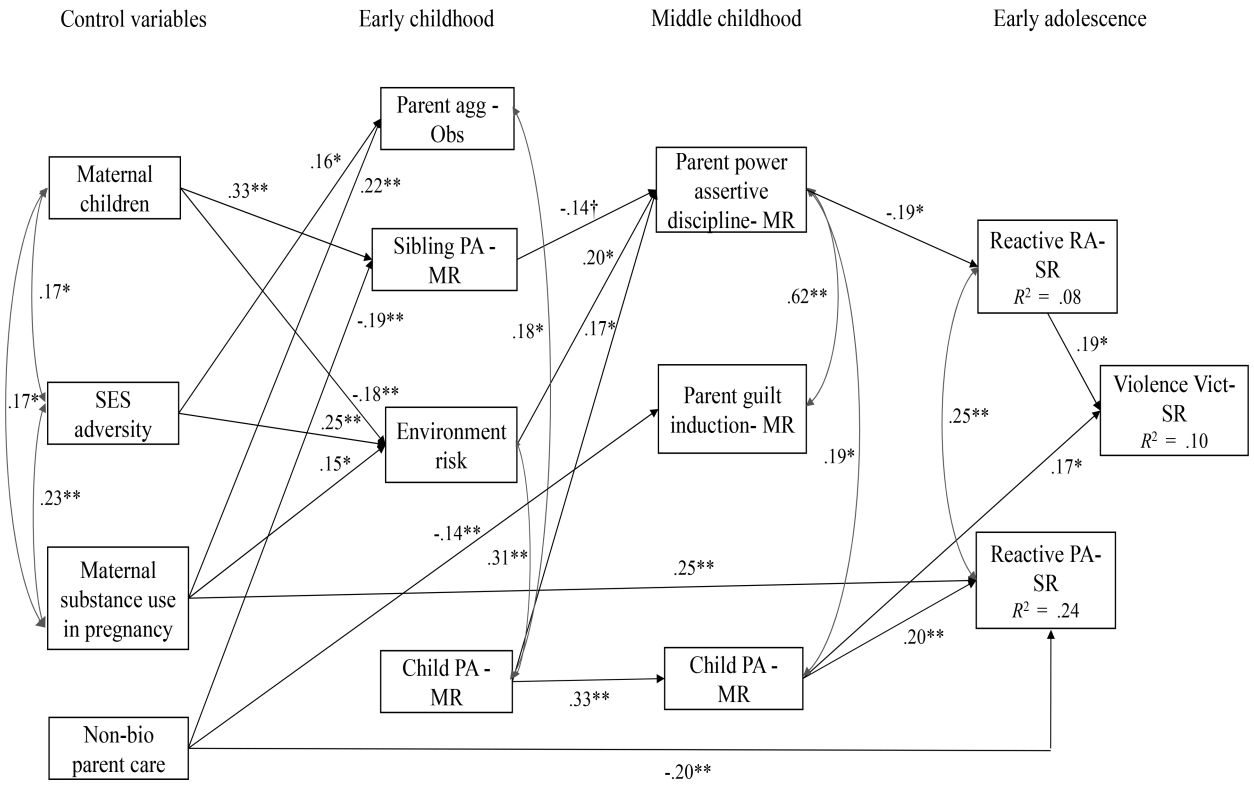

Figure 2.

Final model with significant standardized paths shown. This figure illustrates significant standardized path estimates for the final model. ** $p < .01$, * $p < .05$, † $p < .10$. *a*gg = aggression, bio = biological, MR = maternal report, Obs = observations, PA = physical aggression, RA = relational aggression, SES = Socioeconomic status, SR = self-report, Vict = victimization. Only significant paths are shown. Proactive aggression is not shown but is controlled for, was regressed on the control variables, and was significantly related to future reactive physical and relational aggression (*ps* < .001). Non-bio parent care (i.e., whether or not the child remained in the care of a biological parent) was controlled for at every time point in all models. Maternal children refers to the number of children a mother has at the 1 month assessment.

Table 1

Descriptive Statistics and Correlations of Key Study Variables

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.
1. T1 Parent Agg – OBS	—									
2. T1 Sibling PA – MR	− .06	—								
3. T1 Env risk	− .004	.07	—							
4. T1 Child PA – MR	.20**	.11	.33**	—						
5. T2 Parent guilt induction – MR	.05	− .003	.15*	.18*	—					
6. T2 Power assertive discipline – MR	.08	− .11	.24**	.23**	.64**	—				
7. T2 Child PA – MR	.13	.08	.19*	.37**	.16*	.25**	—			
8. T3 Reactive RA – SR	− .09	.06	− .04	− .08	− .04	− .09	.02	—		
9. T3 Reactive PA – SR	.10	.09	.13	.06	.11	.04	.21*	.30**	—	
10. T3 Violence victimization – SR	.05	− .07	.11	.09	− .03	.08	.19*	.22**	.20*	—
M	1.55	5.47	3.87	14.75	1.83	2.33	0.79	0.08	0.07	7.56
SD	1.12	3.51	2.79	2.92	0.92	1.01	0.86	0.14	0.13	6.05
Range	0 – 4.00	0 – 16.13	0 –12.00	5.61 – 20	1 – 5.01	1 – 5.39	0 – 3.00	0 – .52	0 – .51	0 –23.31