Longitudinal examination of pathways to peer problems in middle childhood: a siblings-reared-apart design

Article (Accepted Version)

Longitudinal Examination of Pathways to Peer Problems in Middle Childhood: A Siblings-Reared-Apart Design

Leslie D. Leve[1]

Amanda M. Griffin[1]

Misaki N. Natsuaki[2]

Gordon T. Harold[3]

Jenae M. Neiderhiser[4]

Jody M. Ganiban[5]

Daniel S. Shaw[6]

David Reiss[7]

[1]Prevention Science Institute, University of Oregon, Eugene, USA

[2]University of California, Riverside, CA, USA

[3]School of Psychology, Andrew and Virginia Rudd Centre for Adoption Research and Practice, University of Sussex, Brighton, UK

[4]Department of Psychology, The Pennsylvania State University, University Park, PA, USA

[5]Department of Psychology, George Washington University, Washington DC, USA

[6]University of Pittsburgh, Pittsburgh, PA, USA

[7]Yale Child Study Center, New Haven, CT, USA

Corresponding author: Leslie Leve, leve@uoregon.edu, 1600 Millrace Drive, Suite 105 Eugene, OR 97403

Acknowledgments

This project was supported by grants R01 HD042608 from the National Institute of Child Health and Human Development and the National Institute on Drug Abuse (PI Years 1–5: Reiss; PI Years 6–10: Leve), UG3 OD023389 and UH3 OD023389 from the Office of the Director (PIs: Leve, Neiderhiser, Ganiban), R01 MH092118 from the National Institute of Mental Health (PIs: Neiderhiser, Leve), R01 DA020585 from the National Institute on Drug Abuse (PI: Neiderhiser), and R01 DA035062 from the National Institute on Drug Abuse (PI: Leve). Amanda Griffin's work was supported by a training grant (F32 HD093347A). The content is solely the responsibility of the authors and does not necessarily represent the official views of the Eunice Kennedy Shriver National Institute of Child Health and Human Development or the National Institutes of Health.

Longitudinal Examination of Pathways to Peer Problems in Middle Childhood: A Siblings-Reared-Apart Design

Abstract

To advance research from Dishion and others on associations between parenting and peer problems across childhood, we used a sample of 173 sibling pairs reared apart since birth (because of adoption of one of the siblings) to examine associations between parental hostility and children's peer problems when children were ages 7 and 9.5 years (n = 326 children). We extended conventional cross-lagged parent–peer models by incorporating child inhibitory control as an additional predictor and examining genetic contributions via birth mother psychopathology. Path models indicated a cross-lagged association from parental hostility to later peer problems. When child inhibitory control was included, birth mother internalizing symptoms were associated with poorer child inhibitory control, which was associated with more parental hostility and peer problems. The cross-lagged paths from parental hostility to peer problems were no longer significant in the full model. Multigroup analyses revealed that the path from birth mother internalizing symptoms to child inhibitory control was significantly higher for birth parent–reared children, indicating the possible contribution of passive gene–environment correlation to this association. Exploratory analyses suggested that each child's unique rearing context contributed to their inhibitory control and peer behavior. Implications for the development of evidence-based interventions are discussed.

Keywords: parental hostility, peer problems, inhibitory control, adoption design, siblings

A large, extant body of research demonstrates concurrent and longitudinal associations from parental hostility to children's peer problems during late childhood and adolescence. Emanating from a significant body of work by Dishion and colleagues in the 1980s and 1990s that examined connections between coercive family processes and children's affiliation with antisocial peers, this research has shown that coercive interactions that begin in the family can spill over to the peer context, predicting negative or deviant peer interactions (e.g., Ary, Duncan, Duncan, & Hops, 1999; Dishion, Duncan, Eddy, Fagot, & Fetrow, 1994; Dishion, Patterson, Stoolmiller, & Skinner, 1991; Patterson & Dishion, 1985; Patterson, Reid, & Dishion, 1992; Snyder, Dishion, & Patterson, 1986). We sought to extend the work of Dishion and others by examining links between parenting and peer relationships prior to adolescence, and by more fully examining the role of child characteristics, particularly those that may be genetically influenced. To do so, we examined longitudinal associations between parental hostility and children's peer problems during middle childhood in a unique sample of genetically related sibling pairs who had been reared apart since birth.

A Multilevel Framework for Examining Pathways to Peer Problems in Middle Childhood

The conceptual model that guided our study integrates multiple conceptual frameworks to further the understanding of the mechanisms underlying the development of peer problems in middle childhood, including the conflux of individual and family predictors. As described by Cicchetti and colleagues, an individual's developmental trajectory is influenced by a range of socioemotional, genetic, familial, and societal influences that are layered and transactional (Cicchetti, 2016; Cicchetti & Lynch, 1993; Cicchetti & Toth, 2016; Masten & Cicchetti, 2010). A multilevel framework can be useful to better understand the conflux of individual and family predictors, and inform targets for preventive interventions.

.

One conceptual framework that is foundational to understanding connections between parent and peer processes is coercion theory (Patterson, 1982). It posits a social-interactional model of development wherein challenging child behavior, disrupted parenting, and coercive parent–child interactions that begin in early childhood (Shaw & Bell, 1993), and continue in middle childhood, reinforce and maintain children's problem behavior, escalating negative parent-child interactions within the family over time. Children initially experience coercive patterns in the home with parents and siblings, due in part to their own challenging behaviors (e.g., whining, hitting, non-compliance) and parents' attempts to stop such behaviors. Children then "practice" similar coercive interactional styles outside of the home, in a cycle of "deviancy training." Negative interactions with deviant peers during early adolescence are then posited to shape new and more serious forms of problem behavior (e.g., substance use, antisocial behavior, high-risk sexual behavior) (Dishion, Patterson, & Griesler, 1994; Patterson, Forgatch, Yoerger, & Stoolmiller, 1998). A core assumption of coercion theory is that, in response to challenging child behaviors, disrupted parenting behaviors, such as more hostile and less warm parenting, escalate child behavior problems over time that then spill over into the peer realm. Theoretically, parental hostility is reinforced after repeated attempts to shape the child's behaviors are met with child compliance. This coercive process in the home serves to model hostile interactions as appropriate behavior for the child, which are then played out in the child's interactions with peers outside of the home.

In addition to the parent–child social-interactional pathways from coercion theory that are hypothesized to lead to negative peer behavior, individual child characteristics are also relevant to the development of peer problems. Beginning with Bell's seminal work on child effects on

socialization processes (Bell, 1968, 1979, 2017; Bell & Chapman, 1986; Shaw & Bell, 1993), a large and growing body of research has shown that not only do parents socialize their children, but children play an active role in influencing their social context and parenting environments. Support for Bell's model of reciprocal effects comes from longitudinal studies that have examined child and parent behavior over time and found, for example, bidirectional effects between child antisocial behavior and parental negativity from child age 4 to 7 years (Larsson, Viding, Rijsdijk, & Plomin, 2008), bidirectional effects from conduct problems to changes in parenting behavior from child age 6 to 16 (Pardini, Fite, & Burke, 2008), and bidirectional effects between children's antisocial behavior and parents' depressive symptoms from childhood to adolescence (Gross, Shaw, Burwell, & Nagin, 2009). In addition, as reviewed in greater detail later in this article, researchers have used genetically informed study designs to illustrate how variation in child behavior that is genetic in origin can influence environmental factors, such as parenting behaviors (Plomin, DeFries, & Loehlin, 1977; Neiderhiser, Reiss, Hetherington, & Plomin, 1999; Scarr & McCartney, 1983). Thus, in addition to a focus on parenting behaviors as a predictor of peer problems, our study included a measure of children's inhibitory control as a child characteristic that is genetically influenced (Polderman et al., 2009) and may predict hostile parenting and peer problems.

Prediction of Peer Problems: Parental Hostility

A large body of evidence indicates that parental hostility is associated with children's problematic peer relationships (Brown & Bakken, 2011). The vast majority of this research has been conducted with children age 9 and older and has reliably found concurrent and prospective associations between disrupted parenting (e.g., hostile discipline, poor monitoring, low level of warmth) and peer problems (e.g., affiliation with delinquent peers, peer rejection, poor peer

social skills; Allen, Hauser, O'Connor, & Bell, 2002; Ary et al., 1999; Bank, Burraston, & Snyder, 2004; Benson & Buehler, 2012; Brody et al., 2001; Dishion, 1990; Dishion et al., 1991; Patterson & Dishion, 1985).

Although substantially less research has been conducted on the prediction of peer problems from hostile parenting in children younger than age 9, a small number of studies has found a similar pattern of associations during middle childhood. For example, in a community sample of children followed longitudinally from ages 5 to 9, researchers found that poor parental discipline (e.g., hostile parenting) exacerbated the association between peer delinquency and later child antisocial behavior, suggesting that hostile parenting increased the sensitivity of children to the effects of peer delinquency (Snyder et al., 2010). Another school-based sample of children ages 6–10 found that children's coercive interactions with their parents were associated with their antisocial behavior with peers (Dishion, Duncan, et al., 1994). Two rigorous intervention studies provided additional support for the association between hostile parenting and peer problems in middle childhood. First, Forgatch and colleagues (2016) examined 6-year follow-up data from a preventive intervention study of divorced mothers and their 5- to 8-year-old sons. They found a significant intervention effect on changes in parenting (more-effective discipline), which was then associated with a reduction in children's affiliation with delinquent peers. Second, Chang and colleagues (2017) showed intervention effects on positive parent–child interaction during early childhood (ages 3–5) that led to greater peer acceptance and less peer rejection at ages 7–10, via increases in children's effortful control. In other words, the intervention indirectly predicted peer preference by sequentially improving parent–child interactions and children's effortful control. Although the body of work conducted among children in middle childhood is substantially smaller than that conducted in late childhood and

adolescence, it provides evidence for the link between hostile parenting and peer problems and suggests that it is modifiable during middle childhood.

Prediction of Peer Problems: Low Child Inhibitory Control

There is also evidence that specific child characteristics are predictive of peer problems during middle childhood. In particular, children who have more difficulty controlling their own behavior or inhibiting impulses may be more likely to have difficulty establishing prosocial peer relationships. Eisenberg has proposed that inhibitory control deficits can affect peer relations by undermining children's social competence, as indexed by prosocial peer interactions and behavioral problems (Eisenberg, Hofer, & Vaughan, 2007; Eisenberg et al., 2005). In this model, *inhibitory control* is defined as the ability to willfully inhibit a prepotent behavior, especially when a child prefers not to do so but should, to adapt to the context or to achieve a goal (Rothbart & Bates, 2006). Inhibitory control can facilitate children's ability to engage in prosocial interactions with peers, such as sharing an attractive toy or game, or turn-taking.

Conversely, low inhibitory control may reduce children's ability to cooperate, share, and turn-take, particularly when it means putting others' needs and interests ahead of one's own immediate interests. The association between poor inhibitory control and later behavior problems is well documented (Nigg & Huang-Pollack, 2003), with lower levels of inhibitory control predicting behavior problems in early and middle childhood (Eisenberg et al., 2005; Kochanska & Knaack, 2003; Olson, Sameroff, Kerr, Lopez, & Wellman, 2005). Poor inhibitory control also appears to moderate the association between affiliation with delinquent peers and later antisocial behavior, in that youth with lower levels of inhibitory control are more susceptible to the negative influence of delinquent peers (Dishion & Patterson, 2006; Gardner, Dishion, & Connell, 2008). In a study of 8- to 10-year-old boys, Trentacosta and Shaw (2009) found a direct

association between children's inability to actively distract themselves during a temptation task (indicating lower inhibitory control) and peer rejection, suggesting that higher inhibitory control skills may protect children from developing peer problems. This pattern of findings was replicated in a large longitudinal study with a national sample of children studied from age 4.5 to age 15 that found that better inhibitory control skills reduced the likelihood of experiencing peer problems later in childhood and in middle adolescence (Holmes, Kim-Spoon, & Deater-Deckard, 2016).

Children's inhibitory control skills have also been shown to be a partial mediator of the association between parenting and children's later social competence (Spinrad et al., 2007), highlighting the connection between inhibitory control and parenting and peer relationships. Findings from a meta-analysis revealed concurrent associations between children's self-regulation (including inhibitory control), measured via observation and questionnaire, and more positive, less negative parenting behavior, also measured via observation and questionnaire (Karreman, Van Tuijl, van Aken, & Deković, 2006). Longitudinal studies have demonstrated that inhibitory control in middle childhood is predictive of decreases in maternal rejection (Lengua, 2006), even when earlier parenting is controlled for. This finding is consistent with those of the child effects models of development, with studies showing that children's inhibitory control deficits can elicit hostile parenting (Bates, Schermerhorn, & Petersen, 2012), ultimately leading to poor peer relations (Holmes et al., 2016; Kiff, Lengua, & Zalewski, 2011). Our study extended this line of research through the use of a siblings-reared-apart design, to test whether the pattern of associations between child inhibitory control, hostile parenting, and peer problems has heritable origins. For reasons discussed in the next two sections, our study's innovative genetically informed design facilitated an investigation of whether heritable child characteristics

are, in part, responsible for evoking hostile parenting and peer problems, and also whether genes shared between family members who are living together contribute to the magnitude of associations between inhibitory control, harsh parenting, and peer problems (passive gene–environment correlation, discussed later in this article).

Genetic Influences on Inhibitory Control, Hostile Parenting, and Peer Problems

As noted earlier, the multilevel framework applied in our study also incorporates the role of genetic influences on development and parent–peer processes. We considered whether there are direct genetic influences on child behavior, including children's inhibitory control and children's behavior with their peers. There is clear evidence that inhibitory control is influenced, in part, by genetic factors; findings from a study of 9-year-old twins reported that genetic factors accounted for 39% of the total variance for inhibitory control (Polderman et al., 2009) and similar findings were found for a sample of 2-year-old twin pairs (38% of the variance was due to genetic factors; Gagne & Saudino, 2016). A meta-analysis of 58 independent samples from twin, family, and adoption studies estimated that about half of the total variance was accounted for by genetic influences on impulsivity, with individual-specific environmental influences accounting for the remaining variance (shared environmental influences were not significant in the meta-analysis; Bezdjian, Baker, & Tuvblad, 2011).

A handful of genetically informed studies has also examined genetic influences on peer relationships during middle childhood, and found that both genetic and nonshared environmental influences accounted for individual differences in peer difficulties for children ages 5 to 7 years (Boivin et al., 2013). Similarly, genetic and nonshared environmental influences account for individual differences in peer delinquency (Beaver, Wright, & DeLisi, 2008; Bullock, Deater-Deckard, & Leve, 2006; Iervolino et al., 2002) and peer interaction (Pike & Atzaba-Poria, 2003)

during late childhood and adolescence.

In addition to the direct contribution of genetic factors to children's inhibitory control and peer problems, it is likely that child behaviors that are genetically influenced may *evoke* more harsh negative responses from parents and peers. That is, a child's behavior may evoke a particular behavioral response from others in the environment and this response can be explained, at least in part, by the child's genotype (Plomin et al., 1977; Scarr & McCartney, 1983), a process known as *evocative gene–environment correlation*. For example, a child with a genetically influenced predisposition towards lower inhibitory control may be more likely to evoke harsh parenting from a parent over time and more negative peer behavior than a child with a genetic predisposition to higher inhibitory control. There is ample evidence for the presence of evocative gene–environment correlation with regard to parenting behavior, particularly during adolescence (Ayoub et al., 2018; Ge et al., 1996; Klahr & Burt, 2014; Knafo & Jaffee, 2013; Narusyte et al., 2008; Neiderhiser, Reiss, Hetherington, & Plomin, 1999; O'Connor, Deater-Deckard, Fulker, Rutter, & Plomin, 1998; Reiss, Neiderhiser, Hetherington, & Plomin, 2000).

Specific to middle childhood, a study of adoptees ages 7–12 found that children at genetic risk based on the antisocial behavior of their birth mothers were more likely to receive negative parenting from their adoptive parents than were children not at genetic risk (O'Connor et al., 1998). However, this study also showed that most of the association between negative parenting and children's externalizing behavior was not explained by evocative gene–environment correlation and that additional environmentally mediated parenting effects on children's behavior were plausible. There is also some evidence for evocative gene–environment correlation with regard to children's peer relationships. For example, a study that included some of the same participants as in our study examined children's disruptive peer behavior at age 4.5

and found that heritable influences on children's low social responsivity to parenting attempts predicted adoptive mother–child and father–child hostility, which then indirectly predicted children's later disruptive behavior with peers (Elam et al., 2014). This finding suggests that evocative gene–environment correlation via child effects on hostile parenting is one pathway to peer problems earlier in development. Although additional evidence for evocative gene–environment correlation with regard to peer problems has been found in later adolescence and young adulthood (e.g., Brendgen, 2012; Burt, McGue, & Iacono, 2009; Manke et al., 1995; Tarantino et al., 2014), research on this topic during middle childhood is quite sparse. In our study, we sought to fill this gap.

The Sibling-Adoption Design

Our study combined data from a study of children adopted at birth by genetically unrelated parents with data from a study of the adoptees' biological siblings who remained in the care of the birth mother. In the adoption sample, because the adoptees were genetically unrelated to their rearing parents, associations between a parenting behavior and a child behavior could not be attributed to shared genes, commonly known as *passive gene–environment correlation.* Conversely, the biological siblings were reared by their birth parent(s), and thus children shared 50% of their genes with their rearing parent(s). By including both adoptees and biological siblings in a single study and including parenting data from their respective rearing parents (i.e., adoptive parents or birth parents), we could examine the possible role of passive gene–environment correlation underlying associations between a child behavior and a parent behavior. An additional feature of the adoption design portion of this study was the ability to trace evocative gene–environment correlation via a characteristic in birth parents, to a child characteristic, to an evoked effect on a parenting variable. Finally, a third novel feature of the

sibling-adoption design was the ability to examine the behavioral similarity of biological sibling pairs (i.e., siblings who share at least one biological parent) who are reared apart. If biological siblings who are reared apart resemble one another on a given behavior, then genetic factors are a valid explanation for this similarity, particularly if full biological siblings reared apart resemble one another more than do half biological siblings reared apart. If biological siblings reared apart differ from one another, the role of the family and contextual environment are implicated as important contributors to the unique sibling behavior.

Consistent with the multilevel analytical approach in this special issue, we used longitudinal data (two time points) from the sibling-adoption design to test three primary hypotheses and one exploratory hypothesis related to the development of peer problems in middle childhood. First, we tested a conventional cross-lagged model of the paths between hostile parenting and peer problems and hypothesized that the cross-lagged path from parenting to later peer problems that has been rigorously documented in studies of older youth would be detectable in middle childhood. In doing so, we attempted a replication of many studies, not informed by genetic designs, reviewed earlier in this manuscript. Second, extending many previous analyses focusing on parenting effects, we hypothesized that when child inhibitory control is added to the conventional cross-lagged model, it would predict later parental hostility and peer problems. After accounting for the effects of parenting and peers, we further hypothesized that inhibitory control would be genetically influenced and would mediate associations between birth parent psychopathology and later parent hostility and peer problems. Third, as adoptees were not genetically related to their rearing parents (the adoptive parents) and the biological siblings were genetically related to their rearing parents (the birth parents), we hypothesized that the paths from birth mother psychopathology to child inhibitory control would

be stronger in birth homes than in adoptive homes. Fourth, to augment the multilevel analytic approach used in our study, we tested an exploratory fourth hypothesis using within-sibling pair analyses that examined sibling similarity. Specifically, because previous research has identified genetic contributions to inhibitory control, peer problems, and parental hostility, we explored if the magnitude of cross-sibling correlations for these constructs depended on the siblings' genetic relatedness. We hypothesized that the biological siblings reared apart would be correlated on their inhibitory control and peer problem behavior scores, and that cross-sibling correlations would be greater for full biological siblings than for half siblings, indicating genetic influences on these constructs.

Method

Participants and Procedures

Study participants were a subsample of linked adoptive and birth families (N = 135 linked families, with 173 linked sibling sets) from two related studies: the Early Growth and Development Study (EGDS), which is a prospective parent–child adoption study (Leve et al., 2013), and its companion study, Early Parenting of Children (EPoCh), which includes siblings of the EGDS adoptees who are reared by the birth mother (Leve et al., 2018). The full EGDS sample consists of 561 linked sets of adoptive families (adopted child, adoptive parents, and birth parents) that were recruited through 45 adoption agencies from 15 states throughout the United States. Eligibility criteria for participation in the original EGDS study included (a) the adoption placement was domestic, (b) the infant was adopted within 3 months of birth (M = 6.20 days, SD = 12.45 days), (c) the infant was placed with a nonrelative, (d) both the birth parents and adoptive parents had at least an eighth grade education, and (e) the infant did not have any major medical conditions. The EGDS participants were recruited shortly after the birth of the adoptee

and followed longitudinally until early adolescence.

The full EPoCh sample consists of 216 biological children living with their birth family. The EPoCH eligibility criteria included (a) birth mother enrolled in EGDS between 2003 and 2009 following the birth of the EGDS adoptee, (b) birth mother was parenting a biological sibling of an EGDS adoptee, (c) this biological sibling was born between 2005 and 2012. The EPoCh participants were recruited at biological sibling age 7 and assessed one additional time in late childhood. All EGDS and EPoCh families in our study were assessed with in-person and web-based interviews at child age 7 (T1) and via web-based or phone interview at the subsequent assessment approximately 2.5 years later (T2). Assessments covered a wide range of topics, including parenting, child psychosocial behavior, peer relationships, and cognitive development.

Our study focused on a subsample of EGDS/EPoCh sibling pairs reared apart who completed a follow-up phone assessment during 2014–2018 as part of separate, follow-up studies. This resulted in 135 linked adoptive–birth families that included adopted children living with their adoptive parent(s) (n = 135 adoptees) and their biological sibling(s) living with their birth families (n = 191 biological siblings), resulting in a total of 326 children (50% males). The sample size of biological siblings was larger than that of adoptees because 38 birth parents were parenting multiple children who were genetically related to the adoptee. The sample thus included 173 linked sibling pairs and 18 additional biological children who were missing the linked adoptive home data but had full data on the linked birth parent and biological sibling (see Table 1). Of the linked sibling pairs, there were 32 pairs of full siblings and 141 pairs of half siblings; 94 sibling pairs were the same sex and 79 pairs were opposite-sex. Data were collected from 135 adoptive mothers, 118 adoptive fathers, 138 birth mothers, and 118 birth fathers. In the adoptive homes, 10 sets of parents were same-sex couples (two male–male couples and eight

female–female couples). The adoptee and the linked biological siblings were assessed at age 7 (T1: *M* = 7.26; *SD* = 0.61) and age 9.5 (T2: *M* = 9.69; *SD* = 1.59).

In the analytic sample of families in our study, the median total annual household income at T1 for adoptive families was between $100,001 and $125,000. The median educational attainment for adoptive mothers and adoptive fathers was least a 4-year college degree. Most adoptive mothers and adoptive fathers were Caucasian (91.3% and 90.6%, respectively); others were African American (2.9% and 0.9%, respectively), Latinx (1.4% and 2.6%, respectively), more than one race/ethnicity (2.2% and 0.9%, respectively), and other/unknow (2.1% and 4.3%, respectively). Adoptive mothers and adoptive fathers were *M* = 44.01 (*SD* = 5.79) and *M* = 45.04 (*SD* = 5.77) years old, respectively, at the T1 assessment.

For birth families, the median total annual household income at T1 was $25,001–$40,000. The median educational attainment was a high school diploma for birth mothers and birth fathers. The majority of birth mothers and birth fathers were Caucasian (64.2% and 58.1.5%, respectively); others were African American (20.9% and 25.7%, respectively), Latinx (9% and 4.1%, respectively), more than one race/ethnicity (3% and 10.8%, respectively), and other/unknown (2.9% and 1.4%, respectively). Birth mothers and birth fathers were *M* = 32.78 (*SD* = 4.97) and *M* = 35.51 (*SD* = 7.25) years old at T1, respectively. The T1 assessment was conducted in the home and via web-based questionnaires, and the T2 assessment was conducted by phone and web questionnaire. All research activities were approved by the institutional review boards of the participating institutions. All adult participants provided consent and all children provided assent to participate in the research activities.

Measures

Inhibitory control. Child inhibitory control was assessed for adoptees and biological

siblings at T1 with the 13-item Inhibitory Control scale from the short form of the Children's Behavior Questionnaire (Putnam & Rothbart, 2006; Rothbart, Ahadi, Hershey, & Fisher, 2001). Items were rated by the child's rearing parent(s) on a 7-point Likert scale ranging from *extremely untrue of your child* to *extremely true of your child*. When data were available from both rearing parents (n = 164 children), a mean score of both parents was used (interparental r = .51, p < 001). The Inhibitory Control scale measures children's capacity to plan and to suppress inappropriate responses under instructions or in novel or uncertain situations. In our study, this measure was reverse coded so that higher scores indicated poorer inhibitory control. The scale demonstrated acceptable reliability across reporters (Cronbach's α = .76–.86).

Parental hostility. Parental hostility to the study child was assessed for adoptive and birth parents at T1 and T2 with the 5-item Hostility scale from the Iowa Family Interaction Scales (Melby & Conger, 2001). Items were rated by the rearing parent(s) on the 7-point Likert scale ranging from *always* to *never*, with higher scores indicating greater hostility. Items assessed the parent's hostility toward the child, for example, *how often did you criticize him/her or his/her ideas.* The scale demonstrated acceptable reliability in our study (Cronbach's α = .79–.95). Interparental ratings were correlated (T1: r = .35, p < .01; T2: r = .44, p < .01), thus when data were available from both parents (T1: n = 167 children, T2: n = 76 children), a mean score was used.

Child peer problems. At T1, adoptee and biological sibling peer problems were assessed using rearing parents' report on the Walker-McConnell Peer-Preferred Social Behavior Scale (PSBQ; Walker & McConnell, 1988). Intended to measure peer-related social behaviors that are highly valued by peers, the PSBQ is a 17-item parent report that ranges from 1 (*never*) to 5 (*frequently*). Example items include *offers to help peers when needed, makes friends easily*, and

plays games and activities skillfully. The scale demonstrated acceptable reliability in our study (Cronbach's α = .94–.95). There was high consistency in parental ratings (r = .71, p < .001), and thus when both parents completed the PSBQ (n = 165 children), a mean score was used. In our study, this measure was reverse coded so that higher scores indicated more peer problems/fewer peer skills.

At T2, rearing parents or the child responded to the Peer scale of the Strengths and Difficulties Questionnaire (SDQ; Goodman, 2001) using a 5-item scale ranging from 0 (*not true*) to 2 (*certainly true*). The SDQ Peer scale includes items such as *tends to play alone*, *has at least one good friend*, and *generally liked by other children*. The SDQ was administered to either the child (n = 118) or to a rearing parent (n =134), depending on the particular assessment protocols for the T2 assessment, with children born earlier in the course of the study completing the SDQ via child report and those born later having parent report data. Although in no case did a child have both parent-report and child-report data, the mean-level scores on the SDQ did not differ as a function of reporter (child report M = 2.01, SD = 1.83; parent report M = 1.94, SD = 1.89; t[250] = 0.29, p = .77). Although the study used different measures of peer problems at T1 (PSBQ) and T2 (SDQ), the two measures had somewhat comparable items, suggesting that they measured similar underlying constructs, and they were significantly correlated (r = .50, p < .001).

Genetic risk inherited from birth mothers. Birth mother psychopathology was used as a broadband indicator of genetic risk for child behavior problems (e.g., low inhibitory control, peer problems). It was measured using composite externalizing and internalizing scores previously created in this study (Marceau et al., in press). Four indicators relating to internalizing and externalizing problems, which took into account symptoms, age of onset, and family history,

were used to compute separate scores for birth mother internalizing and externalizing problems. The indicators were derived from birth mothers' self-report data 18 and 54 months after the birth of the adoptee. For the first three indicators, the Composite International Diagnostic Instrument (Kessler & Ustun, 2004) and the Diagnostic Interview Schedule (Robins et al., 1981) were used to create birth mother counts for (a) number of disorders on the diagnostic interview, (b) number of symptoms endorsed on the diagnostic interview, and (c) age of onset of each disorder. For internalizing disorders, the diagnostic categories surveyed included major depression, brief recurrent depression, dysthymia, separation anxiety, adult separation anxiety, social phobia, agoraphobia (with and without panic), panic disorder, specific phobia, and generalized anxiety. For externalizing disorders, the diagnostic categories surveyed included antisocial personality, conduct disorder, alcohol abuse and dependence, drug abuse and dependence, and tobacco dependence. For both externalizing and internalizing scores, episodes of prenatal symptoms were excluded.

The fourth indicator comprised the number of first-degree relatives (mother, father, and up to three siblings) who had ever been diagnosed with an internalizing or externalizing disorder, assessed by birth mother reports for each relative, derived from the Family History-Research Diagnostic Criteria (Andreasen, Endicott, Spitzer, & Winokur, 1977). This indicator was calculated as the maximum proportion of first-degree relatives rated by the birth mother as having externalizing or internalizing problems. The composite scores for internalizing and externalizing problems were intended to be more robust measures of inherited psychopathology than any single measure, as internalizing and externalizing disorders load highly onto separate liability factors and show strong genetic overlap in adulthood (Caspi et al., 2014; Kendler et al., 2011; Krueger & Markon, 2006; Waszczuk, Zavos, Gregory, & Eley, 2014). Indicators were

entered into a principal component analysis separately for each disorder, and extracted factor scores were aggregated to create a composite score for internalizing symptoms and for externalizing symptoms.

Covariate: household income. Self-reported annual household income in adoptive and birth homes was assessed at T1 on an 11-point Likert scale: 1 = less than $15, 000; 2 = $15,001–$25,000; 3 = $25,001–$40,000; 4 = $40,001–$55,000; 5 = $55,001–$70,000; 6 = $70,001–$100,000; 7 = $100,001–$125,000; 8 = $125,001–$150,000; 9 = $150,001–$200,000; 10 = $200,001–$300,000; and 11 = more than $300,000. None of the correlations between household income and the independent variables (birth mother externalizing composite, birth mother internalizing composite, T1 parental hostility, T1 peer problems, T1 child inhibitory control) was significant. As expected, income differed significantly by home type, with higher income levels in adoptive homes than in birth homes (adoptive home $M = 6.82$, $SD = 2.12$; birth parent home $M = 2.83$, $SD = 1.70$; $t[303] = 18.16$, $p < .01$). Models accounted for differences between adoptive and birth parent household income by regressing each study variable on income and using the standardized residuals in subsequent analyses.

In addition, we examined whether there were differences in study variables by child sex. Child sex was not significantly correlated with any of the study variables and there were no mean-level difference by sex, and thus child sex was not considered further. Means and standard deviations for all study variables are presented in Table 1, and the correlation matrix is shown in Table 2.

[INSERT TABLES 1 & 2 HERE]

Analytic Plan

Analyses were conducted using the Mplus 6.1 software (Muthén & Muthén, 1998–2010).

Model 1 tested a model that examined the cross-lagged paths between parental hostility and peer problem pathways at T1 and T2 (Hypothesis 1), including the potential for genetic effects via birth parent internalizing and externalizing symptoms on T1 parental hostility and T1 peer problems. Model 2 added child inhibitory control to the model as a child characteristic that may genetically influenced, in part, and was hypothesized to be associated with later parental hostility and peer problems. This allowed us to examine Hypothesis 2: (a) the association between children's genetic risk for behavior problems (birth mother externalizing and internalizing symptoms) and poor inhibitory control, (b) the longitudinal paths from child poor inhibitory control to T2 parent hostility and T2 peer problems, and (c) the indirect effect of children's genetic risk for behavior problems on T2 outcomes via child inhibitory control. Model 3 then tested the invariance between adoptive and birth homes for path estimates to examine Hypothesis 3, that the association between birth parent characteristics and child inhibitory control would be stronger in the biological sibling group than in the adoptee group.

Fit was assessed using comparative fit index (CFI), Tucker-Lewis Index (TLI), root mean square error of approximation (RMSEA), and maximum likelihood and adjusted Satorra-Benter chi-square values (Satorra & Bentler, 1994). The CFI and TLI range in value from 0 to 1 indicates the proportion of improvement in the overall fit of the hypothesized model relative to a null model in which all covariances between variables are zero. Values of .95 or greater are desirable for the CFI and TLI (Bentler, 1990; Hu & Bentler, 1999). The RMSEA is a measure of lack of fit per degrees of freedom; values that range upwards from 0 through to 0.05 indicate a good fit, and up to 0.08, a fair fit (Browne & Cudeck, 1993). Because the chi-square is highly sensitive to sample size and distributional assumptions (Hu & Bentler, 1995), three other measures of the overall goodness-of-fit were used.

Following the model fitting, which examined between-family differences, we conducted exploratory analyses to examine within-sibling pair differences (Hypothesis 4). Specifically, we conducted a descriptive examination of the biological sibling pair correlations, where we correlated scores for the member of the sibling pair that was living in the adoptive home (adoptee) with scores for the child in the birth home (biological sibling). These intraclass correlations were computed for each adoptee-biological sibling pair, separately by the type of sibling (full siblings who share 50% of their segregating genes, on average, or half siblings who share 25% of their segregating genes, on average). If sibling correlations decrease with decreasing genetic similarity (e.g., full sibling correlations are larger than half-sibling correlations) then genetic influences are suggested. Model fitting was not conducted for the within-sibling pair analyses because of the exploratory nature of this hypothesis and the small sample size for full-sibling pairs.

Models were also examined with only one child per birth home, to account for multiple siblings in the same birth home. The pattern of associations remained the same, and thus the full sample was retained in the current study.

Results

Prior to analyses, we examined whether there were mean differences in the study variables based on rearing environment. Two significant differences were identified: children in birth homes had more peer problems than did children in adoptive homes at T1, $t(263) = 2.4$, $p = 0.01$, and adoptive parents reported more parental hostility than did birth parents at T2, $t(293) = 2.91$, $p = 0.004$; see Table 1. As shown in Table 2, after controlling for household income, most of the rearing-parent and child variables were significantly correlated with one another. Specifically, poor child inhibitory control ($r = 0.36$, $p < .01$), more parental hostility ($r = 0.56$, p

< .01), and more peer problems ($r = 0.22$, $p < .01$) at T1 were robustly correlated with more parental hostility at T2. In addition, poor child inhibitory control ($r = 0.37$, $p < .01$), more parental hostility ($r = 0.21$, $p < .01$), and more peer problems ($r = 0.49$, $p < .01$) at T1 were correlated with more peer problems at T2.

Next, we proceeded with modeling testing. Model 1, which tested the reciprocal cross-lagged paths between parental hostility and peer problems, is displayed in Figure 1. In addition to stability in parental hostility from T1 to T2 ($b = 0.54$, $p < .01$) and peer problems from T1 to T2 ($b = 0.46$, $p < .01$), T1 parental hostility was associated with T2 peer problems ($b = 0.12$, $p < .05$), consistent with Hypothesis 1. T1 peer problems were not associated with T2 parental hostility ($b = 0.10$, *ns*). Birth mother internalizing and externalizing symptoms were not directly associated with T1 parental hostility ($b = 0.08$, *ns*; $b = 0.08$, *ns*). However, birth mother internalizing symptoms were associated with T1 peer problems ($b = .15$, $p < .01$); externalizing symptoms were not ($b = 0.03$, *ns*).

[INSERT FIGURE 1 HERE]

Model 2 (shown in Figure 2) built on Model 1 by examining T1 child poor inhibitory control as a child characteristic that may be genetically influenced and predict parental hostility and peer problems at T2. Model 2 included the association between birth mother internalizing symptoms and T1 peer problems, but dropped nonsignificant birth mother externalizing and internalizing symptoms paths, accounting for their effect as correlations to present the most parsimonious model. In Model 2, birth mother internalizing symptoms were associated with T1 child inhibitory control ($b = 0.14$, $p < .05$), but birth mother externalizing symptoms were not ($b = −0.01$, *ns*). In addition, child inhibitory control predicted T2 parental warmth ($b = 0.16$, $p < .01$) and T2 peer problems ($b = 0.13$, $p < .05$). There was a significant indirect effect from birth

mother internalizing symptoms through T1 peer problems to T2 peer problems (b = .16, p < .01). In addition, consistent with Hypothesis 2, there was a trending indirect effect from birth mother internalizing symptoms through T1 inhibitory control to T2 parental hostility (b = 0.03, p = .08).

[INSERT FIGURE 2 HERE]

Model 3 presents group differences by adoptive and birth homes for paths estimated in Model 2. With the exception of the correlation between birth mother externalizing and internalizing symptoms, which was fixed to be the same across groups because the children had the same birth mother, all other paths were allowed to vary (shown in Figure 3). This model allows for a test of whether passive gene-environment correlation may be present, suggested when correlations for children reared in birth homes are larger than those for children reared in adoptive homes. Figure 3 shows that for differences in path estimates with regard to adoptive and birth homes, the magnitude of parameter estimates differed for birth mother internalizing symptoms to T1 peer problems, birth mother internalizing symptoms to T1 inhibitory control, T1 parental hostility to T2 parental hostility, T1 peer problems to T2 parental hostility, and T1 parental hostility to T2 peer problems (designated in bold in Figure 3). We used the adjusted Satorra-Bentler scaled chi-square difference test to examine invariance by constraining path coefficients to be equal/free to vary (shown in Table 3). There was a significant detriment to model fit when parameter estimates were constrained to be equal from birth mother internalizing symptoms to T1 inhibitory control (χ_{diff}^2 [1] = 7.08, p = .008). The magnitude of the parameter estimate for the path from birth mother internalizing symptoms to T1 inhibitory control was significantly higher in birth parent–reared homes (b = 0.28, p < .01) than in adoptive homes (b = -0.07, *ns*), indicating the possible presence of passive gene–environment correlation. In addition, there was significant detriment to model fit when parameter estimates were constrained to be

equal from T1 peer problems to T2 parental hostility (χ_{diff}^2 (1) = 4.67, p = .03). In this case, there was a stronger association between T1 peer problems and T2 parental hostility in adoptive homes, suggesting a stronger effect of the adoptee's peer problems on their rearing parents' hostility than of birth-reared children's peer problems on their rearing parents' hostility.

[INSERT TABLE 3 HERE]

[INSERT FIGURE 3 HERE]

To examine exploratory Hypothesis 4, we present the means, standard deviations (Table 1), and correlations (Table 4) of the paired-household dyads as part of exploratory analyses to test within–sibling pair differences. As shown in Table 4, there were no significant correlations between siblings reared apart with regard to their inhibitory control levels, parental hostility scores at T1 or T2, or peer problem scores at T1 or T2, regardless of full sibling or half-sibling status. Although the correlations were higher for full- versus half-sibling pairs in some cases, none of the correlations or correlation differences was significant.

[INSERT TABLE 4 HERE]

Discussion

Our study used data from a set of linked adoptee and biological sibling studies that, together, facilitated an innovative approach to examining the prediction of peer problems in middle childhood. In addition to testing a conventional cross-lagged model that examines the cross-over effects from parental hostility to later peer problems, we were able to examine child effects on these pathways. This included an examination of whether children's low inhibitory control predicted later peer problems and parental hostility, and if such pathways existed, whether they were, in part, the result of genetic influences on child inhibitory control. Further, because we could compare the path coefficients from birth mother internalizing and externalizing

problems to the adoptees' and biological siblings' inhibitory control, we were able to examine whether passive gene–environment correlations may be present in associations between birth parent psychopathology and child inhibitory control.

The primary findings from this study replicate and extend Dishion's foundational research describing how coercive family processes in the home can spill over into the peer realm (Dishion et al., 1991; Patterson & Dishion, 1985; Patterson et al., 1992; Snyder et al., 1986). Yet, the findings also pose new questions and challenges for future research and intervention activities focused on understanding and preventing the pathways to peer problems. First, consistent with our first hypothesis, results from the conventional cross-lagged model replicated findings from prior studies of adolescents showing that higher levels of parental hostility to the child are linked with an increase in children's peer problems later in development (e.g., Ary et al., 1999; Patterson, Reid, & Dishion, 1992). The downward extension of this finding to ages 7–9.5 years expands the evidence base on the applicability of Dishion's deviancy training model to younger children, and suggests that parenting interventions delivered during middle childhood may be well timed to prevent the spillover from coercive processes in the home to negative or delinquent interactions with peers. Of note, although the reverse path – from T1 peer problems to T2 parental hostility—was not significant in our test of the conventional cross-lagged model, it was also not significantly different than the T1 parental hostility to T2 peer problems path, suggesting that this pathway may also be worth attending to when considering family-peer process pathways.

When we added children's inhibitory control to the cross-lag model, it signficantly predicted later parental hostility and later peer problems. This finding provided support for one aspect of our second hypothesis—that low inhibitory control can impair children's abilities to

develop positive peer relationships and will tax parents' ability to refrain from hostile interactions with their child. The addition of child inhibitory control paths to the cross-lagged model also resulted in a change in significance for the initial cross-lagged path from parental hostility to peer problems; that is, it became nonsignificant. This was unexpected and may indicate that the previously identified and replicated pathway from hostile parenting to child peer problems may, in part, be confounded with child characteristics that were not measured in some of the prior studies. Child inhibitory control has been shown to mediate associations between maternal behavior (depression) and later child behavior (oppostional behavior) in other studies of young children (Choe et al., 2014).

We also examined the possible role of genetic influences on parent–peer pathways via genetic influences on children's inhibitory control. If an association was found from birth mother psychopathology to children's inhibitory control in the adoptee group, it would suggest a heritable association. Our results identified a significant association between birth mother internalizing problems and children's inhibitory control, supporting our second study hypothesis. However, when estimates from the subsequent multigroup analysis tested for invariance in this path coefficient between adoptees and biological siblings, the fit estimates indicated that the association between birth mother internalizing symptoms and child inhibitory control differed by group, with a stronger association in the biological sibling group than in the adoptee group. Although this supported our third hypothesis—that passive gene–environment correlation may explain a portion of the association between rearing parents' characteristics and a child's charactersitic—that the association between birth parent psychopathology and adoptee inhibitory control was not signficant suggests that we did not capture a heritable aspect of children's inhibitory control. One potential reason for this null finding is that our study relied on birth

mother data to ascertain genetic influences on child inhibitory control. This was because of the lack of birth father data for the vast majority of the sample, regardless of which rearing home the child resided in. In the absence of birth father data, only 50% of the possible genetic contributions to child characteristics can be modeled. Because prior twin studies (e.g., Polderman at el., 2009) have shown genetic influences on children's inhibitory control, it may also be that our birth parent mesaures (internalizing and externalizing symptoms) were not ideal for detecting an intergenerational genetic signal between parent and child.

Consistent with our second set of study hypotheses, there was partial support (trend level) for an indirect effect of birth mother internalizing symptoms on parental hostility, via lower levels of child inhibitory control. Considered together with the aforementioned result showing that the birth parent internalizing–child inhibitory control link was present only in the biological sibling group, this chain of associations from birth parent psychopathology, to child low inhibitory control, to hostile parenting may ultimately reflect an underlying shared genetic predisposition toward behavior that is maladaptive, rather than a causal association from child inhibitory control to parental hostility. This identification of a possible passive gene-environment correlation warrants additional research with larger sample sizes and more rigorous measurement, or intervention designs, to further examine this pattern of results.

The multigroup analysis also revealed an additional group difference: the path from age 7 peer problems to age 9.5 parental hostility differed between adoptees and biological siblings and was significant in the former but not the latter. This group difference was not anticipated, in part because when passive gene–environment correlation is present, it is expected to increase the similarities among biological family members. However, in our study, peer problems in adoptees were associated with later adoptive parent hostility, but this effect was not seen in the biological

siblings. In addition, examination of the mean-level differences (Table 1) indicated that birth parents rated the biological siblings as having more peer problems at age 7 than adoptive parents rated the adoptees as having, and yet by age 9.5, adoptive parents rated themselves as more hostile toward their child than birth parents rated themselves. Together, this set of findings may indicate that shifts in the level of peer problems as children progress through middle childhood can be taxing for parents. In the case of the adoptees, in which the mean levels of peer problems were very low at age 7 but of the same magnitude as those of the biological siblings at age 9.5, this increase may elicit more hostile parenting strategies from rearing parents, potentially as part of a normative shift in parenting that becomes necessary as children develop new skills and challenges as they enter new developmental periods. An alternative explantion is that perhaps biological parents in our study (who were younger and lower income than adoptive parents) perceived higher levels of peer problems as more normative than did adoptive parents, and thus such behaviors at age 9.5 were less taxing on their parenting. Additional follow-ups of this sample are planned when the children are adolescents, which will enable a more refined examination of whether this apparent perturbation in adoptive parent hostility at age 9.5 is temporary or whether it persists into later stages of development.

Our final set of analyses was exploratory; rather than a between home comparison as was used in the earlier analyses, we sought to link the biological sibling data aross homes, and examine sibling similarities in inhibitory control, peer problems, and hostile parenting. Because the siblings were reared apart since birth, in the absence of contact between adoptive and birth families, similarities in their behaviors can be considered to reflect genetic (or shared prenatal) influences. We hypothesized that biological sibling pairs would be correlated for their behaviors, and that the correlations would be higher in full-sibling pairs than in half-sibling pairs. However,

the correlations suggested that, by and large, the siblings were not correlated for any study variable. None of the associations was significant, and the largest correlation was negative (r = -.21 for full siblings' T2 peer problems), indicating that when one sibling scored high on peer problems, the other sibling scored low). The absence of similarities between biologically related children in a sibling pair suggests that the unique environments that children are reared in serve as an important predictor of behavior, including peer problems. This has important implications for the development of preventive interventions, because it shows that children can develop unique developmental trajectories as a function of the family and context within which they reside. This does not mean that genetic influences are not important. Given the small numbers of full and half sibling pairs in the current sample, our analyses are underpowered to detect small genetic effects. In fact, examination of the magnitude of correlations suggests that full sibling pairs tend to have more similar levels of inhibitory control than half sibling pairs (r = .17 vs. r = .05). If larger samples were available, it is possible that a genetic signal would be more apparent.

Nevertheless, our findings also highlight the importance of environmental influences on children's peer problems. The contexts that the adoptees and biological siblings lived in differed on a large number of qualities, including income and education (e.g., Natsuaki et al., in press), and yet many of the associations tested in our study were not significantly different between birth and adoptive homes. For example, the stability of parental hostility and peer problems and the associations between child inhibitory control and parental hostility and peer problems were similar for adoptees and biological siblings. This suggests underlying parallels in associations between inhibitory control, parenting, and peer problems, regardless of the other aspects of the family context. It also suggests the relevance of the unique environment that every child experiences as key in shaping sibling differences. Additional follow-up of this sample into

adolescence will provide an opportunity to examine whether the siblings reared apart grow more similar as they enter adolescence, or further diverge, commensurate with the large body of literature on the importance of nonshared environment influences during adolescence (e.g., Reiss et al., 2000).

Implications for Theory and Prevention

Although the current study was not experimental in nature, the pattern of findings provides some new leads for theory development and the development of preventive interventions aimed at reducing children's peer problems. First, the results support the notion that Dishion's peer deviancy training theory and model could be expanded to include a more prominent role for child factors, in particular inhibitory control, in the cascade of coercive interactions originating in the family and continuing into the peer realm. Our study tested direct and mediated effects, but it is likely that interactions between children's inhibitory control problems, parental hostility, and peer problems exist. Such models could also incorporate measured genetic contributions into this cascade via inclusion of polygenic risk scores, along the lines that Dishion was beginning to pursue in the final months of his remarkable career (e.g., Lemery-Chalfant et al., 2018). Given the possibility of passive gene-environment correlations in the association between parent psychopathology and child inhibitory control, expanded iterations of Dishion's peer deviancy training model might also benefit from considering how shared genetic liabilities may fuel coercive processes in the home during childhood.

The current study's suggestion of shared genetic liabilities also has implications for the development of preventive interventions. Where both biological parent and child have a predisposition toward low inhibitory control, it may be useful to directly coach the parent about this shared challenge, and discuss how this shared attribute can prompt parent-child interactions

to easily escalate into non-compliance and/or hostile interactions. Strategies could be discussed with parents for how to recognize this coercive cycle and parents could be guided on the use of effective parenting approaches to prevent escalation (e.g., time-out, calm and consistent responses). Teaching parents to use such parenting strategies may also help prevent the cross-lagged effect we found from children's peer problems to later hostile parenting in adoptive families, and to prevent the elicited effect from child inhibitory control challenges to parental hostility seen in both biological and adoptive families. Second, the very modest levels of behavioral similarity between biological siblings reared apart suggests that the family and local context that each child lives in are important contributors to variation in children's peer problems. Although a wide range of parenting and context variables were not included in the current study, as thus the precise mechanism of action is not discernable, the pattern of results generally suggests that the experiences children have in their homes and communities are influential forces that help shape their peer problems. This provides additional support for preventive interventions that focus on strengthening family relationships, building child skills, and/or increasing community supports (e.g., Hawkins et al., 2008; Leve & Chamberlain, 2005; Shaw et al., 2006) and suggests their relevance for preventing the cascade from low child inhibitory control to parental hostility and peer problems.

Study Limitations and Future Directions

Our study had a number of limitations that should be considered when evaluating the results. First, the study did not contain observational data for the constructs measured here, and thus parent- and child-report data were used. Although we used aggregate mother–father reports when available, the correlation between our primary variables may be inflated because of rater bias issues when the same individuals are rating both themselves (parental hostility) and their

child (peer problems). Relatedly, teacher- or peer reports of children's peer behavior would provide a more ecologically-valid measure of peer problems. A related measurement limitation is that the levels of parental hostility were fairly low for both groups, which may limit the clinical significance of the study.

Second, we had only two time points of data for this subsample of families. Although that enabled us to examine behavior change across an understudied period of development, we could not examine trajectories or earlier precursors of parent–peer cross-over effects. The extant literature on associations between parenting and peer problems suggests that there is a normative surge in peer delinquency during adolescence that is not captured by our study (Patterson et al., 1992). Further, longitudinal research that has been conducted earlier in development suggests that peer relations during this period can have long-term associations with later well-being, and that parent–child relationship quality earlier in development measured using attachment paradigms also predicts subsequent peer relations (Acar, Rudasill, Molfese, Torquati, & Prokasky, 2015; Ettekal & Ladd, 2015; Groh, Fearon, van IJzendoorn, Bakermans-Kranenburg, & Roisman, 2017; Parke & Ladd, 2016).

Third, unmeasured variables could have played an important role in the pathways studied. For example, we did not have a robust measure of prenatal risk exposure, which is important to help disentangle genetic from prenatal influences in our study design. These data are forthcoming in future data collection activities and will be incorporated in subsequent studies with this sample. We also did not have a measure of child externalizing problems that came from a measure different than our T2 peer problems measure. Child externalizing problems are an important mechanistic component of Dishion's deviancy training model (e.g., Dishion, 1990; Patterson et al., 1998), and its absence in our study is notable. As noted earlier, data from birth

fathers were also lacking, which resulted in a study approach that could test only 50% of genetic contributions to child inhibitory control and child peer problems. In addition, adoptive and birth families likely varied on many unmeasured variables, that could be important in explaining the between family differences found in our multi-group analysis. For example, in addition to the between group income difference reported here, earlier reports from some of the adoptive and birth families in our study indicate significant differences in maternal depressive symptoms, education level, and guiding parenting (Natsuaki et al., in press). These are other unmeasured variables such as the neighborhood or school context might interact with hostile parenting in its associations with children's inhibitory control or peer problems. Last, the sample size for full-sibling sets was small, which reduced our power to detect genetic influences in the within-sibling pair exploratory analyses. Future data collection plans include obtaining longitudinal data from many different types of siblings in this sample (e.g., genetically unrelated, genetically related living together), thus increasing the power for detecting genetic and shared environmental influences on child behavior.

Despite these limitations, our study provides new evidence that the well-known association from parental hostility to peer problems exists in middle childhood, but it may result in part from inhibitory control challenges that children may have that precipitate peer problems and parental hostility toward the child. Further, common genes that are shared between parents and children who live in the same household may also underlie associations between child inhibitory control and parent and peer relations. Finally, an important takeaway from our study is that in addition to the role of inhibitory control on peer and parent processes, the unique family context that each child resides in makes an important contribution to peer relations. The effect of the family context is such that full siblings who are reared apart in very different households

since birth tend not to highly resemble one another in terms of their inhibitory control, peer problems, or parental hostility received. These findings provide promise for improved family- and context-based interventions to prevent the cascade from coercive family processes to peer problems across childhood and adolescence.

References

Acar, I. H., Rudasill, K. M., Molfese, V., Torquati, J., & Prokasky, A. (2015). Temperament and preschool children's peer interactions. *Early Education and Development, 26*(4), 479–495. DOI: 10.1080/10409289.2015.1000718

Allen, J. P., Hauser, S. T., O'Connor, G. T., & Bell, K. L. (2002). Prediction of peer-rated adult hostility from autonomy struggles in adolescent–family interactions. *Development and Psychopathology*, *14*(1), 123–137.

Andreasen, N. C., Endicott, J., Spitzer, R. L., & Winokur, G. (1977). The family history method using diagnostic criteria: Reliability and validity. *Archives of General Psychiatry*, *34*(10), 1229–1235.

Ary, D. V, Duncan, T. E., Duncan, S. C., & Hops, H. (1999). Adolescent problem behavior: The influence of parents and peers. *Behaviour Research and Therapy*, *3*, 217–230. DOI: 0.1016/S0005-7967(98)00133-8

Ayoub, M., Briley, D. A., Grotzinger, A., Patterson, M. W., Engelhardt, L. E., Tackett, J. L., . . . Tucker-Drob, E. M. (2018). Genetic and environmental associations between child personality and parenting. *Social Psychological and Personality Science*. Online First. doi: 10.1177/1948550618784890

Bank, L., Burraston, B., & Snyder, J. (2004). Sibling conflict and ineffective parenting as predictors of adolescent boys' antisocial behavior and peer difficulties: Additive and interactional effects. *Journal of Research on Adolescence*, *14*(1), 99–125. DOI: 10.1111/j.1532-7795.2004.01401005.x

Bates, J. E., Schermerhorn, A. C., & Petersen, I. T. (2012). Temperament and parenting in developmental perspective. In M. Zentner & R. L. Shiner (Eds.), *Handbook of temperament*

(pp. 425–441). New York, NY: Guilford Press..

Beaver, K. M., Wright, J. P., & DeLisi, M. (2008). Delinquent peer group formation: Evidence of a gene X environment correlation. *The Journal of Genetic Psychology*, *169*(3), 227–244. DOI: 10.3200/GNTP.169.3.227-244

Bell, R. Q. (1968). A reinterpretation of the direction of effects in studies of socialization. *Psychological Review*, *75*(2), 81–95. DOI: 10.1037/h0025583

Bell, R. Q. (1979). Parent, child, and reciprocal influences. *American Psychologist*, *34*(10), 821–826. DOI: 10.1037/0003-066X.34.10.821

Bell, R. Q. (2017). A reinterpretation of the direction of effects in studies of socialization. In *Interpersonal Development* (pp. 93–107). Routledge. DOI: 10.4324/9781351153683-4

Bell, R. Q., & Chapman, M. (1986). Child effects in studies using experimental or brief longitudinal approaches to socialization. *Developmental Psychology*, *22*(5), 595–603. DOI: 10.1037/0012-1649.22.5.595

Benson, M. J., & Buehler, C. (2012). Family process and peer deviance influences on adolescent aggression: Longitudinal effects across early and middle adolescence. *Child Development*, *83*(4), 1213–1228. DOI: 10.1111/j.1467-8624.2012.01763.x

Bentler, P. M. (1990). Fit indexes, lagrange multipliers, constraint changes and incomplete data in structural models. *Multivariate Behavioral Research*, *25*(2), 163–172. DOI: 10.1207/s15327906mbr2502_3

Bezdjian, S., Baker, L. A., & Tuvblad, C. (2011). Genetic and environmental influences on impulsivity: A meta-analysis of twin, family and adoption studies. *Clinical Psychology Review*, *31*(7), 1209–1223. DOI: 10.1016/j.cpr.2011.07.005

Boivin, M., Brendgen, M., Vitaro, F., Forget-Dubois, N., Feng, B., Tremblay, R. E., & Dionne,

G. (2013). Evidence of gene–environment correlation for peer difficulties: Disruptive behaviors predict early peer relation difficulties in school through genetic effects. *Development and Psychopathology*, *25*(01), 79–92. DOI: 10.1017/s0954579412000910

Brendgen, M. (2012). Genetics and peer relations: A review. *Journal of Research on Adolescence*, *22*(3), 419–437. DOI: 10.1111/j.1532-7795.2012.00798.x

Brody, G. H., Conger, R., Gibbons, F. X., Ge, X., McBride Murry, V., Gerrard, M., & Simons, R. L. (2001). The influence of neighborhood disadvantage, collective socialization, and parenting on African American children's affiliation with deviant peers. *Child Development*, *72*(4), 1231–1246. DOI: 10.1111/1467-8624.00344

Brown, B. B., & Bakken, J. P. (2011). Parenting and peer relationships: Reinvigorating research on family–peer linkages in adolescence. *Journal of Research on Adolescence*, *21*(1), 153–165. DOI: 10.1111/j.1532-7795.2010.00720.x

Browne, M., & Cudeck, R. (1993). Alternative ways of assessing model fit. *Sage Focus Editions*, *154*, 136–136.

Bullock, B. M., Deater-Deckard, K., & Leve, L. D. (2006). Deviant peer affiliation and problem behavior: A test of genetic and environmental influences. *Journal of Abnormal Child Psychology*, *34*(1), 29–41. DOI: 10.1007/s10802-005-9004-9

Burt, S. A., McGue, M., & Iacono, W. G. (2009). Nonshared environmental mediation of the association between deviant peer affiliation and adolescent externalizing behaviors over time: Results from a cross-lagged monozygotic twin differences design. *Developmental Psychology*, *45*(6), 1752–1760. DOI: 10.1037/a0016687

Caspi, A., Houts, R. M., Belsky, D. W., Goldman-Mellor, S. J., Harrington, H., Israel, S., . . . Moffitt, T. E. (2014). The p factor: One general psychopathology factor in the structure of

psychiatric disorders? *Clinical Psychological Science, 2*(2), 119–137. DOI: 10.1177/2167702613497473.

Chang, H., Shaw, D. S., Shelleby, E. C., Dishion, T. J., & Wilson, M. N. (2017). The long-term effectiveness of the Family Check-Up on peer preference: Parent-child interaction and child effortful control as sequential mediators. *Journal of Abnormal Child Psychology*, *45*(4), 705–717. DOI: 10.1007/s10802-016-0198-9

Choe, D. E., Shaw, D. S., Brennan, L. M., Dishion, T. J., & Wilson, M. N. (2014). Inhibitory control as a mediator of bidirectional effects between early oppositional behavior and maternal depression. *Development and Psychopathology*, *26*, 1129-1147, http://dx.doi.org/10.1017/s0954579414000613

Cicchetti, D. (2016). Socioemotional, personality, and biological development: Illustrations from a multilevel developmental psychopathology perspective on child maltreatment. *Annual Review of Psychology*, *67*, 187–211. DOI: 10.1146/annurev-psych-122414-033259

Cicchetti, D., & Lynch, M. (1993). Toward an ecological/transactional model of community violence and child maltreatment: Consequences for children's development. *Psychiatry*, *56*, 96–118. DOI: 10.1521/00332747.1993.11024624

Cicchetti, D., & Toth, S. L. (2016). *Child maltreatment and developmental psychopathology: A multilevel perspective*. In D. Cicchetti (Ed.), *Developmental psychopathology: Maladaptation and psychopathology* (pp. 457–512). Hoboken, N.J.: Wiley.

Dishion, T. J. (1990). The family ecology of boys' peer relations in middle childhood. *Child Development*, *61*(3), 874–892. DOI: 10.1111/j.1467-8624.1990.tb02829.x

Dishion, T. J., Duncan, T. E., Eddy, J. M., Fagot, B. I., & Fetrow, R. (1994). The world of parents and peers: Coercive exchanges and children's social adaptation. *Social*

Development, *3*(3), 255–268. DOI: 10.1111/j.1467-9507.1994.tb00044.x

Dishion, T. J., & Patterson, G. R. (2006). The development and ecology of antisocial behavior. In D. Cicchetti & D. J. Cohen (Eds.), *Developmental psychopathology: Vol. 3. Risk, disorder and adaptation* (pp. 503–541). New York, NY: Wiley.

Dishion, T. J., Patterson, G. R., & Griesler, P. C. (1994). Peer adaptations in the development of antisocial behavior. In L. R. Huesmann (Ed.), *Current perspectives on aggressive behavior* (pp. 61–95). New York, NY: Plenum. DOI: 10.1007/978-1-4757-9116-7_4

Dishion, T. J., Patterson, G. R., Stoolmiller, M., & Skinner, M. L. (1991). Family, school, and behavioral antecedents to early adolescent involvement with antisocial peers. *Developmental Psychology*, *27*(1), 172–180. DOI: 10.1037/0012-1649.27.1.172

Eisenberg, N., Hofer, C., & Vaughan, J. (2007). Effortful control and its socioemotional consequences. In J. J. Gross (Ed.), *Handbook of emotion regulation* (pp. 287–288). New York, NY: Guilford Press.

Eisenberg, N., Sadovsky, A., Spinrad, T. L., Fabes, R. A., Losoya, S. H., Valiente, C., … Shepard, S. A. (2005). The relations of problem behavior status to children's negative emotionality, effortful control, and impulsivity: Concurrent relations and prediction of change. *Developmental Psychology*, *41*(1), 193–211. DOI: 10.1037/0012-1649.41.1.193

Elam, K. K., Harold, G. T., Neiderhiser, J. M., Reiss, D., Shaw, D. S., Natsuaki, M. N., … Leve, L. D. (2014). Adoptive parent hostility and children's peer behavior problems: Examining the role of genetically informed child attributes on adoptive parent behavior. *Developmental Psychology*, *50*(5), 1543–1552. DOI: 10.1037/a0035470

Ettekal, I., & Ladd, G. W. (2015). Developmental pathways from childhood aggression–disruptiveness, chronic peer rejection, and deviant friendships to early-adolescent rule

breaking. *Child Development, 86*(2), 614–631. DOI: 10.1111/cdev.12321

Forgatch, M. S., Snyder, J. J., Patterson, G. R., Pauldine, M. R., Chaw, Y., Elish, K., … Richardson, E. B. (2016). Resurrecting the chimera: Progressions in parenting and peer processes. *Development and Psychopathology*, *28*(03), 689–706. DOI: 10.1017/s0954579416000250

Gagne, J. R., & Saudino, K. J. (2016). The development of inhibitory control in early childhood: A twin study from 2–3 years. *Developmental Psychology*, *52*(3), 391–399. DOI: 10.1037/dev0000090

Gardner, T. W., Dishion, T. J., & Connell, A. M. (2008). Adolescent self-regulation as resilience: Resistance to antisocial behavior within the deviant peer context. *Journal of Abnormal Child Psychology*, *36*(2), 273–284. DOI: 10.1007/s10802-007-9176-6

Ge, X., Conger, R. D., Cadoret, R. J., Neiderhiser, J. M., Yates, W., Troughton, E., & Stewart, M. A. (1996). The developmental interference between nature and nurture: A mutual influence model of child antisocial behavior and parent behaviors. *Developmental Psychology*, *32*(4), 574–589. DOI: 10.1037/0012-1649.33.1.2

Goodman, R. (2001). Psychometric properties of the strengths and difficulties questionnaire. *Journal of the American Academy of Child and Adolescent Psychiatry*, *40*(11), 1337–1345. DOI: 10.1097/00004583-200111000-00015

Groh, A. M., Fearon, R. P., van IJzendoorn, M. H., Bakermans-Kranenburg, M. J., & Roisman, G. I. (2017). Attachment in the early life course: Meta-analytic evidence for its role in socioemotional development. *Child Development Perspectives, 11*(1), 70–76. DOI: 10.1111/cdev.12677

Gross, H. E., Shaw, D. S., Burwell, R. A., & Nagin, D. S. (2009). Transactional processes in

child disruptive behavior and maternal depression: A longitudinal study from early childhood to adolescence. *Development and Psychopathology*, *21*(1), 139–156. DOI: 10.1017/S0954579409000091

Hawkins, J. D., Brown, E. C., Oesterle, S., Arthur, M. W., Abbott, R. D., & Catalano, R. F. (2008). Early effects of Communities That Care on targeted risks and initiation of delinquent behavior and substance use. *Journal of Adolescent Health*, *43*, 15-22.

Holmes, C. J., Kim-Spoon, J., & Deater-Deckard, K. (2016). Linking executive function and peer problems from early childhood through middle adolescence. *Journal of Abnormal Child Psychology*, *44*(1), 31–42. DOI: 10.1007/s10802-015-0044-5

Hu, L., & Bentler, P. M. (1999). Cutoff criteria for fit indexes in covariance structure analysis: Conventional criteria versus new alternatives. *Structural Equation Modeling: A Multidisciplinary Journal*, *6*(1), 1–55. DOI: 10.1080/10705519909540118

Hu, L. T., & Bentler, P. M. (1995). Evaluating model fit. In R. H. Hoyle (Ed.), *Structural equation modeling: Concepts, issues, and applications* (pp. 76–99). Thousand Oaks, CA: Sage Publications, Inc.

Iervolino, A. C., Pike, A., Manke, B., Reiss, D., Hetherington, E. M., & Plomin, R. (2002). Genetic and environmental influences in adolescent peer socialization: Evidence from two genetically sensitive designs. *Child Development*, *73*(1), 162–174. DOI: 10.1111/1467-8624.00398

Karreman, A., Van Tuijl, C., van Aken, M. A., & Deković, M. (2006). Parenting and self-regulation in preschoolers: A meta-analysis. *Infant and Child Development: An International Journal of Research and Practice*, *15*(6), 561–579. DOI: 10.1002/icd.478

Kendler, K. S., Aggen, S. H., Knudsen, G. P., Røysamb, E., Neale, M. C., & Reichborn-Kjeennerud, T. (2011). The structure of genetic and environmental risk factors for syndromal and subsyndromal common DSM-IV Axis I and Axis II disorders. *American Journal of Psychiatry, 168,* 29–39. DOI: 10.1176/appi.ajp.2010.10030340

Kessler, R., & Ustun, T. (2004). The World Mental Health (WMH) survey initiative version of The World Health Organization (WHO) Composite International Diagnostic Interview (CIDI). *International Journal of Methods in Psychiatric Research, 13,* 93–121. DOI: 10.1002/mpr.168

Kiff, C. J., Lengua, L. J., & Zalewski, M. (2011). Nature and nurturing: Parenting in the context of child temperament. *Clinical Child and Family Psychology Review*, *14*(3), 251–301. DOI: 10.1007/s10567-011-0093-4

Klahr, A. M., & Burt, S. A. (2014). Elucidating the etiology of individual differences in parenting: A meta-analysis of behavioral genetic research. *Psychological Bulletin*, *140*(2), 544–586. DOI: 10.1037/a0034205

Knafo, A., & Jaffee, S. R. (2013). Gene–environment correlation in developmental psychopathology. *Development and Psychopathology*, *25*(1), 1–6. DOI: 10.1017/S0954579412000855

Kochanska, G., & Knaack, A. (2003). Effortful control as a personality characteristic of young children: Antecedents, correlates, and consequences. *Journal of Personality*, *71*(6), 1087–1112. DOI: 10.1111/1467-6494.7106008

Krueger, R. F., & Markon, K. E. (2006). Reinterpreting comorbidity: A model-based approach to understanding and classifying psychopathology. *Annual Review of Clinical Psychology, 2,* 111–133. DOI: 10.1146/annurev.clinpsy.2.022305.095213.

Larsson, H., Viding, E., Rijsdijk, F. V., & Plomin, R. (2008). Relationships between parental negativity and childhood antisocial behavior over time: A bidirectional effects model in a longitudinal genetically informative design. *Journal of Abnormal Child Psychology*, *36*(5), 633–645. DOI: 10.1007/s10802-007-9151-2

Lemery-Chalfant, K., Clifford, S., Dishion, T. J., Shaw, D. S., & Wilson, M. N. (2018). Genetic moderation of the effects of the Family Check-Up intervention on children's internalizing symptoms: A longitudinal study with a racially/ethnically diverse sample. *Development and Psychopathology*, *30*(5), 1729-1747.

Lengua, L. J. (2006). Growth in temperament and parenting as predictors of adjustment during children's transition to adolescence. *Developmental Psychology*, *42*(5), 819–832. DOI: 10.1037/0012-1649.42.5.819

Leve, L. D., & Chamberlain, P. (2005). Association with delinquent peers: Intervention effects for youth in the juvenile justice system. *Journal of Abnormal Child Psychology*, *33*, 339-347.

Leve, L. D., Neiderhiser, J. M., Harold, G. T., Natsuaki, M. N., Bohannan, B. J. M., & Cresko, W. A. (2018). Naturalistic experimental designs as tools for understanding the role of genes and the environment in prevention research. *Prevention Science*, *19*, 68–78. DOI: 10.1007/s11121-017-0746-8

Leve, L. D., Neiderhiser, J. M., Shaw, D. S., Ganiban, J., Natsuaki, M. N., & Reiss, D. (2013). The Early Growth and Development Study: A prospective adoption study from birth through middle childhood. *Twin Research and Human Genetics: The Official Journal of the International Society for Twin Studies*, *16*(1), 412–423. DOI: 10.1017/thg.2012.126

Manke, B., McGuire, S., Reiss, D., Hetherington, E., & et al. (1995). Genetic contributions to

adolescents' extrafamilial social interactions: Teachers, best friends, and peers. *Social Development, 4*(3), 238-256.

Marceau, K., Rolan, E., Leve, L. D., Ganiban, J. M., Reiss, D., Shaw, D. S., Natsuaki, M. N., Egger, H., & Neiderhiser, J. M. (2019). Parenting and prenatal risk as moderators of genetic influences on conduct problems during middle childhood. *Developmental Psychology.* Online First.

Masten, A. S., & Cicchetti, D. (2010). Developmental cascades. *Development and Psychopathology*, *22*(3), 491–495. DOI: 10.1017/s0954579410000222

Melby, J. N., & Conger, R. D. (2001). The Iowa Family Interaction Rating Scales: Instrument summary. In P. K. Kerig & K. M. Lindahl (Eds.), *Family observational coding systems: Resources for systemic research* (pp. 33–58). Mahwah, NJ: Lawrence Erlbaum Associates Publishers.

Muthén, L. K., & Muthén, B. O. (1998–2010). *Mplus user's guide* (6th ed.). Los Angeles, CA: Author.

Narusyte, J., Neiderhiser, J. M., D'Onofrio, B. M., Reiss, D., Spotts, E. L., Ganiban, J., & Lichtenstein, P. (2008). Testing different types of genotype–environment correlation: An extended children-of-twins model. *Developmental Psychology*, *44*(6), 1591–1603. DOI: 10.1037/a0013911

Natsuaki, M. N., Neiderhiser, J. M., Harold, G. T., Shaw, D. S., Reiss, D., & Leve, L. D. (in press). A sibling comparison study on rearing environment differences. *Developmental Psychology.*

Neiderhiser, J. M., Reiss, D., Hetherington, E. M., & Plomin, R. (1999). Relationships between parenting and adolescent adjustment over time: genetic and environmental

contributions. *Developmental Psychology*, *35*(3), 680-692. DOI:10.1037/0012-1649.35.3.680

Nigg, J. T., & Huang-Pollock, C. L. (2003). An early-onset model of the role of executive functions and intelligence in conduct disorder/delinquency. In B. B. Lahey, T. E. Moffitt, & A. Caspi (Eds.), *Causes of conduct disorder and juvenile delinquency* (pp. 227–253). New York, NY: Guilford Press.

O'Connor, T. G., Deater-Deckard, K., Fulker, D., Rutter, M., & Plomin, R. (1998). Genotype–environment correlations in late childhood and early adolescence: Antisocial behavioral problems and coercive parenting. *Developmental Psychology*, *34*(5), 970–981. DOI: 10.1037/0012-1649.34.5.970

Olson, S. L., Sameroff, A. J., Kerr, D. C. R., Lopez, N. L., & Wellman, H. M. (2005). Developmental foundations of externalizing problems in young children: The role of effortful control. *Development and Psychopathology*, *17*(1), 25–45. DOI: 10.1017/S0954579405050029

Pardini, D. A., Fite, P. J., & Burke, J. D. (2008). Bidirectional associations between parenting practices and conduct problems in boys from childhood to adolescence: The moderating effect of age and African-American ethnicity. *Journal of Abnormal Child Psychology*, *36*(5), 647–662. DOI: 10.1007/s10802-007-9162-z

Parke, R. D., & Ladd, G. W. (Eds.). (2016). *Family-peer relationships: Modes of linkage*. Hillsdale, NJ: Lawrence Erlbaum Associates, Inc.

Patterson, G. R. (1982). *Coercive family process* (3rd ed.). Eugene, OR: Castalia Publishing Company.

Patterson, G. R., & Dishion, T. J. (1985). Contributions of families and peers to delinquency.

Criminology, *1*, 63–79. DOI: 10.1111/j.1745-9125.1985.tb00326.x

Patterson, G. R., Forgatch, M. S., Yoerger, K. L., & Stoolmiller, M. (1998). Variables that initiate and maintain an early-onset trajectory for juvenile offending. *Development and Psychopathology*, *10*(3), 531–547. DOI: 10.1017/S0954579498001734

Patterson, G. R., Reid, J. B., & Dishion, T. J. (1992). *Antisocial boys*. Eugene, OR: Castalia.

Pike, A., & Atzaba-Poria, N. (2003). Do sibling and friend relationships share the same temperamental origins? A twin study. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, *44*(4), 598–611. DOI: 10.1111/1469-7610.00148

Plomin, R., DeFries, J. C., & Loehlin, J. C. (1977). Genotype–environment interaction and correlation in the analysis of human behavior. *Psychological Bulletin*, *84*(2), 309–322. DOI: 10.1037/0033-2909.84.2.309

Polderman, T. J. C., de Geus, E. J. C., Hoekstra, R. A., Bartels, M., van Leeuwen, M., Verhulst, F. C., … Boomsma, D. I. (2009). Attention problems, inhibitory control, and intelligence index overlapping genetic factors: A study in 9-, 12-, and 18-year-old twins. *Neuropsychology*, *23*(3), 381–391. DOI: 10.1037/a0014915

Putnam, S. P., & Rothbart, M. K. (2006). Development of short and very short forms of the Children's Behavior Questionnaire. *Journal of Personality Assessment*, *87*(1), 102–112. DOI: 10.1207/s15327752jpa8701_09

Reiss, D., Neiderhiser, J. M., Hetherington, E. M., & Plomin, R. (2000). *The relationship code : deciphering genetic and social influences on adolescent development*. Cambridge, Ma.: Harvard University Press.

Robins, L. N., Helzer, J. E., Croughan, J., & Ratcliff, K. S. (1981). National Institute of Mental Health Diagnostic Interview Schedule: Its history, characteristics, and validity. *Archives of General Psychiatry*, *38*(4), 381–389. DOI:10.1001/archpsyc.1981.01780290015001

Rothbart, M. K., Ahadi, S. A., Hershey, K. L., & Fisher, P. (2001). Investigations of temperament at three to seven years: The children's behavior questionnaire. *Child Development*, *72*(5), 1394–1408. DOI: 10.1111/1467-8624.00355

Rothbart, M. K., & Bates, J. E. (2006). Temperament. In W. Damon & N. Eisenberg (Eds.), *Handbook of child psychology: Vol. 3. Social, emotional, and personality development* (6th ed., pp. 99–166). Hoboken, NJ: Wiley.

Satorra, A., & Bentler, P. M. (2010). Ensuring positiveness of the scaled difference chi-square test statistic. *Psychometrika*, *75*(2), 243–248. DOI:10.1007/s11336-009-9135-y

Scarr, S., & McCartney, K. (1983). How people make their own environments: A theory of genotype → environment effects. *Child Development*, *54*(2), 424–435. DOI: 10.2307/1129703

Shaw, D. S., & Bell, R. Q. (1993). Developmental theories of parental contributors to antisocial behavior. *Journal of Abnormal Child Psychology*, *21*(5), 493–518. DOI: 10.1007/BF00916316

Shaw, D. S., Dishion, T. J., Supplee, L., Gardner, F., & Arnds, K. (2006). Randomized trial of a family-centered approach to the prevention of early conduct problems: 2-year effects of the family check-up in early childhood. *Journal of Consulting and Clinical Psychology*, *74*, 1 - 9.

Snyder, J., Dishion, T. J., & Patterson, G. R. (1986). Determinants and consequences of associating with deviant peers during preadolescence and adolescence. *The Journal of Early*

Adolescence, *6*(1), 29–43. DOI: 10.1177/0272431686061003

Snyder, J., McEachern, A., Schrepferman, L., Just, C., Jenkins, M., Roberts, S., & Lofgreen, A. (2010). Contribution of peer deviancy training to the early development of conduct problems: Mediators and moderators. *Behavior Therapy*, *41*(3), 317–328. DOI: 10.1016/j.beth.2009.05.001

Spinrad, T. L., Eisenberg, N., Gaertner, B., Popp, T., Smith, C. L., Kupfer, A., … Hofer, C. (2007). Relations of maternal socialization and toddlers' effortful control to children's adjustment and social competence. *Developmental Psychology*, *43*(5), 1170–1186. DOI: 10.1037/0012-1649.43.5.1170

Tarantino, N., Tully, E. C., Garcia, S. E., South, S., Iacono, W. G., & McGue, M. (2014). Genetic and environmental influences on affiliation with deviant peers during adolescence and early adulthood. *Developmental Psychology*, *50*(3), 663–673. DOI: 10.1037/a0034345

Trentacosta, C. J., & Shaw, D. S. (2009). Emotional self-regulation, peer rejection, and antisocial behavior: Developmental associations from early childhood to early adolescence. *Journal of Applied Developmental Psychology*, *30*(3), 356–365. DOI: 10.1016/j.appdev.2008.12.016

Walker, H., & McConnell, S. R. (1988). *The Walker-McConnell Scale of Social Competence and School Adjustment*. Austin, TX: Pro-Ed.

Waszczuk, M. A., Zavos, H. M., Gregory, A. M., & Eley, T. C. (2014). The phenotypic and genetic structure of depression and anxiety disorder symptoms in childhood, adolescence, and young adulthood. *JAMA Psychiatry*, *71*(8), 905–916. DOI: 10.1001/jamapsychiatry.2014.655

Table 1. Means and Standard Deviations of Study Variables

	M (*SD*)		
	Adoptive home (*n* = 135 children)	Birth home (*n* = 173 children)	Total (*N* = 308)
Inhibitory control T1	4.59 (0.74)	4.69 (0.96)	4.64 (0.86)
Parental hostility T1	11.05 (2.89)	11.06 (3.6)	11.06 (3.33)
Peer problems T1	13.72 (10.52)	17.51 (13.62)	15.80 (12.44)*
Parental hostility T2	11.61 (3.18)	10.54 (3.53)	11.02 (3.42)*
Peer problems T2	1.97 (1.95)	1.97 (1.78)	1.96 (1.86)
BM internalizing symptoms	−0.07 (1.29)	−0.03 (1.33)	−0.05 (1.31)
BM externalizing symptoms	0.24 (1.46)	0.27 (1.41)	0.24 (1.43)

Note. *$p < .05$ group differences between children in adoptive home versus birth home. The total sample ($n = 308$) included 173 linked sibling sets and 18 additional biological siblings who were missing the linked adoptive sibling data but had linked birth parent data.

Table 2. Correlation Matrix of Study Variables After Controlling for Income

	1	2	3	4	5	6
1. Inhibitory control T1	–					
2. Parental hostility T1	.37**	–				
3. Peer problems T1	.48**	.22**	–			
4. Parental hostility T2	.36**	.56**	.21**	–		
5. Peer problems T2	.37**	.21**	.49**	.25**	–	
6. BM internalizing symptoms	.14*	.10	.17**	−.02	.10	–
7. BM externalizing symptoms	.03	.10	.06	.11	.17**	.30**

Note. *$p < .05$, **$p < .01$. BM = birth mother.

Table 3. Tests of Model Fit Path Estimate Differences Between Adoptive and Birth Home, Corresponding to the Model Shown in Figure 3.

	CFI	TLI	RMSEA	Model comparison	Diff	*p*-value
M1 = Autoregressive parental hostility	0.98	0.96	0.05	M1 vs M10	3.45	0.06
M2 = Autoregressive peer problems	0.99	0.97	0.04	M2 vs M10	2.83	0.08
M3 = BM internalizing to inhibitory control T1	0.97	0.94	0.05	M3 vs M10	7.08	0.01
M4 = BM externalizing to inhibitory control T1	0.99	0.99	0.03	M4 vs M10	0.64	0.42
M5 = BM internalizing to peer problemsT1	0.99	0.97	0.04	M5 vs M10	2.12	0.15
M6 = Inhibitory control T1 to parental hostility T2	1.00	0.99	0.02	M6 vs M10	0.00	0.95
M7 = Cross-lagged peer to parental hostility T2	0.98	0.93	0.05	M7 vs M10	4.67	0.03
M8 = Inhibitory control T1 to peer problems T2	0.99	0.98	0.03	M8 vs M10	0.97	0.32
M9 = Cross-lagged parental hostility to peer problems T2	0.99	0.98	0.04	M9 vs M10	2.14	0.14
M10 = All free	0.99	0.98	0.03			

Note. **BOLD** = differences between adoptive and birth home at $p < .05$; CFI = comparative fit index; TLI = Tucker–Lewis index; RMSEA = root mean square error of approximation; M = model; BM = birth mother. Differences in path estimates were tested with adjusted chi-square using the Satorra-Bentler scaling correction.

Table 4. Within-Sibling-Pair Correlations by Genetic Relatedness, for Rearing Parent and Child Variables

	Full-sibling pairs	Half-sibling pairs	Full sample
Inhibitory control T1	0.17	0.05	0.06
Parental hostility T1	0.05	0.04	0.04
Peer problems T1	0.06	−0.02	−0.02
Parental hostility T2	0.16	0.001	0.03
Peer problems T2	−0.21	0.01	−0.02

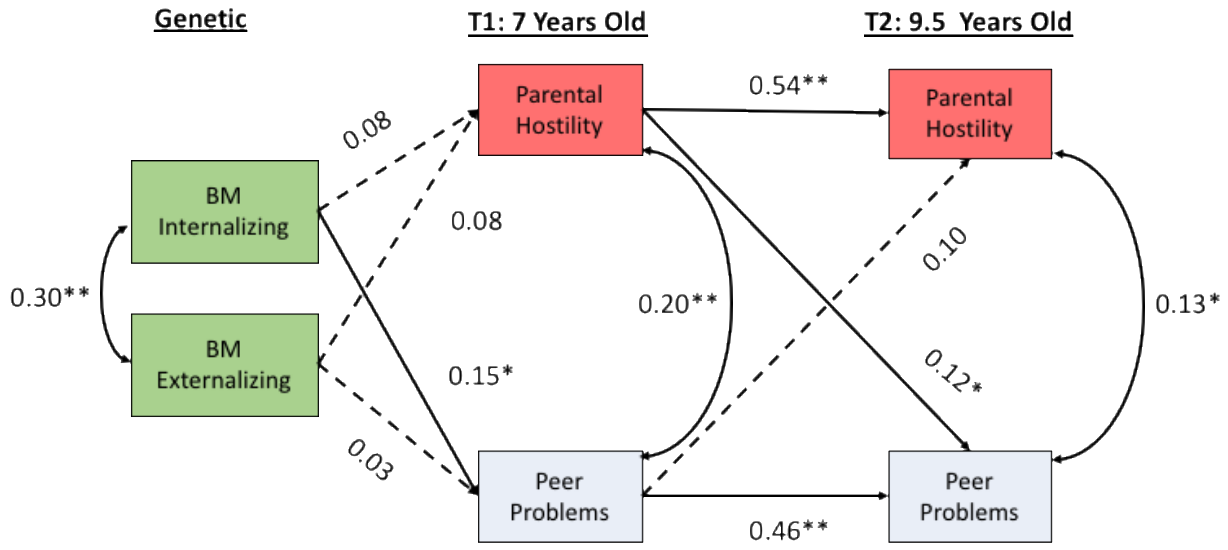

Figure 1. Conventional cross-lagged model testing the competing effects of parental hostility and peer problems.

Note. *$p < .05$, **$p < .01$. Black lines indicate significant paths, dashed lines indicate non-significant paths, and curved paths depict correlations. BM = birth mother; $\chi^2(4) = 7, 79$, $p = .10$; CFI = 0.98; TCI = 0.93; RMSEA = 0.05, SRMR = 0.03.

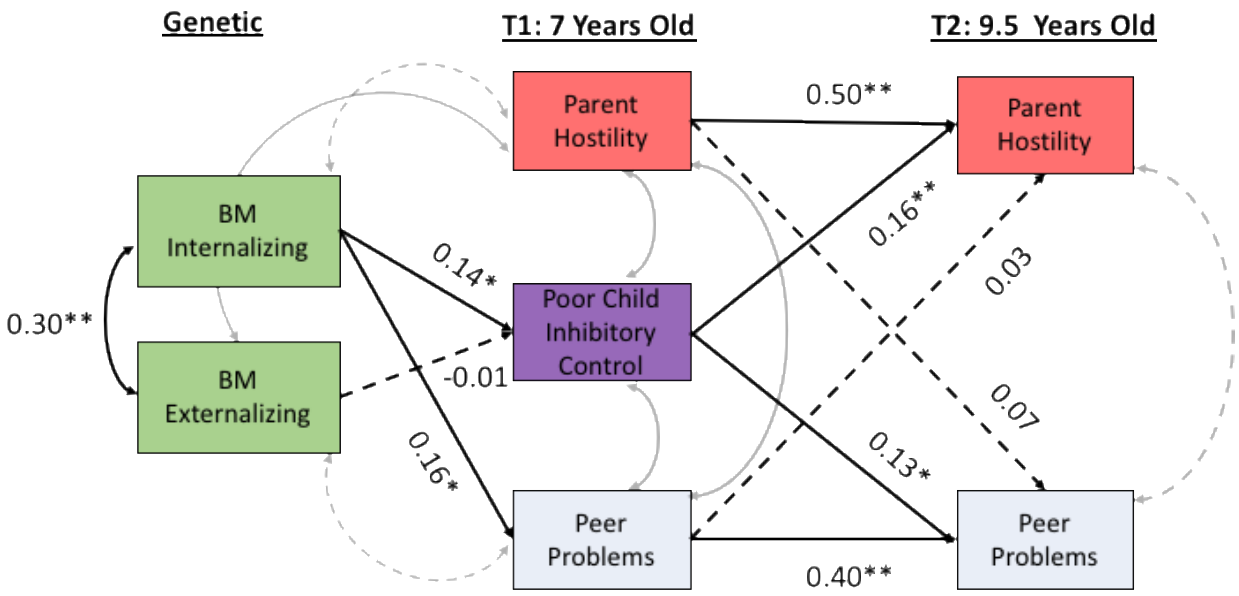

Figure 2. Cross-lagged model that incorporates the association between child inhibitory control (T1) on T2 outcomes.

Note. *$p < .05$, **$p < .01$. Black lines indicate significant paths, dashed lines indicate non-significant paths, and curved paths depict correlations. BM = birth mother; $\chi^2(5) = 9.57$, $p = .09$; CFI = 0.98; TCI = 0.94; RMSEA = 0.05; SRMR = 0.03.

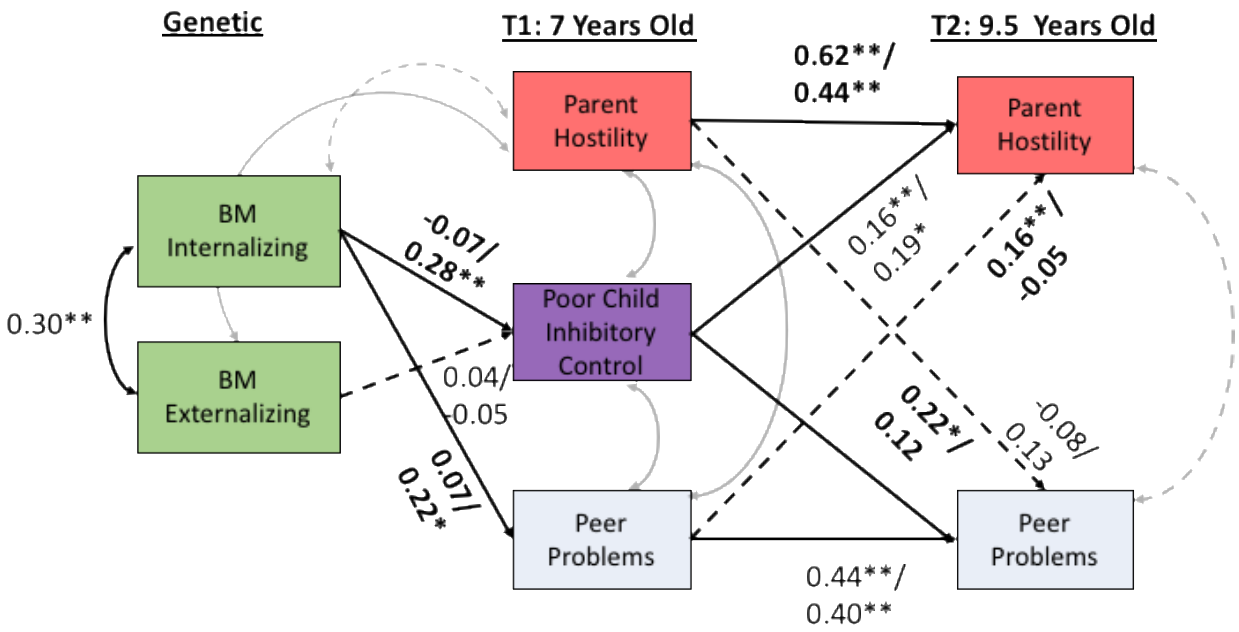

Figure 3. Multigroup model that tests path invariance in Figure 2 by allowing paths to vary for adoptees versus birth parent–reared children.

Note. *$p < .05$, **$p < .01$. Black lines indicate significant paths, dashed lines indicate non-significant paths, and curved paths depict correlations, and bolded coefficients indicate the magnitude of parameter estimates visually differed. Path coefficients presented as adoptive parent home/birth parent home.